SECONDARY ACUTE LETHAL CATATONIA

By

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AT various times attempts have been made to establish a specific pathological entity "acute lethal catatonia". Stauder and Scheid (quoted by Mayer Gross *et al.*, 1954) among the older writers have collected a number of fatal cases and more recently Locher (1941) has suspected qualitative difference in the type of illness of those mental patients who die suddenly in an acute catatonic state. However the general view nowadays seems to be that death is due to exhaustion or intercurrent illness. A fatal outcome is very unusual in current practice perhaps because of improved therapy. The symptoms of acute catatonic schizophrenia are so striking that a determined quest for a biochemical basis for the whole schizophrenic process has been sought in this, its most dramatic manifestation. Success in this direction has often been claimed (e.g. Fischer, 1953; Rieder, 1954; Gjessing, 1953, *inter alia*) but has also frequently been disputed (Georgi, Rieder and Weber, 1954). The following case is reported because the symptoms seemed to be clearly related to physiological change. It tends also to throw further doubt on the entity of acute lethal catatonia of Locher. Interesting EEG findings and characteristic response to amytal are also noted.

CASE REPORT

A woman aged 38, was admitted to mental hospital on 25 February, 1956. She was married, a housewife and lived at home with her husband and five children. She had been treated as an in-patient at a near-by general hospital for the previous six days as a middle lobe pneumonia and altogether had been ill for ten days. She had been treated at home by her own doctor for respiratory infection for the first four days but admission to hospital became advisable because of inadequate nursing care at home. She was found to have extensive physical signs in the chest and X-ray on 20 February, 1956 showed an opacity in the right lower zone which looked like consolidation of the middle lobe (radiologist's report). She is described as being apparently in an extremely toxic state, mildly cyanosed, rather confused mentally. Haemoglobin 11·4 gm., leucocytes 11,600 including 80 per cent. polymorphs. A series of agglutination and complement fixation tests were all negative (salmonella, Br. abortus, influenza, psittacosis, "Q" fever). Her respirations were 30–36 per minute and the rhythm was irregular. She was nursed in an oxygen tent and given penicillin 500,000 units stat. and 250,000 six-hourly thereafter, and vitamins A, B, C, D, E, K, P. Although considerable improvement took place in her lung condition, mental changes became apparent. She was apprehensive and detached, she would start sentences without finishing them, was unable to give any account of herself, and gradually became completely inaccessible and mute. She was noted to have general muscular hypertonus but such examination of the central nervous system as the patient allowed showed no focal signs. Eventually a period of excitement and motor overactivity greatly disturbed the ward and the patient was removed to observation at a mental hospital. ? Schizophrenia, ? toxic confusional state, ? encephalitis.

Condition on Admission

Shapiro (1956) has classified catatonic symptoms. The following description of the patient's condition is in accordance with his schema.

A. Hypokinetic phenomena:

(1) Diminished motor phenomena
- (*a*) Greatly retarded impulse to movement and action.
- (*b*) Maintenance of motionless posture for long periods.
- (*c*) Mute and stuporose.
- (*d*) Mask-like facies.
- (*e*) Grimacing and gesturing.
- (*f*) Fixed blank stare.
- (*g*) Sluggish rigid musculature.

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(2) Catatonic phenomena
  (*a*) Refusal of food and need to be fed by others.
  (*b*) Refusal to dress and undress.
  (*c*) Incontinence.
  (*d*) Absence of reaction to pin-prick.

(3) Physiological negativism
  (*a*) Active negativism.

B. Hyperkinetic phenomena

(1) Irregular hyperkinesis: Unorganized activity.
Active negativism.

C. Autonomic phenomena
(1) Respiratory changes—irregular respiration.
(2) Acrocyanosis.
(3) Hyperidrosis.

Of the total aggregate of 25 catatonic phenomena described by Shapiro therefore, this patient showed 17—a very high proportion in such a protean disease. She was a great nursing problem, holding herself rigid, actively resisting all care and attention and refusing food. The rigidity was extreme but plastic. Even tube feeding was difficult because of the patient's negativism. Breathing was laboured, temperature 100, pulse 100, respiration 32. She appeared to resist even her own cough reflex with the result that her cough was completely ineffectual. She did not trouble to cough up sputum as she easily could have done. "Estopen" 500,000 units daily was continued, and she was given 100 c.c. Parentrovite intravenously daily from 26–29 February, along the lines recommended by Gould (1954).

Response to Amytal

In this state of akinetic mutism some awareness of the environment was certainly present for negativism was of an active kind. The effect of intravenous amytal was therefore tried (15 gr. in 20 c.c. water). After 3–4 c.c. the patient at once relaxed, focused her attention and the following conversation ensued: "Do you know where you are?" "Yes." "Where?" "In hospital." "Which hospital?" "I don't know." "Well, it's in Gosforth." "I like the — best." "How many children have you?" "Five." "What is your husband's name?" "John Edward." "Where does he work?" "In the factory—ten pounds a week!" Here she smiled pleasantly and her mood became quite cheerful. Soon she began to cough violently, it seemed that the cough reflex was now able to operate more effectively. She stated that she was thirsty and took her first voluntary meal since admission. She asked for and was given the bedpan. For the rest of that evening she talked a good deal of confused and delusional material, but when her husband visited she asked after the children, and she asked for and drank milk. She had 6 c.c. amytal in all. Next morning she had reverted to her mute resistive state.

EEG

1 March. The patient was in a state of coma-vigil, but still showing active negativism. For example, when asked repeatedly to close her eyes, she kept them widely open, but when asked to open her eyes she would close them for up to half a minute at a time. Satisfactory recordings were obtained, showing a stable and mature alpha rhythm at 10 c.p.s. but paroxysmal slow activity at 3–5 c.p.s. occurred freely throughout the record (Fig. 1) having a generalized distribution, and suggesting a central origin.

2 March. This record was very similar to that of the previous day with a general all round reduction in amplitude. The patient was notably weaker.

3 March. Although the patient was by this time desperately ill, this EEG is the most "normal" of the three. The alpha rhythm is not yet disorganized, and the paroxysmal slow has disappeared. At this point the patient no longer displayed such clear cut active negativism.

Fatal Issue

Lumbar puncture on 1 March showed a clear fluid not under increased pressure. Cells 1/3 per c.mm. Globulin positive, protein 40 mg. per cent., chlorides (NaCl) 900 mg. per cent. Lange 0000000000. Blood sugar 110 mg. per cent. Blood urea 85–90 mg. per cent. The patient's course was steadily downhill and she died on 7 March.

Post-mortem report (Dr. B. E. Tomlinson). Death was probably due to acute pulmonary oedema precipitated by bronchopneumonia. The brain showed deep congestion over the entire cortex and there was a fairly marked tentorial pressure line over the uncus on each side. The vessels at the base of the brain were normal. There was no cerebellar pressure cone and no abnormality was revealed by a single horizontal cut through the cerebral hemispheres, exposing the lateral ventricles. Histological examination of numerous brain sections shows no evidence of encephalitis. Sections have been examined from the frontal, temporal, occipital and parietal lobes, from the uncus, thalamus, lentiform nuclei, hypothalamus, pons, medulla and aqueduct and beyond severe congestion with a very occasional pericapillary haemorrhage there is no significant abnormality. The meninges are also normal in appearance. The severe bronchopneumonia is confirmed, as is the fibrosis and

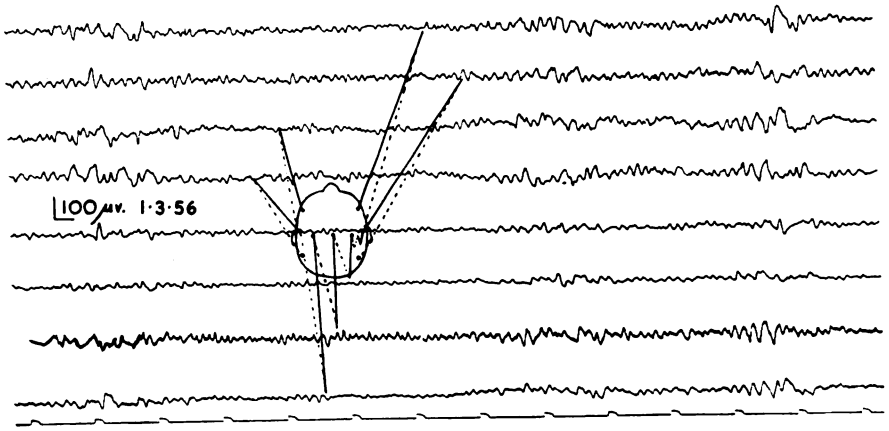

FIG. 1.—During stage of "active" negativism.

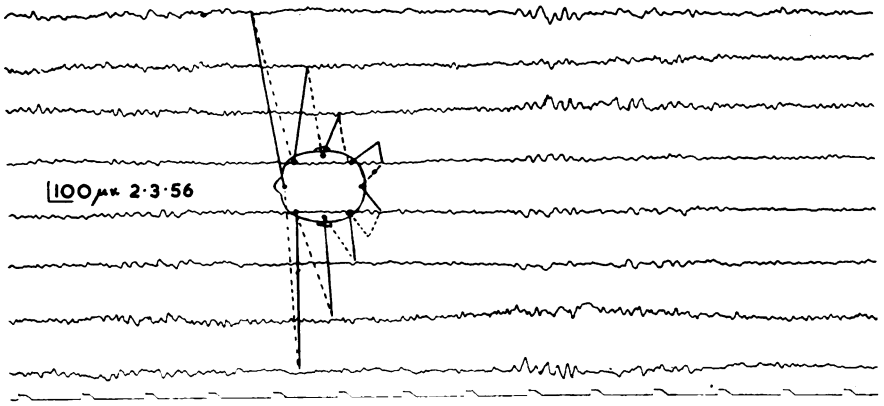

FIG. 2.—The next day. Negativism present but not so marked.

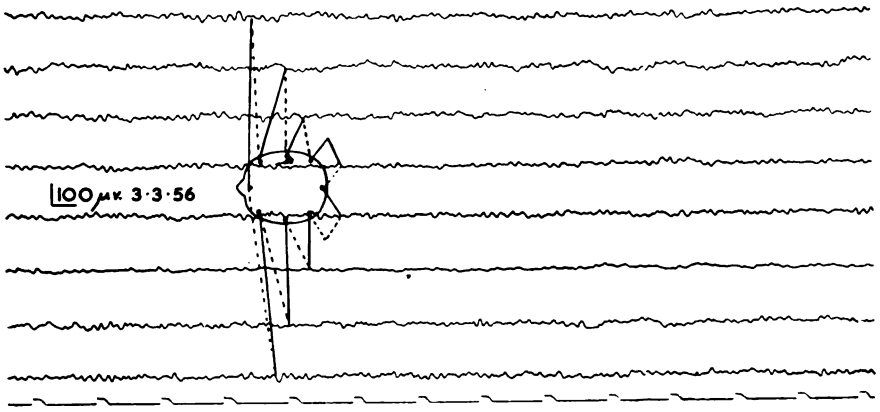

FIG. 3.—The following day. Patient extremely ill. Four days before death.

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mild inflammatory change around the diverticula in the colon. The liver, heart, and pancreas and bone marrow and suprarenals show no significant abnormality. There is some arteriosclerosis in the kidney, and in the pituitary several small colloid cysts in the pars intermedia. No adenoma is present.

Previous History

The patient suffered every winter from severe coughs and colds. She was always highly strung and excitable and whenever she got the slightest thing wrong with her, she always thought she was going to die. She was a great believer in patent medicines, and took much self-prescribed medication. There was a history of a mental illness in 1945, shortly after her husband was drafted overseas. She became very depressed, lay in bed all day, neglected her appearance, became careless and untidy in the house. She lived alone in a flat with her little girl at this time and believed the neighbours were against her and talked about her. She lived in constant fear and persistently believed that she was followed by men. She was awkward, unpredictable and difficult to deal with. She was actively hallucinated and would often scream out because she saw a man in her room. The illness lasted 2–3 months and she recovered spontaneously two weeks after going to live with relatives. No admission to hospital was necessary on this occasion. The family are convinced that the cause of this breakdown was anxiety over the absence of her husband.

Discussion

Fischer (1953) has suggested that the schizophrenic might have a genetically determined abnormally low threshold for stress. In this case there were two well-authenticated breakdowns, the first one apparently in response to a psychological stress, the second undoubtedly as a result of an infection. Gellhorn (1953) and Hoskins (1946) have attempted to describe schizophrenia in physiological terms, and Gerard (1955) postulates an inherited biochemical aberration as a dominant factor in the causation of schizophrenia.

Gjessing (1953) has repeatedly claimed that a disturbance of nitrogen metabolism in a fluctuating fashion accompanies the behavioural changes in episodic catatonia, and using neurosurgical material, Scharenberg and Brown (1954) describe amoeboid degeneration in astroglia from catatonics. They compare the appearances to similar changes brought about by severe disorders of metabolism—one of which is infectious disease. This seems to them to suggest a profound disturbance of metabolism in catatonic states. Yet the attempt to demonstrate a catatonigenic agent remains disappointing and recent workers have had to refute earlier successful claims, e.g. Edison (1956) using blood serum and urine of catatonic patients and Shapiro (1956) using cerebrospinal fluid have alike reported negative results.

The response to amytal is of course not new (Solomon, Kaufman and D'Elseaux, 1931; Smith and Schwartz, 1934; Thorne, 1938; Elkes, Elkes and Bradley, 1954), though it has more commonly been reported in chronic than acute states. The behavioural change was all the more striking here therefore and is taken as strong evidence that the diagnosis of catatonic schizophrenia was beyond doubt.

The EEGs confirm the clinical impression that catatonic stupor is a state somewhat between sleeping and waking (Mayer-Gross, Slater and Roth, 1954). The records were shown independently and without clinical history to two experienced electroencephalographers (J.W.O. and A.S.) and both agreed that sleep was the most likely explanation of the paroxysmal slow activity. Colony and Willis (1956) in a recent report of the EEGs of 1,000 schizophrenics found only three examples of activity at 1½–3 c.p.s. one of which was a severe acute catatonia. Their material was unusually valuable as it included 822 acute cases. From their results and a good review of the literature they conclude that EEG abnormality is rare in schizophrenia, possibly 5 per cent., and they suggest that schizophrenia is therefore not an organic disease. "Some of the people who become schizophrenic under some disintegrating psychological threat may have

had previously abnormal EEG patterns and the EEG abnormality noted as part of the clinical picture of the schizophrenia was either there to begin with, i.e. was coincidental, or was developed after the inception of schizophrenia as a result of either sequelae or intercurrent factors." It is very unlikely, they say, that EEG changes which are not static and which may change even in the course of the same examination are adequate evidence to support a genetically oriented or organic concept of schizophrenia. They see their results as supporting the view that it is a functional disease of psychological origin.

Conclusion

A case is presented showing the onset of acute catatonia as a sequela of middle lobe pneumonia. The history also shows a schizophrenic breakdown on a previous occasion apparently due to psychological stress. The characteristic response of catatonic schizophrenia to intravenous amytal is once again demonstrated. The state of active negativism is accompanied by changes in the EEG resembling sleep though the subject was "awake". Although the patient was dangerously ill from her pneumonia and for this reason E.C.T. was not given, yet in the end a fatal outcome occurred. It would seem therefore that in such cases, no matter how desperately ill the patient appears to be, it is probably not justifiable to withhold E.C.T. which often specifically terminates the state. "The hyper-acute catatonic patient with confusion, fever, and leucocytosis may very well die under one's hand . . . and three convulsion treatments on successive days or even more as a life saving measure may produce dramatic improvement" (Sargant and Slater, 1955).

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