Brit. J. Psychiat. (1967), **113**, 557–559

Correspondence

THE GRÜNTHAL-STÖRRING CASE OF AMNESIC SYNDROME

DEAR SIR,

In his discussion of this famed case B., Talland (1965) wrote that probably the last interpretation of this case had not yet been published. He was right, as the most competent analysis and interpretation given by Professor Zangwill in the *Journal* for February 1967 proves. Some brief remarks on Zangwill's paper may have some significance. The patient's attitude and responses, described in the reports published by Scheller and by Völkel and Stolze, are somewhat reminiscent of those of the patients described by this writer in 1956. My observations concerned patients involved in the process of claiming disability pensions to avoid being prisoners or being involved in criminal investigations. I labelled their condition as Ganser state, while recently Whitlock has mentioned the possibility "that a proportion of these cases could have been instances of hysterical pseudodementia rather than true examples of the Ganser syndrome". In my paper I also introduced the possible element of organicity.

Now the scope of interpretation of the pathogenesis in the case B. has been narrowed down by Zangwill almost to its limit, under the circumstances. However, I feel that the psychodynamic factors advocated by Syz (1936) and by Völkel and Stolze (1956), although not proven, may add to our understanding and approach to similar cases in the future. If speculation is at all permissible, one might come up with the following ideas:

1. Prior to his accident, the patient B. had been under considerable stress (venereal disease as a practising Roman Catholic; engagement to a much older woman). Was he not in great need of an escape into a psychoneurotic condition?

2. The patient was found unconscious at 1 a.m. at his place of work, apparently under the influence of escaping coal-gas. Was anything known in greater detail about the circumstances of this accident? Could the possibility be ruled out that the accident was in any way the result of the patient's conscious or unconscious need for an accident, not necessarily by his directly causing it but e.g. by his neglecting to take the usual precautionary measures, such as proper ventilation?

3. If these speculations are plausible to any extent, they would only parallel Zangwill's suggestion "that the hysterical elaboration of the memory defect may have arisen in the early stages of the illness within the context of an organic confusional state", in that the path for the developing symptom was pointed to by the usual memory loss due to transient brain damage.

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SIDE-EFFECTS OF PHENOTHIAZINES

DEAR SIR,

The "bogey" of the side-effects due to fluphenazine (moditen) appears to me to be exaggerated. In his review of almost 4,000 patients on oral phenothiazines, Ayd (1) found an incidence of extrapyramidal side reactions in 60 per cent of patients taking trifluoperazine (stelazine) and in 52 per cent of patients taking fluphenazine. None of these patients were taking any anti-parkinson drug. Thus trifluoperazine in such circumstances has a similar incidence of side-effects of this nature to fluphenazine, despite the fact that the latter is a far more potent phenothiazine.

With regard to the increased incidence of dystonic reactions when phenothiazines are given parenterally, I think one must differentiate between short-acting parenteral phenothiazine preparations and, as far as I know, the only long-acting one—namely fluphenazine enanthate. My experience has been mainly with the latter, and in a small series of some 68 patients treated with this drug in the past 14

months I have had only two cases of dystonia—both of which occurred at some considerable time after starting the drug, one 4 weeks and the other 4 months after commencing treatment.

I should like to add to Dr. Simpson's comments (March 1967, p. 331) on Dr. Barker's statements about the onset of later extrapyramidal effects and that the patient who would not take oral phenothiazines is likewise unlikely to take anti-parkinson drugs. If a patient is being treated with fluphenazine enanthate presumably a physician is seeing the patient reasonably often, and even should long-term side-effects appear they would be detected early and at a stage when they are likely to be reversible.

More important, I would consider, is the effect of motivation. My observations of patients who stop taking oral phenothiazines would suggest that most fall into two groups: those who consider they are cured and require no further treatment now that they have lost their symptoms, and those who have no insight into their condition. The number of patients who have complained of side-effects as a reason for stopping treatment has, in my experience, been small. The motivation of such patients to take phenothiazines is small, whereas a patient on fluphenazine enanthate who gets a reaction to the drug has a much greater motivation to seek medical advice, as Dr. Simpson points out.

I agree with Dr. Simpson's sentiment concerning the usefulness of a long-acting anti-parkinson drug, a point I raised in the correspondence columns of the *B.M.J.* (2) recently when discussing fluphenazine enanthate. Even in the absence of such a drug I would support Dr. Simpson's plea that the side-effects of fluphenazine enanthate be viewed dispassionately and not be allowed to prevent its effective use in treating the "chronic relapsing schizophrenic", to whom this form of treatment would appear to be most useful.

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ANAEMIA AND RETICULUM CELL HYPERPLASIA IN SCHIZOPHRENIA

DEAR SIR,

In her article (*Journal*, December 1966, p. 1285) Dr. Beattie described ten cases of blood and lymph gland dyscrasias among 210 autopsy cases with a diagnosis of schizophrenia, which contrasted with only seven such disorders found in 1,216 post-mortem examinations of persons with mental disorders other than schizophrenic reactions. This is a very interesting finding, and Dr. Beattie offers two possible explanations for the observed cellular defects, (1) Kallmann's theory of a hereditary reticulum cell inadequacy associated with schizophrenia, and (2) the hypothesis of the presence of a noxious metabolite in schizophrenic patients.

Dr. Beattie does not mention drug medication, in particular phenothiazine derivatives, which could have played an important role in the aetiology of the observed myeloproliferative disorders. Dr. Beattie's period of investigation essentially spans the entire phenothiazine drug era. Blood dyscrasias following medication by phenothiazine or its derivatives have become commonly known. It has been shown that these compounds can affect among other cells all corpuscular elements of the peripheral blood and their precursors in the hematopoietic tissues. It would be valuable to analyse the pathological data Dr. Beattie collected from the viewpoint of abnormal drug response.

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THE LATE DR. HILDA LEWIS

DEAR SIR,

For many years Dr. Hilda Lewis took a keen, active and practical interest in adoption. She gave generously of her time and energy in this cause as paediatrician and psychiatrist, as Medical Adviser to the Children's Society, as member of the Executive Committee of the Standing Conference of Societies Registered for Adoption, as writer, and as counsellor on adoption matters to a wide variety of adoption societies and other bodies. She played a leading part in the establishment of the Medical Group of the Standing Conference of Societies Registered for Adoption—devoted to improving adoption medical practice—and was its first Chairman.

The Medical Group, in conjunction with the Standing Conference of Societies Registered for Adoption, proposes to establish a Dr. Hilda Lewis Memorial Fund with the object of endowing a Lecturership in her name. Lectures by distinguished workers in the field of adoption will be given from time to time at the Annual General Meeting of the