

be a state prisoner, who must not be treated so hard... (Gruner, 1789).

These strongly condensed translations only yield a dim impression of the original vivid and lengthy reports. They may perhaps cast some doubt on the issue that hallucinatory voices did not vex schizophrenics before 1800.

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SIR: I was intrigued to read some of the observations of Hare (*Journal*, October 1988, 153, 521–531) about accounts of schizophrenia in the late 18th century. It is noteworthy that frequent, good descriptions of schizophrenia (and several other chronic mental illnesses) first appeared when there was a change in the selection of cases for publication, combined with improved methods of case description. This change probably explains the appearance of these descriptions.

The only description of cases of chronic madness from between 1750 and 1810 are those of Thomas Arnold (1782), who only quotes cases from classical authors, Perfect (1787), Haslam (1798, 1809) and Ferriar (1810). The other published case histories are all of people who recovered. They were published by private madhouse proprietors, who were not keen to publicise their failures. This 100% recovery rate shows that most of the published cases do not form a representative sample of the mental illness then prevalent, especially when compared with the cure rates contemporarily advertised of 30%.

Only about 25% of the large series of cases cited by Perfect (1787) were chronic. These chronic cases appear to have been published to illustrate points that Perfect wished to illustrate, such as the dangers of inexpert treatment with mercury, his skill with healing severe wounds, and the danger of trusting lunatics. Despite this, at least two cases he cites could be examples of schizophrenia (cases 8 and 36).

Chronic cases were first cited in quantity by Haslam and Ferriar, who both worked in charity asylums. The cases cited in Haslam's 1809 edition

that are generally accepted to be probable cases of schizophrenia were copied from the 1798 edition (e.g. cases 16 and 23 in both editions), and some of these cases had been admitted 30 years earlier. Unfortunately, Haslam's series of cases must have been biased towards organic cases because it is a series of post-mortems. Ferriar's case descriptions were very brief, and therefore of limited use.

Interestingly, the classificatory systems of mental illness that were published during this period all have categories that would accommodate modern images of schizophrenia: Arnold (1782) in his description of *Ideal insanity*, which includes people who imagine they are conversing with imaginary people, but who are not delirious; Cullen (1810) in his group of *Manias* (and in his classification of *Paracusis imaginaria* – where the sounds are not existing without, but are excited by internal causes, and are distinguished from false hearing); and Darwin (1801) in his species *Mania mutabilis*.

Regarding Harper's claim that insanity was not common in young persons: it is noteworthy that this was part of his argument that insanity was not due to brain disease, but to nervous stimulation and the retention of secretions such as semen. This was a highly unusual view for the period. This compares with the fact that the cases described by Perfect, Ferriar, and Haslam were frequently young.

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Elementary, My Dear Freud

SIR: I rise to defend Rollin (*Journal*, August 1988, 153, 241–242) against the unjust criticism of Johns (*Journal*, November 1988, 153, 712), who uses a succession of specious arguments. Firstly, Dr Johns quotes and implicitly agrees with Holmes' remark that "It is a capital mistake to theorise before one has data". At a stroke, one of the lynchpins of the

scientific process is dismissed. Normally, a null hypothesis is formulated and data is collected to reject this null hypothesis. Sherlock Holmes may be excused his position because tests of significance and modern hypothesis testing had not been developed in Conan Doyle's day, but there is no such excuse for Dr Johns.

Secondly, Dr Johns sweeps aside the practice of making assessments of both fictional and historical figures, and states that if such assessments lead to a diagnosis, it "is to severely debase the term". On the contrary, it is perfectly legitimate to consider the maladies of such personages. The medical and literary world would be impoverished if discussion of Beethoven's deafness or Hamlet's madness were dispensed with. In addition, Dr Johns has a particularly narrow view of literature when he says, "Our appreciation and enjoyment . . . are not increased by psychiatric post-mortems". Indeed, one of the purposes of literature is to encourage us to think, and I wonder if Dr Johns is familiar with the myriad volumes written on literary criticism, much of which deals with psychological issues and which by its very nature is a 'post-mortem'.

Sherlock Holmes may well show features of obsessional neuroticism, and Dr Rollins adduces convincing textual evidence to support his case. Dr Johns' comment that "He complained of no mental illness" is insufficient to disprove Dr Rollins' interpretation. It is well known that there are those who have psychiatric symptoms but deny them, and so the concept of insight seems to have escaped Dr Johns.

Not only are Dr Johns' arguments flawed but so also is his English. As an example, the following sentence defies comprehension: "Lest anyone thinks I am unduly critical of a pleasant literary piece, the Sunday Times of 21 August commented on the article under the headline 'He was quite a case' ". It is clear that Dr Johns would benefit from a greater study of the arts not only to elicit a more mature response to literature, but also to improve his English.

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CT Studies of Schizophrenia

SIR: The interesting findings of Smith *et al* (*Journal*, 1988, 153, 667-674) stimulated me to look at our computerised tomography (CT) findings in the context of the influence of different control-matching in our patient subsamples.

We examined 150 RDC-diagnosed chronic schizophrenic patients from mental hospitals with CT

(Mundt, 1985). Due to personnel problems we had to carry out the CT scans in two different centres: group 1 (71 patients) was examined at the Surgical University Hospital, Heidelberg, and group 2 (73 patients) in the Central Institute of Mental Health, Mannheim. Among the CT measures taken were the III ventricle diameters, the cella media index, and the number of enlarged sulci. For both groups of patients, control groups were recruited as coupled pairs matched for sex, age, and skull diameter. The controls for group 1 were taken from the Medical University Hospital, Heidelberg; those for group 2 from selected physically healthy neurotic patients at the Central Institute Mannheim. We used identical restricted exclusion criteria for both control groups. The results of a comparison within the matched pairs differed between the groups: group 1 displayed no significant differences whatsoever between patients and controls; group 2 showed slightly but significantly larger III ventricles in the schizophrenic patients than in the controls.

In order to better understand this inconsistency we used the following procedure: first we compared group 1 and group 2 patients according to age and sex distribution; no difference was found. Then we calculated a correction for the matrices of the two CT scans, and corrected all the values for the III ventricle diameters in group 1 and group 2 for both the patients and the matched controls. It turned out that the inconsistency was due to the very different values of the controls; the values for groups 1 and 2 are close, and lie between those for the two control groups. We found that the controls from the Medical Hospital showed larger ventricles than the selected control group of physically very healthy neurotics from the Central Institute.

These results raise the question of which control-sampling can be considered to be most adequate for a group of chronic schizophrenic patients. Obviously we know little about non-illness-related factors which may influence the width of the III ventricle and other CSF spaces and so contribute to the considerable variance of these measures in the general population.

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Reference

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