mental self from that of others, the loss of the distinction between the sign and the signified and the loss of the connection between intention and motility (splitting). All these deficits (negative symptoms) imply a loss of 'the voluntary' allowing the expression of the positive symptoms, that is the automatic and repetitive ego-alien modes of thought (delusions) perceiving (hallucinations) and motility (negativism, catalepsy).

According to Jackson (1894) disease does not create, it sets free. Ey (1962, 1969) has used this principle as the basis for his statements—first, that the structure of mental disease is basically negative or regressive and second, that mental diseases represent levels of dissolution of the psychic organization. When schizophrenic symptoms are evaluated in this way it is clear that positive symptoms are dependent for their expression on negative symptoms. The former only seemingly predominate during acute attacks while the latter are present at all times during the illness (Bleuler, 1978). To avoid confusion, it might have been better if Crow and Wing had chosen some other means of describing the clinical phenomena which they believe to be of importance for the ordering and understanding of schizophrenic symptoms.

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References

BLEULER, M. (1978) *The Schizophrenic Disorders: Long-term Patient and Family Studies*. New Haven: Yale University Press.

CROW, T. J. (1980) Positive and negative schizophrenic symptoms and the role of dopamine. *British Journal of Psychiatry*, **137**, 383–6.

—— (1981) Positive and negative schizophrenic symptoms and the role of dopamine. *British Journal of Psychiatry*, **139**, 251–4.

EY, H. (1962) Hughlings Jackson's principles and the organo-dynamic concept of psychiatry. *American Journal of Psychiatry*, **121**, 673–83.

—— (1968) Outline of an organo-dynamic conception of the structure, nosography and pathogenesis of mental diseases. In *Psychiatry and Philosophy* (eds. E. W. Straus, M. Natanson and H. Ey). Berlin: Springer-Verlag, 111–61.

JACKSON, J. HUGHLINGS (1894) The Factors of Insanities. In *Selected Writings*, Vol. 2, p. 411. London: Staples, 1959.

LEVIN, M. (1936) On the causation of mental symptoms: An enquiry into the psychiatric application of Hughlings Jackson's views on the causation of nervous symptoms with particular reference to their application to delirium and schizophrenia. *Journal of Mental Science*, **82**, 1–27.

WING, J. K. (1978) Clinical concepts of schizophrenia. In *Schizophrenia: Towards a New Synthesis* (ed. J. K. Wing). London: Academic Press, 1–30.

INSTITUTIONALIZATION AND THE DEFECTS OF SCHIZOPHRENIA

DEAR SIR,

Am I alone in noting a number of inconsistencies in the recent evidence presented by the Northwick Park group in support of their hypothesis that the schizophrenic 'defect state' has a structural rather than a biochemical basis?

Owens and Johnstone (1980) found significant correlations between cognitive dysfunction, negative schizophrenic symptoms, poor behavioural performance and neurological abnormalities in a group of chronically hospitalized schizophrenic patients. In their later study (Johnstone *et al*, 1981) involving a parallel survey of schizophrenic outpatients, they claimed to find similar, and similarly correlated, deficits once allowance had been made for age and length of illness. On this basis, they concluded that the spectrum of deficits reflected the schizophrenic disease process rather than arising as a consequence of institutionalization.

I feel that some of their conclusions, especially those involving comparisons between in-patient and out-patient groups, are questionable on the following grounds. Firstly, although no significant differences were found in behavioural performance between the in-patient and out-patient groups, the ratings of the former were made by nurses and those of the latter by relatives. It is thus far from clear that direct comparisons of the ratings are legitimate. Secondly, while neurological deficits were present to a similar degree in both groups, they only correlated significantly with the cognitive and behavioural deficits and the negative symptomatology in the in-patient groups; and indeed apart from extra-pyramidal syndromes (explicable at least plausibly in biochemical terms) neurological abnormalities were very rare in both groups. Thirdly, and perhaps most importantly, cognitive deficits were highly significantly ($P < 0.001$) less severe in the out-patient group even after correction for age and duration of illness. Furthermore, the highly significant ($P < 0.001$) correlation in the in-patient group between negative symptoms and cognitive deficits cited particularly by Crow (1981) in support of his argument for the existence of a structurally based 'defect state', was significant at only the $P < 0.05$ level for the out-patient group.

It seems therefore that the spectrum of disability described by Owens *et al* (1980) has only been demonstrated for their in-patient group. This can perhaps most economically be explained as forming the very findings to be expected in that group of chronically institutionalized patients too disabled to have achieved discharge from hospital. The striking discrepancy in cognitive deficits between the two groups may,

indeed, constitute evidence against the existence of a structurally based schizophrenic 'defect state'.

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References

CROW, T. J. (1981) Positive and negative schizophrenia symptoms and the role of dopamine. *British Journal of Psychiatry*, **139**, 251–4.

JOHNSTONE, E. C., OWENS, D. G. C., GOLD, A., CROW, T. J. & MACMILLAN, J. F. (1981) Institutionalization and the defects of schizophrenia. *British Journal of Psychiatry*, **139**, 195–203.

OWENS, D. G. C. & JOHNSTONE, E. C. (1980) The disabilities of chronic schizophrenia—their nature and the factors contributing to their development. *British Journal of Psychiatry*, **136**, 384–95.

DEAR SIR,

May we make the following points in reply to the above letter. Firstly, Dr Katona appears to have confused two questions (a) the relationship between the defects of chronic schizophrenia and institutionalization and (b) the relationship between the defects of chronic schizophrenia and structural abnormalities of the brain. Our recent paper (Johnstone *et al*, 1981) deals with the former question and does not concern structural abnormalities of the brain at all. Further investigations of this question are in progress. It should be made clear that the Current Behavioural Schedule is completed by the interviewers on the basis of information given by the persons regularly caring for the patient. Thus the findings in the institutionalized and non-institutionalized samples are as comparable as it is possible for them to be.

The question of whether the poorer cognitive performance in the institutionalized patients is (a) premorbid, (b) an early effect of the schizophrenic process which has precluded discharge or (c) a late effect associated with institutional care, has been discussed at length in the paper. One of the features of this study is that the non-institutionalized patients who were selected from a total sample of schizophrenics as having been fit for discharge and continued management outside hospital would be expected to have performed better at some stage.

The findings of the study show however that this advantage over the institutionalized sample has now been lost except with regard to the cognitive performance. Dr Katona has emphasized the differences between our samples but overall the similarities are more impressive and we consider that the results of this study provide good evidence that the defects of chronic schizophrenia are not due to institutionalization.

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References

JOHNSTONE *et al* (1981) Institutionalization and the defects of schizophrenia. *British Journal of Psychiatry*, **139**, 195–203.

Dr Crow writes further as follows:

DEAR SIR,

With respect to the question raised by Dr Katona of whether the 'defect state' is associated with structural changes in the brain, a summary of the findings of those radiological studies of schizophrenia in which a relationship with some feature of the clinical state has been demonstrated is of some interest (see Table). Included in this Table are 1 echoencephalographic and 9 recent CT scan studies together with the numbers of patients included and the main structural abnormality detected (i.e. of the third or lateral ventricles or cerebral cortex) and the associated clinical variable.

Features which have been found to correlate with structural anomalies in at least two studies are intellectual impairment, treatment resistance, and negative symptoms. Positive symptoms are conspicuous by their absence. Therefore, there is at least an a priori case that structural changes in the brain in schizophrenia for which there is now a substantial body of evidence, are associated with the defect state or what we have called the Type II syndrome (Crow, 1980; Crow *et al*, 1980).

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References

ANDREASEN, N. (1981) CT Scan abnormalities in schizophrenia: relationship to positive and negative symptoms. Paper presented to the Royal College of Psychiatrists Annual Meeting, July 1981.

CROW, T. J. (1980) Molecular pathology of schizophrenia: more than one disease process? *British Medical Journal*, **280**, 66–8.

—— FRITH, C. D., JOHNSTONE, E. C. & OWENS, D. G. C. (1980) Schizophrenia and cerebral atrophy. *Lancet, i*, 1129–30.