

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

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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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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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TABLE
Structural changes in schizophrenia—association with clinical state

		n	Abnormality	Association
Holden <i>et al</i> , 1973	Echo-encephalogram	65	IIIrd vent.	Treatment resistance
Johnstone <i>et al</i> , 1976	CT scan	18	lat. vent.	Intellectual impairment (Withers and Hinton) ? negative symptoms
Rieder <i>et al</i> , 1980	CT scan	17	lat. vent.	Intellectual impairment (Halstead-Reitan)
Donnelly <i>et al</i> , 1980	CT scan	15	lat. vent.	Intellectual impairment (Halstead-Reitan)
Golden <i>et al</i> , 1980	CT scan	42	lat. vent.	Intellectual impairment (Luria-Nebraska)
Weinberger <i>et al</i> , 1980a	CT scan	51	lat. vent.	Poor pre-morbid adjustment
Weinberger <i>et al</i> , 1980b	CT scan	20	lat. vent.	Treatment resistance
Gross <i>et al</i> , 1981	CT scan	117	IIIrd vent.	'pure defect' state
Andreasen, 1981	CT scan	52	lat. vent.	negative symptoms impaired sensorium
Takahashi <i>et al</i> , 1981	CT scan	169	IIIrd vent. + lat. vent. + cortex	blunted affect unco-operativeness