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ANTIPARKINSONIAN MEDICATION IN DEPRESSED SCHIZOPHRENICS ON DEPOT NEUROLEPTICS

DEAR SIR,

Recent work has confirmed the need for antiparkinsonian medication in neuroleptic treated schizophrenia (Manos *et al*, 1981) and suggested that 68 per cent of patients given placebo developed extrapyramidal side-effects. In another report Johnson (1981) indicated a tendency for depressive-type symptoms to improve when these patients were given orphenadrine 50 mg twice daily. However, he considered that some of the symptoms may have been neuroleptic drug-induced akinesia rather than depressive in origin.

This report relates to the outcome of a single blind cross-over study comparing sustained release benzhexol (Artane Sustets) with procyclidine hydrochloride in schizophrenia treated with depot neuroleptics. The results from 33 patients are available. They consist of 18 men and 15 women with a mean age of 47 years who were receiving depot neuroleptics for chronic schizophrenia. Nineteen patients had been prescribed fluphenazine decanoate and 14 fluphenthixol decanoate. Before entry into the study the patients were not receiving antiparkinsonian drugs. On recruitment the patients were either prescribed sustained release benzhexol 15 mg as a single morning dose or procyclidine hydrochloride 5 mg 3 times daily. On completion of 7 days' treatment the patients prescribed benzhexol were given procyclidine for 7 days and those commenced on procyclidine were given benzhexol. Extrapyramidal signs and symptoms were recorded on day 0, 7 and 14 on a 4 point scale as being absent, mild, moderate or severe. The data analysis showed that the 2 groups of patients were comparable at entry to the study and that there was no significant difference between benzhexol and procyclidine in the management of rigidity, tremor or akathisia. Both drugs were found to be equally effective.

Of 9 patients in the benzhexol-treated group reporting depressive symptoms initially, 7 were found to be improved after one week as compared with only 4 of 9 patients on procyclidine. This result, though interesting, did not reach statistical significance, but the trend observed here accords with that noted by Johnson in patients treated with orphenadrine. Could it be that there is a case for offering schizophrenic patients on neuroleptics an antiparkinsonian rather than antidepressant drug when they present with depressive type symptoms?

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POSITIVE AND NEGATIVE SCHIZOPHRENIC SYMPTOMS

DEAR SIR,

Crow in his discussion papers (1980, 1981), and Wing (1978), have not been explicit about their use of the concept of positive and negative symptoms. This concept has its origins in Hughlings Jackson's theory of evolution and dissolution of the nervous system (Jackson, 1894; Levin, 1936). Crow and Wing designate delusions, hallucinations and motility disorders as positive symptoms, believing that they predominate in acute attacks, whether at onset or later. Volitional defect, withdrawal, and flattening of affect are described as negative symptoms and they predominate in the quiescent phases of the chronic stage of the illness. The two categories of symptoms are presumed to reflect ". . . different underlying pathological processes" (Crow, 1980), within the group of schizophrenias. Supporting evidence is afforded by different responses to drug therapy and by other physical measurements.

Positive symptoms in the Jacksonian sense are the result of damage to healthy mental life. This damage leads first to negative symptoms. They are to be found during acute attacks, in the loss of selective attention, the loss of the capacity to discriminate the bodily and

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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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,