S34-4
NEUROLEPTICS: ARE THEY EFFECTIVE IN AGGRESSION?

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The neuroleptic group of drugs has been employed in the treatment of aggression and behavioural disturbance ever since they were first marketed, hence their alternative name, major tranquillisers. Numerous studies have shown that these drugs are effective in the control of acute behavioural disturbance irrespective of whether this is due to a psychotic process or not. Nevertheless, despite their demonstrable effects in reducing aggression the delay in their onset of action, even when given parenterally, means that alternative agents, such as rapidly-acting benzodiazepines are often given concurrently or separately for emergency treatment.

The value of these agents in chronic aggressive disturbance is much less well researched. Although these drugs are widely employed, particularly in the control of aggression in people with mental retardation, there is meagre evidence from randomised controlled trials that these agents are effective. Recent work has shown that risperidone and zuclopenthixol have an ameliorating effect in chronic aggression although the effect size of the trials carried out was only small.

The relative value of the neuroleptic drugs compared with other agents in the treatment of chronic aggression is not yet established. Controlled studies need to be carried out to compare the value of the newer antipsychotic drugs with lithium, β-adrenergic blocking drugs, anticonvulsants and antidepressants.

S34-5
THE EXPERIMENTAL MEASUREMENT OF HUMAN AGGRESSION

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Aggressive behaviour is an increasing problem so there is a need to seek effective treatments. Drugs can both increase and decrease aggression. Violence is often associated with drug abuse but newer drug treatments can help to reduce the incidence of aggressive acts without producing excessive sedation. There are 3 main ways of studying behaviour: by observation, by report and by manipulation. Commonly used methods of recording aggression are observer scales and self-report inventories but standardised experimental laboratory techniques have also been developed. Six methods have been identified, ranging from group dynamics to competition with provocation. The objective of the reported studies was to evaluate the effects of manipulating serotonin on anger and aggression measured in the laboratory.

Methods: Both a competitive reaction time task and a co-operative computer game were used to provoke aggression experimentally. In two independent studies, an amino acid drink, either enhanced or depleted with tryptophan, was administered according to a double blind independent groups design to high hostility male subjects. Five hours later they took part in a task. Blood samples were taken to determine plasma tryptophan values and rating scales were completed pre drink and pre and post task.

Results: Subjects in the tryptophan-depleted groups reported more anger and responded more aggressively than those in the enhanced groups.

Conclusion: The results confirmed other sources of evidence that the neurotransmitter, serotonin, is involved in aggression. Laboratory paradigms can be useful in assessing the effects of drugs on aggression. It is suggested that a combination of techniques is most useful in drug studies in order to try and identify the mechanisms by which aggression is being altered.

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FC36-1
SYMPTOM DIMENSIONS, CLUSTERS AND SUBTYPES OF SCHIZOPHRENIA PRIOR TO AND 5 YEARS AFTER FIRST ADMISSION

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The study investigates the symptom dimensions and the formation of empirical subgroups of patients in the prodromal phase, the psychotic prephase, at index admission and at different cross-sections up to 5 years after admission. It is based on a population-based sample of 232 first-episode schizophrenics (Mannheim ABC-Schizophrenia-Study) with a clinical ICD-9 diagnoses of 295., 297., 298.3 and 298.4. The symptomatology is measured with the SANS, the PSE and the "Instrument for the Retrospective Assessment of the Onset of Schizophrenia" (IRAOS).

Compared with Crow's dual process model and Andreasen's bipolar model, Liddle's three factor model (positive symptoms, negative symptoms, disorganisation) fitted in best with the data at first admission (confirmatory factor analysis). Liddle's model could also be replicated at all 5 cross-sections over the 5-year follow-up. While the negative dimension is stable over the 6 cross-sections, positive dimension and disorganisation are not. There were no association between these three dimensions and sex, age at onset and type of onset, but an association between genetic load and disorganisation and between obstetric complications and negative symptoms was found. The negative symptoms correlated positively with social disability and negatively with social development after 5 years. The clustering of the 232 patients into different subgroups resulted in 5 clusters for the prodromal phase and 6 clusters for the psychotic prephase. For the psychotic prephase the clusters are determined through single dimensions like delusion, psychotic thought disorders, negative symptoms, hallucinations and disorganisation, while in the prodromal phase the 5 clusters are determined through more than one dimension. The stability from the prodromal phase to the psychotic phase is moderate. The six clusters of the psychotic prephase had no predictive value for symptomatology, social disability and objective indicators of social development over the 5 year-period.

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