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# Correspondence

*Letters for publication in the Correspondence columns should be addressed to:*

**The Editor, British Journal of Psychiatry, 17 Belgrave Square, London SW1X 8PG**

## GRID TEST OF SCHIZOPHRENIC THOUGHT DISORDER

DEAR SIR,

A. B. Hill's cogent paper on the validity and clinical utility of the Grid Test of Schizophrenic Thought Disorder (*Journal*, March 1976, **128**, p 251) fairly argues that statistical validity does not necessarily imply clinical utility. But then does 'clinical judgement' necessarily have any 'clinical utility' if it is accepted that clinical utility refers to the capacity of a procedure to be helpful to patients? Does it help patients to designate them 'thought disordered schizophrenics' whether we do this by clinical judgement or by grid test? I suggest that it probably does not—certainly A. B. Hill has not sought to find out which kind of judgemental procedure is most helpful to the patient. He has arbitrarily assumed that clinical judgement is to be *criterion* 'because it is difficult to find an alternative' and the grid test is to be *predictor* and be evaluated purely in terms of its correlation with clinical judgement.

The only virtue in the grid is that it has what Hill refers to as a 'clear and appealing rationale', whereas clinical judgement has little by way of rationale, it is a descriptive response which says nothing as to the 'why' or the 'how' or the 'what do we do about' of schizophrenic thought disorder.

Till the day when either grid or clinical judgement provides us with an argument from which we can derive a way of helping the so-called thought-disordered schizophrenic (such as was attempted by Bannister *et al*, 1975), neither grid nor clinical judgement has 'clinical utility'. They both do no more than provide bases for exploration.

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## PSYCHOSIS DUE TO NASAL DECONGESTANT ABUSE

DEAR SIR,

The schizophrenia-like reactions caused by abuse of amphetamines and related substances are well known. In 1964 amphetamine sulphate ('Benzedrine') was withdrawn as a constituent of nasal decongestant preparations because of these effects. It was replaced by propylhexedrine, a sympathomimetic agent with similar chemical structure and vasoconstrictor properties but with little stimulant action on the central nervous system. I wish to report a psychotic reaction due to abuse of the propylhexedrine inhaler, 'Benzedrex'.

A 24-year-old single female student had for a year found increasing difficulty in concentrating on her course and had failed to sit class examinations. She felt continually tired. Her parents found her withdrawn and depressed. She presented to the psychiatric department on 9 October 1975 shortly after becoming delusionally convinced that she was radioactive and that radio and television were 'bugged' and were watching her. There was blunting of affect but no evidence of visual or auditory hallucinations. She had no insight into her condition. She had a successful record at art school and no history of psychiatric illness. However, both an elder sister and a maternal uncle are schizophrenic.

An initial diagnosis of schizophrenia was reconsidered when the patient admitted later to having taken one or two 'Benzedrex' inhalers (each 250 mg) a day for much of the previous two years, removing and chewing the contained propylhexedrine-impregnated strip. This gave her 'a lift' with the impression of more efficient studying. When the effect wore off she was left tired and lethargic. She had occasionally experimented with cannabis, but never for any length of time.

Following admission to hospital the symptoms at first persisted despite chlorpromazine therapy. However, an illicit source of inhalers was discovered, and when this was finally stemmed she made a gradual

recovery without further treatment over two weeks, losing all delusional features and regaining normal emotional expression. No withdrawal symptoms were noted. She was discharged from hospital on 8 January 1976 and remained well when seen as an out-patient four weeks later.

Similar psychosis from abuse of 'Benzedrex' inhalers has previously been reported on three occasions in a total of four patients (1, 2, 3). Each had chewed the propylhexedrine strip for its stimulant effect. Three had continued the habit for several months, while the fourth consumed the contents of eleven inhalers in ten days. The clinical features in each case resemble those of amphetamine psychosis, with variable auditory and visual hallucination, paranoid delusions, loss of affect, difficulty in concentration, and sleep disturbance. In all previous reports there is a past history of psychiatric illness—two cases of amphetamine psychosis and one each of manic-depressive psychosis and schizophrenia. This and our patient's family history of schizophrenia suggest the possible uncovering of a latent schizophrenic tendency, as is sometimes thought to be the case in amphetamine psychosis. There was no evidence of schizophrenia following recovery in this case.

'Benzedrex' inhalers are readily available over the chemist's counter. Our patient was in the habit of attending several shops to obtain the necessary supply. Like previous addicts, she started the practice at the suggestion of others who had experience of it, rather than through its use as a decongestant. Abuse, although probably limited, clearly does occur. 'Anahist', a proprietary preparation containing the sympathomimetic phenylpropanolamine, has also been reported as causing a psychotic reaction (4). Non-scheduled preparations must still be considered in the differential diagnosis of psychotic states.

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## MAINTENANCE THERAPY IN CHRONIC SCHIZOPHRENICS

Dear Sir,

Dr Johnson's account (*Journal*, March 1976, p 246) on the relatively high relapse rate of schizophrenic patients treated by long-acting injectable neuroleptic drugs leads me to remark that since we, in this area, started this type of treatment in 1966 it has been the universal practice for a community nurse to visit patients at home where they have their injection. This has produced a refusal rate which has averaged 4 per cent over the years and which is, I believe, rather lower than can be achieved by encouraging patients to come to clinics to have their injections. Although such a method may appear expensive, the money saved by keeping patients out of hospital more than pays for the extra nurses' salaries.

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## THE POWER OF A TEST FOR SEASONALITY OF BIRTH WITH REFERENCE TO SCHIZOPHRENIA

Dear Sir,

It seems to be well established that the births of people who later develop schizophrenia occur seasonally (Dalén, 1975; Hare, 1975). The cause of this is unknown and more work will be needed to test

(a) how widespread this phenomenon is, and
(b) whether a similar phenomenon exists in regard to the sibs of schizophrenics.

In testing these points, researchers may wish to know the power of their procedures to detect, at statistically significant levels, seasonality of the same magnitude in further samples. Hare (1975) considers the size of the sample necessary to detect the effect at the 5 per cent and 1 per cent levels: these sample sizes vary according to the chance we wish to have of detecting the effects (the 'test power'). It is conventional to set test power at 0·8 (Cohen, 1969, p 51): in other words, we want our test to have 4 chances in 5 of detecting the effect at a preset level of significance. The four parameters (1) test power, (2) significance level, (3) 'effect size', and (4) sample size are interrelated: if three are set, the fourth can be evaluated. We may set test power at ·8, and the significance level at ·05. The effect size is defined (Cohen, 1969, p 210) as

$$e = \sum_{i=1}^{m} \frac{(P_{1i} - P_{oi})^2}{P_{oi}}$$

where

$P_{oi}$ is the proportion in cell $i$ posited by the null hypothesis, $P_{1i}$ is the proportion in cell $i$ posited by the alternative hypothesis and reflects the effect for that cell, and $m$ is the number of cells (four for quarters of the year).

Now Hare (1975) suggests that the seasonality has a deviation of about 8 per cent. Let us accordingly