endogenous or exogenous (e.g. amphetamine or bromocriptine), we may all be liable to develop psychotic symptoms. Thus a *sine qua non* of psychoses would be excessive dopamine, but a secondary susceptibility would also be required. Complementary research into the genetic and neurochemical aspects of such symptoms need not, therefore, be dissociated from allowing dopamine a central role in the generation of psychiatric illness.

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Imipramine Versus Phenelzine in Melancholias and Dysthymic Disorders

SIR: At a recent statistics seminar for students studying for Membership of the Royal College of Psychiatrists, the recent clinical trial by Vallejo *et al* (*Journal*, November 1987, **151**, 639–642) was discussed. It became apparent that the study was defective in a number of ways, so that the conclusions are difficult to support, and I feel it necessary to report some of the problems.

(i) The study is in fact two clinical trials, one for patients with melancholia and one for patients with dysthymic disorders. There were 32 patients in each trial. With this size sample, if 50% of patients improved on one drug, one would need a 95% improvement on the other to obtain a significant difference between the drugs at the 5% significance level with 80% power, giving a wide range in which to conclude that for imipramine and phenelzine 'patients responded equally well to both drugs'. In other words, the trial lacks power to conclude that the drugs were equivalent. This is clearly a case where confidence intervals should be given.

(ii) There is a statistical blunder in that the authors show that the variance of HRSD scores differs between imipramine and phenelzine (by 'Snedecor's test', which should have been referenced) and then proceed to compare means using the *t*-test. In fact, one of the assumptions underlying the validity of the *t*-test is that the variances are equal. Also, since the mean and standard deviation of the HRSD score are of similar size it is clear that the data are highly skewed. It would have been better to (a) give a graph of the data and (b) try a logarithmic transformation to see if this stabilised the variability. The graph would show the distribution of the data, and indicate whether their assumption about 'homogeneity' was valid. If the logarithmic transformation failed to stabilise the variance, a non-parametric test should be used.

(iii) It would have been better to compare changes in scores, rather than simply post-treatment values. Also, in view of the imbalance in the sexes between the two drugs in the melancholia group, an allowance for sex should have been made in the analysis.

It is now some years since White (1979) pointed out statistical errors in the *Journal*, but it is clear that there is still much room for improvement.

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SIR: We would like to make the following points regarding Dr Campbell's comments.

(i) In accordance with the norms of publication, it is not necessary to describe common statistical methods. In our case the interpretation of Snedecor's *F* does not lead to errors, given the context in which it appears.

(ii) Just as we indicated, the variance of the HRSD scores for the two major depression with melancholia groups are significantly different. But neither this fact nor the absence of normality in the distribution invalidates the use of Student's *t*-test. In fact, quite some time ago Bonneau (1960) demonstrated empirically that this test is extremely insensitive to the abnormality of the distribution and the heterogeneity of the variance when the *n* of the two groups is the same. This fact, added to the difficulty of interpreting the transformed scores, justifies not using them.

(iii) The use of non-parametric tests would reduce the power of the design.

(iv) Regarding power limits, it is important to point out that it is not the percentage of patients improved that is compared as Dr Campbell supposes but the difference in means in the HRSD score for both groups. By way of comparison, in the case of equality of variances and a 5% statistical significance

level, 17 subjects per group are required to obtain a significant effect with 80% power in the detection of an effect size 1 (group means separated by one standard deviation) (Meredith, 1967).

(v) Designs both of change and of final evaluation have advantages and disadvantages, so that both can be considered valid (Cronbach & Furby, 1970).

(vi) Articles like that of White (1979) are useful both in statistics seminars and in the planning of a clinical trial, but in the latter it is necessary to take into account other considerations, like those of Kraemer (1981), more removed from "research work that essentially exists only in textbooks" but which help us in the "real world of psychiatric clinical research".

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The Motor Disorders of Severe Psychiatric Illness: A Conflict of Paradigms

SIR: After examination of 100 chronically ill inpatients, Rogers (*Journal*, September 1985, **147**, 221–232) concluded that the traditional separation of their motor abnormalities into 'neurological' and 'psychiatric' was fruitless; in particular, there was no sharp division between the extrapyramidal signs of dyskinesia and parkinsonism on the one hand and the catatonic motor disorders of psychosis on the other. Choreic, athetotic, and dyskinetic signs could be demonstrated in untreated schizophrenic patients, and phenomenologically related abnormalities like tics and mannerisms had acquired dual terminologies. One of us (McKenna, 1987) argued further that this apparent continuum between involuntary movement disorders and motor catatonic phenomena might be consistent with a ventral striatal contribution to a basal ganglia dysfunction.

In a recent study of the relationship of negative symptoms to various types of motor disorder (McKenna *et al*, 1988), we unexpectedly obtained what we believe to be preliminary clinical evidence for such a position. Eighty patients meeting DSM–III/RDC criteria for schizophrenia and encompassing all grades of chronicity and severity were rated for negative symptoms, and also for tardive dyskinesia and parkinsonism using established scales (Simpson & Angus, 1970; Simpson *et al*, 1979). General motor disorder was measured using the scale developed by Rogers, modified slightly to incorporate a measure of severity. This scale allows a detailed assessment of abnormal motor behaviour, from simple abnormal movements to complex disturbances in overall behaviour, in a way which does not pre-empt their designation as neurological or psychiatric. It also permits a separation of schizophrenic motor phenomena into 'productive' (distinguished by their presence) and 'deficit' (distinguished by the absence/diminution of normal function) analogous to the positive/negative dichotomy, this being a particular focus of interest of the study.

During analysis of correlations using Spearman's non-parametric correlation coefficient we observed a striking pattern of inter-correlations among the various motor disorder ratings. In particular, tardive dyskinesia total scores correlated highly significantly with Rogers' 'productive' motor disorder scores ($r = 0.68$, $P < 0.001$), but there was no correlation with Rogers' 'deficit' scores ($r = 0.11$, NS). A mirror image pattern of correlations was seen between parkinsonism scores and Rogers' 'productive' ($r = -0.11$, NS) and 'deficit' ($r = 0.47$, $P < 0.001$) scores. The significance of these correlations persisted essentially unchanged even when items on the Rogers' scale which represented or might have been confused with tardive dyskinesia or parkinsonism were removed from consideration, leaving more purely catatonic 'productive' and 'deficit' scores.

This patterning of clinical associations is difficult to explain on the traditional basis that extrapyramidal signs and catatonic phenomena are entirely separate domains of pathology. On the other hand, it is just what would be predicted on Rogers' 'conflict of paradigms' view. If extrapyramidal and catatonic phenomena are merely points along a continuum of motor abnormality, then their frequent co-occurrence would be anticipated. Our findings also point to an extension of the concept of hyperkinetic and hypokinetic motor abnormalities beyond dyskinesia and parkinsonism, a finding which fits well with their postulated basis in a combined ventral striatal:basal ganglia dysfunction. These results can only be considered preliminary, as the relevant ratings were not made independently of one another;