Other psychiatrists seem less reticent about offering explanations for this strange symptom. Arieti (1974) summarises thus a case originally reported by another investigator: "Reitman (1951) reported a patient who thought that as a private in the army he had a dog's life. While on parade he disclosed his manifest outbreak of schizophrenia. He suddenly went on all fours and started to bark. His thought 'I am treated like a dog' became 'I am a dog', and consequently he acted as a dog."

Arieti, thus, discusses the symptom as the behavioural manifestation of concreteness of thinking in schizophrenia. Concrete thinking seems to be the underlying mechanism suggested by Shapira & Roy (*Journal*, March 1988, **152,** 432) when they attribute the "over-representation" of the syndrome in their hospital to "the proximity of the Newham Health Disrict to Barking and the Isle of Dogs".

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ARIETI, S. (1974) *Interpretation of Schizophrenia* (2nd edn). London: Crosby, Lockwood & Staples.

SIR: In response to Dr Buchanan's letter (*Journal*, October 1987, **151,** 562–563) and the subsequent case reports describing animal-like symptoms among patients (*Journal*, March 1988, **152,** 432–433), I wish to draw attention to the syndrome of lycanthropy, as so far no reference has been made to this in the correspondence.

Lycanthropy is a delusion where an individual believes that he or she has been transformed into an animal or whose behaviour is suggestive of such. It is the syndrome from which the 'werewolf' phenomenon has arisen. However, delusional transformation is not confined to wolves, and may involve any type of animal. Accompanying the virtual extinction of wolves in Europe has been a corresponding decline in reports of the 'werewolf' phenomenon and an increase in cited cases of transformation into other animals, most commonly the domestic type.

A detailed case report of a woman suffering from psychotic depression who believed she was a dog and adopted canine-like behaviour (including getting down on all fours and barking) has previously been reported in this Journal (Coll *et al*, 1985). Recently a further twelve cases of lycanthropy were reported involving delusional transformation into dogs, wolves, cats, rabbits, gerbils, etc. (Keck *et al*, 1988).

Lycanthropy is most commonly related to severe psychosis, and the differential diagnosis includes schizophrenia, manic-depressive disorder, psychotic depression, hysterical neurosis, and organic brain syndrome. It appears that lycanthropy is still very much alive as a clinical entity, and it warrants consideration whenever patients present with animal-like symptoms such as the recent cases reported in this journal.

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The Dopamine Hypothesis

SIR: I was surprised to read in the recent commentary by Crow (*Journal*, October 1987, **151,** 460–465) that "direct dopamine receptor agonists (e.g. apomorphine, bromocriptine) are not found to be psychotogenic in the same way" as amphetamines. In a review of over 600 endocrine cases treated with dopamine agonists, mainly bromocriptine (Turner *et al*, 1984) we found that at least eight patients had suffered severe psychotic side-effects. These were largely paranoid psychoses, and one of them was an extremely complex delusional parasitosis with additional first-rank symptoms. These reactions occurred in individuals with no previous history of psychotic illness, and at a wide range of dosage levels. The survey was not exhaustive, although all patients had been closely followed up by the Endocrine Department. Nevertheless, an incidence of at least 1% cannot be dismissed. Nor were the patients suffering from a primary disorder of dopamine metabolism, such as those with Parkinson's disease who have also been reported as suffering from psychotic reactions to bromocriptine.

Such findings do seem to support the dopamine theory of psychoses, albeit in a small way. Perhaps we should consider dopamine as similar to the stimulus that causes epileptic seizures. Thus those with 'epilepsy' have a very low threshold to having fits, yet most of us can be induced to have one if enough voltage is applied through cerebral electrodes. Likewise, given enough excess dopamine, whether

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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Reference

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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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WHITE, S. J. (1979) Statistical errors in papers in the British Journal of Psychiatry. *British Journal of Psychiatry*, **135**, 336–342.

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