

contented, more civilized, honest and profound mode. This kind of experience *can* be reduced to other forms than the broadly anecdotal one, but only at the price of disguising and distorting the actual experience.

If it were the policy of the *Journal* to exclude the intuitive, the subjective, the anecdotal, the green-fingered, the personal, then it would set a very severe constriction on the scope of its representation of British psychiatry as it is practised in the field.

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#### MATERNAL AGE AND PARENTAL LOSS

DEAR SIR,

Professor Moran, in his discussion (*Journal*, this issue, p. 207) of my study on parental age and schizophrenia (*Journal*, September 1966, pp. 899-905), raises some questions which call for comment.

The first is concerned with whether or not the distributions of parental ages of the schizophrenics differ to a statistically significant degree from those of the sibs. Professor Moran himself has pointed out the difficulty in determining this. My own calculations showed trends suggesting that schizophrenics had the older parents, but, using the Chi squared test, the differences did not prove statistically significant. I am happy, however, to accept Professor Moran's criticism of the method used and his conclusion that my figures for schizophrenics are probably significantly different from those on the controls.

A second question is whether or not the differences found are due to artefact, and in particular to the inclusion of incomplete sibships. This possibility was considered in my paper, but because the trends did not appear significant at the time it was not investigated further; it seems appropriate to do so now.

The method employed, of comparing probands

with sibs, is similar to that used by Greenwood and Yule (1) for the investigation of birth order effects, which McKeown and Record (2) have shown can be modified to detect parental age effects. Like the Greenwood-Yule method, however, the inclusion of incomplete sibships would tend to produce an artificial association between the probands and advanced parental age. An incomplete sibship is one into which further members are born after the data have been collected (in this case, after the time of admission of the proband to hospital). If data on these later sibs eventually became available, their inclusion in the study would tend to raise the parental age of the control group. Thus if there are a large number of incomplete sibships the parental age of the sibs will be artificially low, and the probands may seem to have the older parents.

We can test the hypothesis that the results obtained are due to this bias, because the older a schizophrenic is at the time of his admission the more likely he is to come from a complete sibship; in particular, patients aged 30 years or more at that time are likely to have complete fraternities because their mothers would then be at least 45 years of age and probably outside the reproductive period. To test the hypothesis, the parental ages of patients admitted before they were 30 years were compared with the parental ages of their sibs, and, similarly, comparisons were made between the over-30 probands and their sibs. In each comparison adjustments were made to the distributions to eliminate any effect of sibship size, in the same way as described in the original paper.

The table sets out the results. In each case the difference between the mean parental ages of the schizophrenics and their sibs is greater for the under-30s than for the over-30s, in some instances strikingly so. These findings are consistent with artefact due to incomplete sibships and do not indicate a true parental age effect in schizophrenia.

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*Standardized Mean Parental Ages (in Years) of Schizophrenics and Their Sibs*

	Maternal Age						Paternal Age					
	Probands under 30 at admission		Probands over 30 at admission		All probands		Probands under 30 at admission		Probands over 30 at admission		All probands	
	No.	Mean Age	No.	Mean Age	No.	Mean Age	No.	Mean Age	No.	Mean Age	No.	Mean Age
Male probands ..	202	31·515	174	30·996	376	31·275	199	35·033	167	34·777	366	34·916
Sibs of male probands ..	202	29·971	174	13·030	376	30·461	199	33·577	167	34·421	366	33·962
Difference ..	..	+1·544	..	-0·034	..	+0·814	..	+1·456	..	+0·356	..	+0·954
Female probands ..	184	29·835	268	30·840	452	30·431	174	34·218	251	34·558	425	34·419
Sibs of female probands ..	184	29·211	268	30·332	452	29·876	174	33·480	251	31·487	425	34·075
Difference ..	..	+0·624	..	+0·508	..	+0·555	..	+0·738	..	+0·071	..	+0·344

## REFERENCES

1. GREENWOOD, M., and YULE, G. U. (1914). "On the determination of size of family and of the distribution of characters in order of birth." *J. roy. statist. Soc.*, 77, 179.
2. MCKEOWN, T., and RECORD, R. G. (1956). "Maternal age and birth order." *Amer. J. hum. Genet.*, 8, 8-23.

## HETEROCHROMOPHILIA

DEAR SIR,

I wonder whether any of your readers have encountered the condition of *heterochromophilia* which I described some time ago. This is the compulsion in human beings to choose a mate of different colour.

It was shown very nicely in the American engineer, Clarence King (1), who was born into higher American society, and met many wealthy and beautiful white girls, but preferred to sleep with coloured women, and indeed had a long series of coloured mistresses.

I have described elsewhere a similar case I encountered (2).

The condition appears to be rather more than a fetishism since no single article of clothing, etc., is the basis of the attraction, but the whole woman. Indeed, it appears to be in the nature of imprinting in human beings. The man behaves in a similar way to the ducks and geese, described by Lorenz, who find their own type unattractive but the imprinted one overwhelmingly fascinating. In human beings this imprinting seems to be caused by the colour of the nurse in babyhood.

It seems to be that apart from the implications as to its origin the condition is rare and not of great importance, but since we are getting more coloured women nursing white babies these days there is a likelihood of its becoming much more common.

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## REFERENCES

1. WILKINS, T. (1958). *Clarence King*. London.
2. ALLEN, C. (1962). *Textbook of Psychosexual Disorders*. London.

## CHLORPROMAZINE IN CHRONIC SCHIZOPHRENIA

DEAR SIR,

The excellently designed study by Letemendia and Harris which appeared in the *Journal* for September,

1967 (pp. 950-958), on the evaluation of chlorpromazine in the untreated chronic schizophrenic patient, constitutes a real triumph of technique over purpose. The small doses used demonstrate conclusively that inadequate treatment will result in inadequate response. The National Institute of Mental Health co-operative study has already demonstrated that doses of 300 mg. a day of chlorpromazine are ineffective, but it is nice to have this confirmed. The N.I.H. found that doses of 500-600 mg. a day constitute a practical working minimum. On the package inserts in the United States the range is from 400 mg. to 2,000 mg. plus. In one of the standard American textbooks (Noyes and Kolb) the recommended dosage is 600-800 mg. Even Henderson and Gillespie suggest routine doses up to 400 mg. and occasional ones to 800; and Sargent and Slater in their *Physical Methods in Psychiatry* recommend 600-800 mg. and up to 3,000.

Since apparently the patients are still available, it would be a brilliant *tour de force* if Letemendia and Harris would repeat the identical experiment but this time use 600-900 mg. as a minimum. This might help resolve the high dosage versus low dosage controversy.

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DEAR SIR,

Dr. Kline approves our technique but misunderstands our purpose. The title of our paper, which he quotes correctly, should make it clear that we were dealing with *chronic* schizophrenics, and we said explicitly that we shared the view that "the manifestations of *acute* schizophrenic illness can be controlled by chlorpromazine".

The dose of 300 mg. a day in chronic schizophrenia seemed to be in accordance with the practice of the time and with recommendations in the literature. Inquiries made recently in this country at other hospitals suggest that doses averaging 150-300 mg. a day are still customary in chronic schizophrenia, and it was our object to test whether such doses do in fact modify the course of the illness.

In answering Dr. Kline, it is really unnecessary to do more than state this point. However, it would be a pity to allow the impression to remain that all the authorities he cites agree with him in thinking that 300 mg. a day is an inadequate dose, and that much higher doses are always and necessarily required. Sargent and Slater, for example (*An Introduction to Physical Methods of Treatment in Psychiatry*, 4th ed.,