

A. Robin and Dr. R. Ström-Olsen for suggesting and allowing me to report the cases C.S. and P.P.; and to Professor Desmond Pond for reading the manuscript.

S. J. M. FERNANDO.

*Department of Psychiatry,
London Hospital Medical College,
Turner Street,
London, E.1.*

REFERENCES

- BALDUCCI, M., and FRASCELLA, G. (1962). "Haloperidol in the treatment of a case of Gilles de la Tourette's disease." *Symposium internazionale sull' haloperidol e triperidol*. Milan, November 1962.
- CLARK, D. F. (1966). "Behaviour therapy of Gilles de la Tourette's syndrome." *Brit. J. Psychiat.*, **112**, 771-778.
- CONNELL, P. H., CORBETT, J. A., HORNE, D. J., and MATHEWS, A. M. (1967). "Drug treatment of adolescent tiqueurs. A double blind trial of diazepam and haloperidol." *Brit. J. Psychiat.*, **113**, 375-381.
- CREAK, M., and GUTTMAN, E. (1955). "Chorea, tics and compulsive utterances." *J. ment. Sci.*, **81**, 834-839.
- ELLBON, R. M. (1964). "Gilles de la Tourette's syndrome." *Med. J. Aus.*, **i**, 153-155.
- FERNANDO, S. J. M. (1967). "Gilles de la Tourette's Syndrome." *Brit. J. Psychiat.*, **113**, 607-617.
- HEALEY, N. M. (1965). "Gilles de la Tourette syndrome in an autistic child." *J. Irish med. Assoc.*, **57**, 93-94.

DEAR SIR,

Dr. Fernando states (*Journal*, June 1967, p. 614) that Gilles de la Tourette's Syndrome has not been reported outside Europe and America. Two cases have been reported in the *Indian Journal of Psychiatry*, one in 1962 (Vol. 4, p. 187) and the other in 1966 (Vol. 8, p. 228). During a discussion on one of the cases, several colleagues reported that they had seen this disease in different parts of India.

AJITA CHAKRABORTY.

*Neurology Department,
Inst. of Post Graduate Medical Education & Research,
Calcutta.*

HOMOSEXUALITY

DEAR SIR,

Dr. Clifford Allen (*Journal*, October 1967, p. 1158) has kindly shown where many people would disagree with the theory of a sex control centre, and I would like to use his points to explain the misunderstanding that has arisen from my brief letter.

1. The theory depends on an endocrine lack only at the time the suggested centre is maturing, probably around birth. The testable point of the theory only requires a satisfactory test for anti-androgen protein

in mothers near term. This could be used in primigravidae and multiparae to see if there is a variation in titres and if this is dependent on the sex of the infant.

2. I feel the physique of the homosexual is not a guide to the individual's central nervous system at the stage medicine is now.

3. I have insufficient data to agree that some cases of homosexuality are "cured" by psychotherapy, and if the theory is correct prevention should be easier than cure (using techniques similar to that in Rhesus-negative mothers with Rh positive infants.)

4. I agree conditioning is a factor in the behaviour of mothers' favourite sons and also that in an excessively feminine environment unusual behaviour can occur in a male.

5. This final point makes the difficult division of effect of hormone in the *adult* and the direction depending on the psyche. I agree the adult responds to hormones by activity, but the direction is a result of hormone levels at a "critical period" when the sex control centre is maturing, possibly near the time of the person's birth.

D. GREGORY MAYNE.

*Mullalelish,
Richhill,
Co. Armagh,
N. Ireland.
at present 19 Babbacombe Road,
Bromley, Kent.*

KRAEPELIN AND HIS
APPROACH TO NOSOLOGY

DEAR SIR,

The point raised by Professor Fish in his review, (*Journal*, November 1967, p. 1321) which relates to what I wrote about Kraepelin's nosology, seems important from an historical point of view, and also for the understanding of present day diagnostics.

Perhaps the best way to show Kraepelin's mode of thought and his approach to nosology is to let him speak for himself. (Kraepelin, E., (1913) 8th ed., Vol. 2, p. 939) "Whether dementia praecox as circumscribed here is a single disease entity can at present not be decided . . . I always had reservations about including the paranoid forms into dementia praecox. . . . The question (of inclusion) can only be decided on the basis of the entire course of the illness, during which those signs or symptoms will come more and more to the fore which are essential characteristics of the illness, rather than the unessential ones which will tend to move into the background though they may at times be more conspicuous than the former.

"If we apply these principles to the problem in hand, we find that at least some of the illnesses which begin as paranoid forms will eventually develop into end-states which are indistinguishable from those of other forms of dementia praecox. . . . In other cases again the generally paranoid course of the illness may be interspersed by mental states which are unmistakably those of dementia praecox, such as silly excitement with mannerisms and stereotypies, negativistic stupor, etc. . . . Finally it must be pointed out that the delusions and hallucinations that we find in these paranoid illnesses are of the same kind which we also find in the other forms of dementia praecox, although there they may be accompanied by different signs and symptoms. . . ."

As regards the outcome of dementia praecox he actually says this: (Vol. 2, p. 945) "The supposition that dementia praecox can show different signs or a different outcome, that it can sometimes lead to recovery, sometimes to more or less severe deterioration, depending on the severity or extent of the underlying process, is in itself not an unlikely one."

So much for the general principles. As regards the inclusion into dementia praecox of the two conditions which Professor Fish mentions, the following is relevant. (Vol. 2, p. 940).

"In any case we can take it as established that paranoid states can occur in the course of dementia praecox. . . . I have so far not included into the dementia praecox a section of Magnan's 'Délire chronique' and 'Dementia paranoides', now known as 'Dementia phantastica'. On the other hand I have included those paranoid forms which fairly quickly show marked mental deterioration, and also those in which one can demonstrate besides the delusions and hallucinations those disorders of affect and volition, perhaps only in their early stages and not yet fully developed, which we regularly find in cases of dementia praecox. It appears entirely impossible as far as I can see, to separate the latter sharply from those paranoid forms. . . ."

"It has been suggested to keep separate a number of other groups of cases described in the literature. . . . Morsell, e.g. reached the conclusion that catatonia should be regarded as a separate condition; he says it is curable and is caused by infections.

"I have to regard this view as wrong and unsubstantiated. Although catatonic states can sometimes be found after infections, it must be remembered that in the majority of cases this is quite out of the question. . . . Catatonic states can suddenly appear at any

time in the course of dementia praecox, sometimes only after ten years or more. Furthermore, we find endstates in the wake of catatonia which are indistinguishable from those of other forms of dementia praecox; finally catatonic signs can accompany the clinical picture of dementia praecox to varying extent and in a variety of groupings. . . ."

To present Kraepelin's reasoning for inclusion of those forms into one disease simply as Professor Fish did ". . . because they all led to a peculiar kind of psychological defect" is surely misleading. The fact is, as I have pointed out in my article, that Kraepelin aimed in his classification at truly nosological entities, i.e. entities with the same cause. He argued that it can reasonably be expected that the underlying cause will affect the onset, the symptoms, the course, the outcome and the endstate of the clinical picture, and so where the actual cause was not known he took all these factors into account when defining his disease entities.

J. HOENIG.
Reader.

*University of Manchester, Department of Psychiatry,
The Royal Infirmary,
Manchester 13.*

WHAT THEY REALLY SAID

DEAR SIR,

Dr. D. Stafford-Clark claims (*Journal*, June, 1967, p. 683) that Freud and Jung only swapped dreams and speculated on their interpretations during a transatlantic trip in 1909.

This is not in accordance with Jung's explanation to me on this question when I was invited to his home in Küsnacht in 1957.

Jung told me why he introduced the learning analysis, and motivated the need for this from his experiences in analysing Freud on a journey in 1909 when both had received an invitation to the U.S.A. He had realized that Freud suffered from a neurosis and that he was without insight into this. Jung also stated that the essential in the learning analysis was not to come to an old master, but to accept one's analyser and to feel oneself minor in relation to him.

ARNE SANDBU.
Psychiatrist in Charge.

*Dikemark Hospital,
Asker,
Norway.*