in the data sheets of both the companies who market mianserin. However, we should also like to point out that there is still considerable controversy concerning this possibly serious interaction and we do not believe there is an absolute contra-indication for this combination of drugs. We point out under the heading 'Drug Interactions' in our data sheet:–

"Concurrent anticoagulant therapy of the coumarin type (e.g. Warfarin) is also permissible, but close additional monitoring procedures should be carried out."

We cannot account for the omission of this possible interaction in the British National Formulary and C.S.M. 1982 Report, but we trust that the situation has now been put in its true perspective.

R. J. ANCILL

Senior Medical Adviser, Bencard

S. M. PINKERTON

Medical Adviser, Organon

CLASSIFICATION: REVISING DSM-III AND ICD-9

DEAR SIR,

I should be grateful for the opportunity to air some suggestions about future versions of the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III) and the psychiatric section of the International Classification of Diseases, (Ninth Revision, ICD-9). This discussion concerns only the clinical descriptive axes of the future classifications, although it is presumed that both of them are likely to be presented as multi-axial systems.

Both these major classifications have some serious drawbacks, particularly for the busy clinicians who must form the largest group of users. ICD-9 is still very general in its descriptions, gives little guidance about the relative diagnostic importance of symptoms, and contains no comments on differential diagnosis. In contrast, DSM-III has provided for most of its categories a full set of research diagnostic criteria. However, the elicitation and recording of these may not be appropriate in busy clinical situations. In many ways the innovations in DSM-III are praiseworthy attempts to improve the quality of psychiatric diagnosis, but the pressure for innovation and the desire for an all-purpose classification have caused problems. Surely no single classificatory document can satisfy the very different needs of administrators, planners, clinicians and research workers.

The suggestions which follow have two aims; first, to provide ways in which the next revisions of the two classifications can be brought closer together, and second, to improve their suitability for use in clinical and service settings. Even if further divergence cannot be prevented, at least the origins and nature of differences will be made more obvious if some of the suggestions are followed.

It is suggested that those who are responsible for the production of ICD-9 and DSM-III should be prepared to present their classification by means of three separate but closely related documents. The first would be a description of a set of concepts upon which the classification is based. The second would be a statistical classification presented in a form suitable for use by busy clinicians under ordinary working conditions, and also by those interested in reporting the statistics of psychiatric services. The third would be a set of precisely specified diagnostic criteria, suitable for research.

1. *A set of concepts.* This would be in the form of descriptions of the disease entities, syndromes and symptoms which consitute the classification. The descriptions would be in some detail, although strict definitions would not be achieved. Conceptual similarities and differences between different disorders would be described, and notes on the history of the concepts would be included.

2. *A statistical classification for clinical and service use.* Users would need to be familiar with the set of concepts already described. This statistical classification would have a lay-out similar to the ICD-9 *Glossary and Guide*, showing clearly the nomenclature and code numbers at three and four-digit levels, and possibly more besides. Only a brief summary of the clinical features of each condition would be required (perhaps between five and twenty lines of text). A list of the main symptoms in order of importance would occupy most of this space, and comments on differential diagnosis would follow. Guidance would be given as to how many and which symptoms might usually be required before a confident diagnosis could be made, but this would not be as rigid as in the research diagnostic criteria. Some of the problems about confidence of diagnosis or "goodness of fit" could be dealt with by providing an additional digit, to be coded according to the extent to which the clinical diagnosis fulfils the full set of Research Diagnostic criteria (for instance: 1 = full set of RDC met; 2 = RDC not met but a confident clinical diagnosis can be made; 3 = provisional diagnosis only).

3. *Research Diagnostic Criteria.* In this document, the nomenclature and code numbers of the statistical classification would be accompanied by detailed diagnostic criteria suitable for use in research, as in DSM-III. Restrictive criteria that are potentially controversial, such as age-limits, or duration of symptoms, would be justified in preliminary comments upon each set of criteria. Different degrees of restriction could be specified for some conditions by

the provision of extra codes. Extra codes for these and other purposes should pose no special problems for users of *Research Diagnostic Criteria,* since they are designed for use by workers with special time and interest who can master such additions at their leisure.

Even if those who will be working on ICD-10 and DSM-III find it eventually impossible to present their classifications in this way, communication with each other with these three different levels of development and use in mind would be likely to minimise the final degree of divergence.

J. E. COOPER

Mapperley Hospital,
Porchester Road,
Nottingham NG3 6AA

KLINEFELTER'S SYNDROME IN KENYAN PATIENTS

DEAR SIR,

I report two cases of Klinefelter's Syndrome in Kenyan African patients. The hormonal investigations were done using the World Health Matched Reagent Programme Radioimmunoassay Manual, 1980. The normal values in millimoles are: Prolactin (PRL) 68–600; follicle stimulating hormone (FSH) 0.33–4.5; luteinising hormone (LH) 0.9–9.0.; testosterone (T) in nonimole 8–37. The testicular volume was measured using the Praden Orchiometer (normal range 12–25 mls). The buccal smear for Barr bodies was done using the Papanicolou method.

Case 1: Aged 31; first born; arranged marriage two years previously: never had any sexual drive or sexual intercourse, so that the marriage had not been consummated. He was 6ft 1in (186 cm), slim built, shy, and soft spoken. He had gynecomastia and no growth of hair on the chin. There was a history of epileptic attacks since age of 8, well controlled with phenobarbitone. At times he had shown schizophrenic symptoms in the form of auditory hallucinations which on different occasions were either insulting or reassuring; occasionally the voices were making a running commentary on him. He had accomplished the equivalent of only 4 years of formal education as he was intellectually retarded.

Investigations: Hormones: PRL 150; FSH 6; LH 10; T 5 Testicular volume: 10 mls (left) and 8 mls (right) EEG recording—"increased theta activity." Cytology: 30 per cent of the cells had 2 Barr bodies and another 20 per cent had 1 Barr body.

Case 2: Age 35; first born; married 3 years previously under pressure from father, marriage consummated but potency died out completely within months of marriage. He was 5ft. 8ins (173 cm), slim, soft spoken and shy with patchy hairs on the chin and gynecomastia. He had achieved four "O" levels.

Investigations: Hormones: PRL 300; FSH 44; LH 9.1; T 8. Testicular volume: 12 (left) and 13 (right). EEG—"no abnormality". Cytology: 8 per cent of the cells had 1 Barr body.

Comment: The history and clinical features consistent with Klinefelter's syndrome are supplemented in each case by laboratory findings of high or borderline levels of FSH and LH and low or borderline levels of T, and by cytological tests. Case 1, who had more abnormal investigation results than case 2 also had more abnormal clinical features, i.e. he was taller, had no hair on the chin, was more intellectually retarded, had history of epileptic attacks, and of schizophrenic symptoms and had had no sexual potency.

DAVID M. NDETEI

University of Nairobi,
P.O. Box 30588,
Nairobi

ALPHA-BLOCKADE AND IMPOTENCE

DEAR SIR,

We are interested in the observations presented by Professor Brindley (*Journal,* October, 1983, **143,** 332–7) relating to the effect of alpha adrenoceptor antagonism on penile erection. We have for the past two years been using oral phenoxybenzamine as an adjunct to a behavioural approach to the management of erectile inadequacy. Although we have not yet undertaken a controlled trial our impression is that this treatment does facilitate a successful outcome. Failure of ejaculation induced by the drug is accepted by the majority of patients although it is not always necessary to commit the patients to long term treatment.

We started to use alpha adrenoceptor blockade in the management of erectile inadequacy when we demonstrated that labetalol, a combined alpha- and beta- adrenoceptor antagonist, delayed detumescence in volunteers (Riley, Riley and Davies, 1982). Beta adrenoceptor blockade with propranolol does not have this effect. Furthermore, in four of five subjects, the intravenous injection of midodrine, an alpha adrenoceptor agonist, prevented erection in response to stimulation that had previously induced erection in the same subjects. Adminstration of phenoxybenzamine to four hypertensive men who developed erection failure during treatment with beta blocking drugs resulted in return of potency in three of the patients. Withdrawal of the phenoxybenzamine resulted in the recurrence of the erection failure after five to fifteen days. The treatment was then changed to labetalol