

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.

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KLINFELTER'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 Orchimeter (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.

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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. Administration 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

which adequately controlled the blood pressure without adverse effect on erection.

From these observations it would appear that excessive alpha adrenoceptor stimulation, such as can result from the unmasking effect of a beta blocker, inhibits erection. It can be postulated that some patients may have an increased sensitivity of the alpha adrenoceptors involved in the erectile mechanism. Activation of the sympathetic nervous system during sexual arousal and anxiety (induced by previous failure of erection) would therefore inhibit erection in such patients by excessive alpha adrenoceptor stimulation. This hypothesis provides a rationale for a therapeutic trial of an alpha adrenoceptor blocking agent such as phenoxybenzamine in the management of erection inadequacy.

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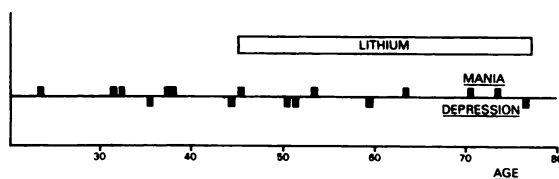
Reference

- RILEY, A. J., RILEY, E. J. & DAVIES, H. J. (1982) A method for monitoring drug effects on male sexual response: The effect of single dose labetalol. *British Journal of Clinical Pharmacology*, **14**, 695–700.

30 YEARS ON LITHIUM

DEAR SIR,

The account by Chiu *et al* (*Journal*, October 1983, **143**, 424–5) of renal function in the patient who must have held the world record for length of time on lithium is very interesting. It is also interesting to prepare from their data on his episodes of illness a chart which shows that the treatment may have helped him only modestly.



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RAPID EYELID TREMOR AFTER A MASSIVE PHENOTHIAZINE OVERDOSE

DEAR SIR,

A 20 year old man with 5 previous admissions, the first 17 months earlier, who was diagnosed variously as suffering from schizo-affective or manic-depressive (manic) psychosis was admitted after an overdose of 80 × 5 mg fluphenazine tablets and 6 × 0.5 mg benzotropine tablets, 2 days previously. On examination he had a rapid twitching of his upper eyelids in both the closed and open position, synchronous with the tremor of his hands and tongue. There was a superimposed slow spontaneous blinking of 8 blinks per minute (normal ± 12 blinks per minute—Carney and Hill, 1982). Ocular movements were normal. The glabellar reflex showed non-habituation, and there were other Parkinsonian features. There were no signs of tardive dyskinesia; and there were no signs of psychosis.

He was given benzotropine 2 mg intravenously. Within 20 minutes the eyelid tremor had markedly decreased. He reported feeling less stiff, and there was decreased jaw stiffness. The glabellar response remained non-habituated. He was subsequently given benzotropine 2 mg bd orally. One week later, the tremor of the eyelids had ceased; but the fine tremor of the hands persisted, and blinking was still slow—10 blinks per minute. The glabellar reflex habituated after 3 taps.

Blink rate is decreased in Parkinson's disease (Hall, 1945), the latter sometimes being indistinguishable from drug-induced Parkinsonism (Baldessarini, 1980); but eye blinking is increased in schizophrenia and tardive dyskinesia (Stevens, 1978), and in Gilles de la Tourette syndrome (Cohen *et al*, 1980).

Penders and Delwaide (1971) showed a return towards normal eye movements in Parkinsonian patients treated with L-dopa or amantidine. Conversely, dopamine blockade by neuroleptics reduces the blink rate and thought disorder in schizophrenics (Karson *et al*, 1981a). Reduction of dopamine blockade as in Stevens' patients (Stevens, 1978) who were medication-free for 1–6 months, leads to an increase in blinkrate. Also, Karson *et al* (1981b) found an increased blink rate to apomorphine after haloperidol discontinuation. The central role of dopaminergic blockade in abnormal eye movements was further illustrated by the finding (Karson *et al*, 1983) of an inverse relationship between spontaneous blink rate and platelet monoamine oxidase activity.

Here, there was a decrease in spontaneous blinking, in keeping with the picture found in drug-induced Parkinsonism i.e. a dopamine blockade. The rapid tremor of the eyelids, synchronous with the tremor of the hands and ameliorated by benzotropine, is thought