X-linkage in Manic-depressive Illness

SIR,—In your leading article entitled "Physical Bases for Mental Illness" (21 April, p. 129) you refer to some recent linkage studies in manic-depressive illness from this institute and elsewhere. May we respectfully comment on three points you made in this editorial.

First, it may not be clear from your formulation that liability to manic-depressive illness is in no way associated with the presence or absence of a colour-blindness gene —that is, there is no aetiological relationship between manic-depressive illness and colour blindness. As a matter of fact, the proportion of families with colour blindness in samples of manic-depressive patients does not exceed the prevalence of colour blindness in the general population. It is just that in those families that happen to carry both genes, analysis for close linkage of the genes on the same chromosome can be carried out.

We would also like to point out that the linkage studies involving the Xg blood group and manic-depressive illness were carried out on families different from the ones reported in the colour-blindness study.

Last but not least, we would like to comment on the statement that "boys who are not colour blind have only a small risk [of manic-depressive illness], but the others have a big risk." This will be true only in families where colour blindness and manic-depressive illness happen to be "in coupling" —that is, with both traits located on the same chromosome. Precisely the opposite will be the case in families where the traits are "in repulsion"—that is, on homologous chromosomes. In such families boys who are not colour blind would be at risk. The two situations are in theory equally probable, and actually two families in the Xg report[1] referred to in your article are of the latter nature.

We are particularly concerned about making these points clear, because any ambiguity in this matter may be misleading to patients suffering from affective disorder and non-patients who are colour blind.—We are, etc.,

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1 Fieve, R. R., and Mendlewicz, J., *Psychopharmacologia* (Berlin), 1972, **26**, 93.

Rehabilitation

SIR,—One regrets having to state a viewpoint with an emphasis different from that of my friend, Dr. I. R. Henderson (21 July, p. 169). It is unfortunate that "rehabilitation" is used in a pejorative sense for a particular department of medicine. There is a practical philosophy involving the generality of medicine and a technique which should be part of every specialization of medicine. The philosophy is that rehabilitation is the process of restoring a disabled person (from whatever the cause) to normal health and competence. The techniques consist primarily in every preventive, diagnostic, and therapeutic process which is involved from the time the casual process is apparent until the cure is achieved. Cure means achievement in all respects as well as the processes leading to it. In the vast majority of disabilities this is well done by every doctor.

Perhaps I may be excused for referring to my own department of medicine, which is orthopaedics—one of the most general. In former times it led the way in developing the full significance of rehabilitation in all three aspects that I have mentioned. In doing so it considered the whole man, and in this it was bound to co-operate with all other relevant departments of medicine and surgery. It was notable for the leadership of such men as Robert Jones, Elmslie, and Gauvain, and the tradition and practice lives on. It is no longer unique. Rehabilitation as a technique certainly does involve many disciplines of medicine, and each has its special requirements in the ancillary fields of physical care, social services, industrial retraining, etc.

While, as in medical diagnosis and therapeutics, there may be special centres for rehabilitation, the need should not be for many more if the practical philosophy is understood and developed. We have advanced greatly in the last 50 years in preventive medicine, in therapeutic techniques, and in the social services. If every clinician could be a good doctor (and all that this word means) we would need fewer rather than more "rehabititationists" (What a word!).—I am, etc.,

NORMAN CAPENER

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SIR,—With reference to the letter from Dr. I. R. Henderson (21 July, p. 169) on rehabilitation—what a delightfully apt description of a general practitioner. Only I trust that when "consultant in rehabilitation" is added to our many titles an appropriate amount will be added to our remuneration.—I am, etc.,

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Penicillins for Haemophilus Infections

SIR,—I agree with several recent correspondents in deprecating the routine use of ampicillin for sore throats, whether on the doubtful supposition that they are caused by *Haemophilus influenzae* or not. But I fear that an alternative suggestion made by Dr. H. Pullen (7 July, p. 47) may be misleading. He writes: "It should be remembered that many strains of *H. influenzae* are as sensitive to pencillin as to ampicillin, and most *H. influenzae* will have their growth at least inhibited by levels of penicillin which should readily be achieved by correctly given oral phenoxymethyl-penicillin."

In my experience the difference in activity against *H. influenzae* of benzylpenicillin and ampicillin is usually fourfold, and I have never seen a strain equally sensitive to both; I suggest that the existence of such strains requires detailed published confirmation. However, what I suggest is misleading is the apparent assumption that benzylpenicillin and phenoxymethylpenicillin (penicillin V) are equivalent for this purpose. In studies made 13 years ago[1 2] when the semisynthetic phenoxy penicillins were being introduced, I showed that both phenethicillin and penicillin V were four to eight times less active against *H. influenzae* than benzylpenicillin. This reduced activity extended to *Neisseria* spp. and to the less penicillin-resistant enterobacteria (*Escherichia coli*, *Proteus mirabilis*, and *Salmonella* spp.). It is doubtful whether a phenoxy penicillin is ever indicated in any Gram-negative infection.

Penicillin V is thus 16 times less active against this organism than ampicillin; commonly stated minimum inhibitory concentration are respectively 4 and 0·25 μg/ml. Add to this that twice as much of an oral dose of ampicillin as of penicillin V is absorbed, and that only 20% of ampicillin in the blood is protein-bound but 80% of pencillin V, and the factors operating against the success of penicillin V as compared with ampicillin may thus be summated as $16 \times 2 \times 4 = 128$. If a genuine *H. influenzae* infection has to be treated, penicillin V is emphatically contra-indicated.—I am, etc.,

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1 Garrod, L. P., *British Medical Journal*, 1960, **1**, 527.
2 Garrod, L. P., *British Medical Journal*, 1960, **2**, 1695.

Speech Pattern Audiometry

SIR,—Both your leading articles on "Auditory Perception" (30 June, p. 728) and the paper by Mr. J. A. M. and Mrs. Dorothy Martin (26 May, p. 459) on which it is based are marred by an important flaw and, though this has been pointed out privately, since I hope that tests of this type will eventually be clinically valuable and widely available, I would be grateful for the opportunity to explain what I think is wrong and how it may be remedied.

The most important aim of the work was to provide the basis for standard test procedures which "could be applied to children of normal hearing with delayed speech" (your leading article) and which would provide a supplement to pure tone threshold audiometry for adults who "... have difficulty in recognizing meaningful sounds yet have no hearing loss" (Mr. and Mrs. Martin). Three types of stimuli were employed in the initial experiments described: the first involved steady sounds of constant spectral envelope which differed in fundamental frequency; the second was based on simple sinusoids which differed only in duration; the third involved the use of differing patterns of percussive beats. These are useful and interesting stimuli but they have in common that they are not speech-like or derived from the patterning of speech sounds. It is wrong to base an appraisal of a patient's speech perceptual ability on tests which do not involve speech-patterned stimuli or the operation of the speech mode of perception.

In the past two decades an appreciable body of knowledge has accumulated which bears on the nature of the acoustic patterns of speech and on the special processing mechanisms which are employed by a normal listener in their perception. We know for example from early experiments[1 2] how a noise burst centred at a constant frequency can be recognized as a quite different consonant if the format pattern of the vowel with which it is associated is changed and how the rate and direction of format changes in the vowel itself carry crucial information for the identification of the whole sequence. In a similar fashion but at a lower level of speech perceptual processing the same acoustic pattern can be judged as a