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.,

JULIEN MENDLEWICZ
JOHN D. RAINER

Department of Medical Genetics,
New York State Psychiatric Institute,
New York, N.Y.

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

Dunchideock, Exeter

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.,

G. M. INGALL

Longtown, Cumberland

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 × 2 × 4 = 128. If a genuine *H. influenzae* infection has to be treated, penicillin V is emphatically contra-indicated.—I am, etc.,

LAWRENCE P. GARROD

Wokingham, Berks.

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

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different speech sound if it is associated with a different speech source.[3] This latter observation is particularly relevant to the earliest stage of language learning—the child will be unable to make progress if he cannot equate his sounds with those made by the adults around him—and possibly to some cases of language impairment in the adult since if this ability to abstract is absent there might be no speech comprehension presented with otherwise normal hearing.

In the fairly near future this background knowledge will provide the basis for a speech pattern audiometry which will be truly intermediate between the classical pure tone and speech audiometric tests. Though its test stimuli are computer-generated and far more complex than those which are at present used, they can be tape-recorded and used clinically with quite simple and reliable apparatus. In addition to this we can expect eventually to see the employment of speech therapy techniques which depend on the analysis and display of speech pattern features,[4] and the use of synthetic speech for the blind[5] and as a prosthetic speech aid for those who are entirely without expressive speech.

These technical developements which are likely to stem from the use of our present knowledge of speech patterns will probably be paralleled by an increase in our understanding of the mechanisms which underly the speech mode of perception. The experimental techniques which have made hemispheric specialization more accessible to measurement[6] and those which are beginning to define the role of short-term auditory memory in speech processing[7] may be capable of extension so that the process of speech pattern encoding itself is better understood.

This rather superficial survey will have achieved its purpose if I have at least indicated how audiometric stimuli could, to advantage, be related to the nature of speech itself.—I am, etc.,

A. J. FOURCIN

Department of Phonetics and Linquistics,
University College,
London W.C.1.

1 Cooper, F. S., Delattre, P. C., Liberman, A. M., Borst, J. M., and Gerstman, L. J., *Journal of the Acoustical Society of America*, 1952, **24**, 597.
2 Delattre, P. C., Liberman, A. M., and Cooper F. S., *Journal of the Acoustical Society of America*, 1955, **27**. 769.
3 Fourcin, A. J., *I.E.E.E. Transactions on Audio and Electroacoustics* 1968, **AU-16**. 65.
4 Fourcin, A. J., and Abberton, E. R. M., *Medical and Biological Illustration*, 1971, **21**, 172.
5 Nye, P. W., Hankins, J. D., Rand, T. Mattingly, I. G., and Cooper, F. S., *Air Force Cambridge Research Laboratories, Special Reports 131 Art* A. 3, p. 23 Cambridge, Massachusetts, 1972.
6 Satz, P., Achenbach, K., and Fennell, E., *Neuropsychologia*, 1967, **5**, 295.
7 Conrad, R., *British Journal of Psychology*, 1964, **55**, 75.

Bronchospasm after Althesin Anaesthesia

SIR,—I read with interest the letter by Lt. Col. T. R. Austin and others (16 June, p. 661) in which it was assumed that Althesin was the drug responsible for bronchospasm occurring during anaesthesia. Apparently no tests were performed to substantiate this conclusion despite the simultaneous use of other drugs, including pancuronium. In a recent similar case, to be published in the *British Journal of Anaesthesia*, bronchospasm occurred following the administration of pancuronium and Althesin. Further investigations during anaesthesia and also by intradermal testing postoperatively showed conclusively that pancuronium and not Althesin was responsible for the bronchospasm. Anaesthesia is a complex situation and adverse reactions may be caused by any one of the drugs used, by drug interactions, or by other factors.—We are, etc.,

R. M. CLARK
F. R. ELLIS

Department of Anaesthesia,
General Infirmary, Leeds

SIR,—In reply to Dr. M. J. Heslop's letter (30 June, p. 775) commenting on our report of a case of bronchospasm following the administration of Althesin and pancuronium (16 June, p. 661), may we say we were most interested to read of the case of bronchospasm following pancuronium, reported by Buckland and Avery.[1] Until this latter report pancuronium had held an unblemished record over its five-year world wide use for not causing histamine release, even in susceptible laboratory animals in large dosage.[2] As this information was not available at the time we felt that our patient's problem had probably been caused by the Althesin.

This correspondence emphasizes, we feel, the very real need for the kind of inquiry which Professor J. W. Dundee and Dr. R. S. J. Clarke are embarking upon,[3] with a scientific evaluation of the extent of unusual reactions to anaesthetic drugs, and especially new anaesthetic drugs.

In reply to the final point in Dr. Heslop's letter we can only say we have issued the patient with a card pointing out the problem we encountered. The value of skin testing is not proved and there remain plenty of alternative agents should he need further anaesthetics.—We are, etc.,

T. R. AUSTIN
J. ANDERSON
J. RICHARDSON

British Military Hospital
Rinteln, Germany

1 Buckland R. W., and Avery A. F., *British Journal of Anaesthesia*, 1973, **45**, 518.
2 Buckett, W. R., Marjoribanks, C. E. B., Marwick, F. A., and Morton M. B., *British Journal of Pharmacology* 1968, **32**, 671.
3 Dundee, J. W., and Clarke, R. S. J., *British Journal of Anaesthesia*, 1973, **45**, 304.

Facial Sweating after Food in Diabetes

SIR,—Dr. P. J. Watkins (10 March, p. 583) having told me about facial sweating after food in 1972, I specifically sought this sign in 200 consecutive patients at our diabetic clinic and found two similar cases.

The first patient was a woman aged 32 with severe insulin-dependent, unstable diabetes for six years with advanced retinopathy, nephropathy, gut neuropathy, and orthostatic hypotension. For two years she had suffered profuse facial and neck sweating after chewing but obtained no relief from anticholinergic drugs. The second patient was a 78-year-old woman with three months' history of thirst, polyuria, and vaginitis. She was thin, normotensive, apparently otherwise healthy, and non-ketotic, with a fasting blood sugar of 180 mg per 100 ml. Gustatory sweating was noticed within two weeks of the onset of polyuria. There was no other evidence of peripheral or autonomic disturbance and no pupillary changes or lesions of the chest or brachial plexus. After diet and four weeks of glibenclamide the glycosuria disappeared and the fasting blood sugar fell to 120 mg/100 ml with disappearance of thirst, etc. Her gustatory sweating has continued, however, and is presumably her only evidence of diabetic autonomic neuropathy.

It is possible that "Watkins's sign" may be more common than has hitherto been realized.—I am, etc.,

A. FREEDMAN

Department of Medicine,
Prince of Wales Hospital,
Randwick, New South Wales

Rootless Wanderers

SIR,—Your leading article (7 July, p. 1) is very timely and I would like to make some further comment on it.

We psychiatrists working in the community have expressed for some time our very great concern over the lack of understanding by the legislators for the implementation of an efficient community psychiatrist service as part of the future comprehensive psychiatric set-up. The Local Authority Social Services Act 1970 unfortunately initiated the split between the hospital and community sectors of the service, and the proposed reorganization of the National Health Service will, I fear, finalize this dichotomy. The community sector of the psychiatric service plays an integral part in any overall psychiatric scheme, and one part cannot function efficiently without the other. The only way to build up such a service is, in my opinion to bring together all the psychiatric facilities, services, and personnel under one authority, the only logical one being the future health authority.

If it is proposed to run down and eventually phase out the psychiatric hospitals, as indeed appears to be Government policy, then this can be attempted only after, not before, efficient community psychiatric services have been established in the respective areas. Furthermore it should not be forgotten that the psychiatric units attached to district general hospitals equally require the help of the community psychiatric counterparts. So let us put the emphasis where it seems to fit best into the envisaged psychiatric service of the future—namely, to encourage the building up of appropriate community services under psychiatric guidance and management.—I am, etc.,

U. P. SEIDAL

London N.15

Excretion Urography in Acute Renal Failure

SIR,—We have read with great interest the very careful report of Dr. W. R. Cattell and others (9 June, p. 575) on urographic findings in acute renal failure. Over the past two years we have carried out a similar study on 25 oliguric patients without any obstructive disease. In 20 cases a diagnosis of acute tubular necrosis was made, based on the sudden onset of renal failure, the presence of an aetiological factor, typical urinary findings, and a typical clinical course including a diuretic phase and complete recovery. In five cases subacute glomerular or vascular disease was diagnosed on histological evidence (subacute glomerulonephritis in two, polyarteritis in two, and malignant

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