until the patient recovers by himself, is cured, dies, or is referred for a second opinion. The GP learns to make decisions quickly and most of them, in most practices, are right.

We could not escape a reference to deputising services. Dr Davies implies that the public is being duped into accepting an inferior service and then, in the next sentence, suggests approvingly that some patients may prefer the emergency locum to their own doctor. This confused attitude makes me think of a squash player trying to play forehand and backhand at the same time. Perhaps an excess of spleen has clouded Dr Davies's thinking. Very few people can talk calmly about deputising services. Some GPs, usually those who work in a large rota, regard their use as betraying shameful weakness. Others welcome them as saviours of their users' health and sanity. Newspaper columnists and politicians make great play of the deputy's having done a day's work in hospital before he comes on duty. Nobody in my recollection has ever complained that the family doctor on call for his practice has also done a full day's work and will do another tomorrow.

Further on the family doctor is told to "join the 20th century and return to the practice of medicine as we understand it today." If he has never been there how can he return to it? Who are "we"? Whose is the concept that family doctors are replacing the clergy as problem solvers and soothers? Do family doctors want to replace the clergy? In what school of alchemy did Dr Davies learn that catalysts act on one another? What does the Isis Centre have to do with the case? Why is there a change of person in the third paragraph under "primary medical care"—is it an example of the well-known conjugation, "I plan, you do, he is an idle slob"?

Dr Davies's plans for the future are a logical extension of his imperfect apprehension of the present. GPs, whatever they are called, have enough to do without taking on, unpaid, the work of miniconsultants. Specialisation in general practice has been tried and found unsatisfactory for doctors and patients.¹ If I have a hernia I want the man who repairs (not "corrects") it to be a competent surgeon, not a dilettante who will scream and turn pale if he uncovers something unexpected and nasty. Like most family doctors I diagnose, investigate, and treat not only the hypertensives and the subthyroids but the thyrotoxicos, maturity-onset diabetics, congestive cardiac failures, and sufferers from a host of other diseases.

There are bad hospitals, bad consultants, and bad GPs; there are fellows, readers, and research associates who work in ivory towers and, filled with the confidence born of ignorance, utter vapid bletherings. But it would be unfair, misleading, and scientifically reprehensible to generalise from the particular.

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¹ Spence, J C, *British Medical Journal*, 1975, 1, 390.

SIR,—Dr T F Davies (4 December, p 1376) refers to "two standards of care in the hospital service: the teaching centres and the rest" and he repeatedly uses that misleading and thoroughly objectionable term "centre of excellence."

I have worked in four of his "centres" and in four peripheral hospitals and I truly believe

that, for all but rare and bizarre conditions, the quality of care, both medical and nursing, is as good in the latter. The attempt to produce factual evidence for his statements is remarkably naive from a research associate in medicine. Hospital mortality is a poor index of quality of care in myocardial infarction, and in any case the results of five of the non-teaching hospitals are as good as, or in three cases better than, those of his two teaching hospitals.

Of all the letters Sir Alec Merrison will receive few will be as arrogantly misleading as the one you have just published.

J M GATE

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Defence against bacterial drug resistance

SIR,—In his excellent review (16 October, p 933) Professor L P Garrod discusses the advisability of restricting the local application of antibiotics. He points out that it is important to preserve bacterial sensitivity to gentamicin and compliments the manufacturers of this antibiotic for not making a preparation in tablet form. However, we are rather worried about the use of the topical preparation of this compound and its potential for promoting resistance.

We have recently seen an outbreak of gentamicin-resistant staphylococcal infection involving 23 patients in a dermatology unit which seemed to be associated with a high usage of topical gentamicin. The total amount of this preparation used in the hospital was 15 804 15-g units in 1974-5 and 23 020 15-g units in 1975-6, of which the pharmacy estimated over 90% were used in the dermatology unit. Our findings are to be published in detail elsewhere.¹ At the beginning of the outbreak the use of topical gentamicin was severely restricted and the number of patients involved then fell dramatically (see table). As can also be seen from the table the number of new patients with gentamicin-resistant staphylococci has since remained at a low level. The reason for the continuing isolations from new patients is obscure, but resistant organisms seem capable of surviving in lesions for extended periods—six and nine months respectively in two patients with varicose ulcers.

Isolations of gentamicin-resistant staphylococci

Month	No of new patients involved
December 1975	3
January 1976	1
February 1976	6
March 1976	15*
April 1976	3
May 1976	1
June 1976	1
July 1976	0
August 1976	2
September 1976	5†
October 1976	6‡

*Restriction of topical gentamicin usage.

†Includes two patients not directly associated with Belfast City Hospital.

‡Includes three patients not directly associated with Belfast City Hospital.

An unfortunate recent trend is the isolation of gentamicin-resistant organisms both from patients in other hospitals and from patients of general practitioners (see table). Perhaps this trend is not surprising since in Northern Ireland 117 528 15-g units of topical gentamicin preparations were prescribed by general practitioners in a typical four-month period, October 1975-January 1976 (source: Chief Pharmacist, DHSS, for Northern Ireland).

We certainly agree with Professor Garrod's

statement that "a decision to apply gentamicin cream should not be taken lightly" and hope that his words will be heeded and will lead to a much reduced usage of these preparations.

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¹ Wyatt, T, *et al*, *Journal of Antimicrobial Chemotherapy*. In press.

Paroxysmal brain stem dysfunction in multiple sclerosis

SIR,—We agree with the need to be aware of the possibility that paroxysmal disturbances may be the first manifestation of multiple sclerosis (MS), as in the patient reported by Drs W H Perks and R G Lascelles (13 November, p 1175), but their statement that such paroxysmal brain stem disturbances have not previously been reported as the sole presenting feature of MS is not correct. Several of the references given by McAlpine *et al*¹ in their section on short-lived and paroxysmal attacks include case reports in which such symptoms preceded other evidence of MS. In six of our 32 patients² with paroxysmal disturbances originating in the brain stem or spinal cord these were the initial manifestations of MS. In a more recent report of different types of paroxysmal attacks in 22 patients with MS³ there were at least four in whom paroxysmal brain stem disturbances were the first symptom of MS.

We believe that such paroxysmal disturbances occur as the first manifestation of MS more commonly than is generally realised. They should be distinguished from transient ischaemic attacks as the paroxysmal disturbances of MS usually occur in a younger age group, tend to be of briefer duration, recur more frequently, and respond to carbamazepine.¹ It is important to recognise them not only because of the rewarding response to carbamazepine but also in anticipation of the day when effective treatment given early may prevent the irreversible stages of the disease.

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¹ McAlpine, D, Lumsden, C E, and Acheson, E D, *Multiple Sclerosis—A Re-appraisal*, 2nd edn, pp 185-190 and 252. Edinburgh, Livingstone, 1972.

² Espir, M L E, and Millac, P, *Journal of Neurology, Neurosurgery and Psychiatry*, 1970, 33, 528.

³ Osterman, P O, and Westerberg, C-E, *Brain*, 1975, 98, 189.

Penicillin-insensitive pneumococci

SIR,—I wish to correct some misconceptions regarding pneumococci with diminished sensitivity to penicillin. Professor L P Garrod writes (16 October, p 933): "Resistant strains [of pneumococci] were first encountered in New Guinea where penicillin was being used in a seemingly not very satisfactory way for the prevention of pneumonia, to which the inhabitants are said to be peculiarly subject for climatic reasons. Two others were then detected in Australia, and later others have been reported in North America, and one

causing meningitis in an infant in Oxford. . . . The degree of resistance is not great (inhibition by 0.5 instead of 0.02 µg/ml) and should be overcome by increased doses . . . but there must be some fear that the change will not stop there."

So far as I am aware, the first penicillin-insensitive (PR) pneumococci to be encountered were isolated from subjects in New South Wales and South Australia in 1967.^{1,2} Although the initial PR pneumococci to be met with in New Guinea were from subjects at Anguganak in the Sepik District, where a controlled trial of procaine penicillin was in progress in the hope of reducing the incidence of sometimes fatal pneumococcal pneumonias, the first PR strain to be isolated there was from a subject in the control group, who did not receive penicillin.^{2,3} As pointed out by Douglas and Sturt,³ who were responsible for this trial, it has never been a custom in New Guinea to use penicillin prophylaxis for pneumonia. PR pneumococci have been isolated from subjects in five other districts of New Guinea, including the Tobriad Islands, all of which are remote from Anguganak. Furthermore, apart from the type 4 and 19 strains of PR pneumococci found at Anguganak, PR pneumococci of eight other serotypes (types 6, 11, 14-16, 23, 34, and 35) have been isolated from New Guineans.⁴ I therefore believe that penicillin-insensitive pneumococci probably evolved independently in several centres of New Guinea.

The range and degree of resistance to penicillin are greater than Professor Garrod states. The most resistant strains so far encountered have a minimum inhibitory concentration of 2.0 µg benzylpenicillin/ml (resistant ratio 100).⁵ Fortunately, PR pneumococci are either fully sensitive to ampicillin and amoxycillin or show only slightly diminished sensitivity.^{6,7}

New Guinea is one of a number of tropical countries where there is a high incidence of pneumococcal pneumonia. It is hoped that the introduction of polyvalent vaccine, prepared from pneumococcal capsular polysaccharides, will reduce the incidence and mortality rate of this important infection.

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¹ Hansman, D, and Bullen, M M, *Lancet*, 1900, 2, 264.
² Hansman, D, et al, *New England Journal of Medicine*, 1971, 284, 175.

³ Douglas, R M, and Sturt, J, *Medical Journal of Australia*, 1975, 1, 82.

⁴ Hansman, D, et al, *Medical Journal of Australia*, 1974, 2, 353.

⁵ Hansman, D, Devitt, L, and Riley, I, *British Medical Journal*, 1973, 3, 405.

⁶ Hansman, D, *Medical Journal of Australia*, 1975, 2, 740.

⁷ Hansman, D. To be published.

*. *We sent a copy of this letter to Professor Garrod, whose reply is printed below.—ED, BMJ.

SIR,—It is useful to have this further information, but I cannot plead guilty to having misquoted Dr Hansman. In his original description of the trial of monthly injections of procaine penicillin in New Guinea (ref 2) PR pneumococci were reported to have been isolated from 11 treated and 4 control subjects. Their occurrence in controls could presumably be explained by transmission from treated subjects. As I understood the paper the treat-

ment, which in fact had been going on for 10 years, was thought to be responsible. It is difficult to believe that resistance can be the result of spontaneous mutation without exposure to the antibiotic. The degree of resistance in these strains was as stated by me (inhibition by 0.5 µg/ml) and is given in his latest paper (ref 7) as varying from 0.1 to 1.0 µg/ml.

L P GARROD

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Alcohol-induced cushingoid syndrome

SIR,—You will be relieved to hear that after reading the interesting short report by Dr A G Smals and his colleagues (27 November, p 1298) I have consigned my next paper to you to the wastepaper basket. Over the past few years I have accumulated details of eight alcoholics (five men, three women) with features of Cushing's syndrome. All had histological evidence of alcoholic hepatitis or cirrhosis. I have not yet seen a similar clinical picture in patients with other types of liver disease nor in alcoholics who did not have liver damage. The question why only some alcoholics develop this syndrome might equally well be asked of the other physical manifestations of alcoholism.

Two of my women patients were not known to be alcoholics until their husbands came to me in some distress, having found evidence of secret drinking after their wives' admission. This is an important point since, unlike Dr Smals's patients, the signs of Cushing's syndrome did not always disappear in hospital and such patients are at risk of inappropriate investigation and treatment. I have come to regard detailed inquiry about the possibility of alcoholism as mandatory in patients with Cushing's syndrome, especially women.

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Is there a future for community medicine?

SIR,—Amid confusion and uncertainty surrounding the specialty of community medicine has come the plea from Dr D H Stone (30 October, p 1086) for a "full-scale, uninhibited professional debate" on the subject. As an illustration of what I believe to be the essence of community medicine I should like to report the findings of an investigation into coronary thrombosis at the Leyland vehicle plant at Bathgate, Scotland.

In the summer of 1975, during discussions on the Health and Safety at Work Act, it became apparent that workers at the plant were alarmed at the apparently excessive number of men falling ill and dying from heart attacks. A preliminary inquiry showed that, although many people were indeed becoming suddenly ill at work, not all were suffering from coronary disease. In a more detailed study the at-risk population by age group was estimated for the mid-1975 work force of 4500. Female and staff employees were excluded because of small numbers. Their coronary experience during 1975 was obtained from the company medical records and the age-specific incidence and fatality rates calculated and compared with those from a community study into acute coronary heart attacks in Edinburgh.² The standardised morbidity ratio of 104 was not significantly raised and no excess mortality was found; this is in agreement

with findings at the Leyland plant at Cowley, Oxford, where production line workers have a similar coronary experience to the general population of the area.³

The point of the investigation, however, was not an empirical epidemiological study. Each shop steward was given a cyclostyled sheet explaining in lay terms the conclusions, and in this way it was possible to allay some of the anxiety of the workers.

Community medicine is not simply "the study of health and disease in populations."⁴; that is the field of politics. Neither is it epidemiology, statistics, social policy, the social sciences, and management; these are merely the tools of the trade.⁵ Nor is it the abstracted empiricism of many epidemiological surveys. Community medicine involves a dialogue between health workers on the one hand and people in every aspect of their daily lives on the other, finding out and investigating their fears, their needs, and their wants and explaining in return what medicine can do for them. Herein lies the future of community medicine.

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¹ Stone, D H, *British Medical Journal*, 1976, 2, 1086.

² Armstrong, A, et al, *British Heart Journal*, 1972, 34, 67.

³ Baxter, P J, et al, *British Journal of Industrial Medicine*, 1976, 33, 1.

⁴ Scottish Home and Health Department, *Doctors in an Integrated Health Service*, Edinburgh, HMSO, 1971.

⁵ Cameron, D, *British Medical Journal*, 1976, 1, 1210.

SIR,—The views expressed in Dr D H Stone's article (30 October, p 1086) are depressing reading, particularly coming from a lecturer in community medicine, and these views must be challenged.

We in community medicine must accept that we are not clinicians and must avoid pathetic attempts to gain acceptance by undertaking clinical work, which can at best be spasmodic and at the expense of community medicine proper. To accept the need to be part-time clinicians implies that community medicine is unsatisfactory to the doctors practising it and that its contribution to the welfare of patients is insufficient to merit recognition by clinicians in its own right.

Our function, broadly, is to study the natural history of disease in our populations (and we include in the term "natural history" both conventional epidemiology and the way in which health services are organised to deal with health problems) and to attempt by whatever means seem feasible and appropriate to influence the natural history in favour of the human population. Such studies are not, as Dr Stone suggests, the province of academics but are the very meat of our professional lives and represent the work for which we are trained. The natural history of disease can be influenced in many ways, including administrative action. Where such action will be beneficial we should encourage and where appropriate participate in it, but we should not regard it as the only tool at our disposal.

The advisory structure in the reorganised NHS presents us with an ideal forum in which to discuss with colleagues, in both hospital and general practice, the implications of our studies, and as much benefit is likely to come from such discussions, based on the hard evidence we have accumulated, as from administrative effort. The discussion of such studies should suggest what the needs of groups of patients are (whether grouped on a geographical basis or on the basis of a common disease) and how best general practitioners, hospital doctors,