practice, though once the patient is euthermic and has re-established an adequate peripheral blood flow venous samples are satisfactory for all follow-up studies other than blood gas analysis and acid-base assessment.

The wide scatter of results for the amylase and glucose levels in our series (29 December 1973, p. 757) probably reflects a major modifying influence of the underlying illnesses on those effects which can be attributed to the hypothermia alone. None the less, as we mentioned, the effects of hypothermia on blood amylase and glucose levels are well established, and our serial studies, like those of others¹⁻³ confirmed that abnormal results quickly revert to normal after the patient becomes euthermic. The comment of Professor J. Malins and Dr. C. H. Walsh (26 January, p. 159) that elevated blood amylase levels may occur in subjects with uncontrolled diabetes mellitus without any evidence of pancreatic disease, while correct, scarcely applies to accidental hypothermia, as more than 80% of cases coming to necropsy have evidence of gross pancreatic damage.⁴

Our diagnosis of ketoacidosis as a part cause for the metabolic acidosis noted in all our hyperglycaemic patients whom we considered for insulin therapy was based on the presence of ketones both in the breath and in the urine. We also found that, irrespective of whether or not ketoacidosis was present, organic acids such as lactic and pyruvic acid certainly contributed to the severity of the metabolic acidosis.

Dr. Sloan surely intended to comment that acid-base calculations made from arterial samples can be misleading unless they are corrected from their value at electrode temperature to what they would be at the patient's (that is, hypothermic) temperature. The question of therapy to correct acid-base disturbances is a controversial matter. Our practice during these investigations has been to avoid medical intervention, as during the slow spontaneous rewarming of our patients the severity of the metabolic acidosis usually gradually lessened and seldom increased. Presumably the slowness of the rewarming process allows the capacity of the liver to metabolize organic acids to increase in pace with their increase rate of "wash-out" from the muscles into the circulation. "Corrected" blood gas and acid-base values can certainly be successfully used to guide therapy in these patients,⁵ but whether it is necessary to use such values is debatable.⁶ Moreover, even "uncorrected" values can be shown to correlate with other serum enzyme evidence of tissue damage in these patients,⁷ though of course this does not imply that the relationship is necessarily a causal one.—We are, etc.,

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1 Holmes, R., and Emslie-Smith, D., *Lancet*, 1961, **1**, 1002.

2 Murphy, E., and Faul, P. J., *Journal of the Irish Medical Association*, 1963, **53**, 4.

3 Henneman, D. H., Bunker, J. E., and Brewster, W. R., *Journal of Applied Physiology*, 1958, **12**, 164.

4 Mant, A. K., *British Journal of Hospital Medicine*, 1969, **2**, 1095.

5 LeVingham, I. McA., and Mone, J. G., *Lancet*, 1972, **1**, 534.

6 Patterson, R. H., jun., and Sondheimer, H. M., *Journal of Surgical Research*, 1966, **6**, 19.

7 Maclean, D., Griffiths, P. D., and Murison, J., *Clinica Chimica Acta*. In press.

Sensitivity of *Haemophilus influenzae* to Cephalosporins

SIR,—Like Dr. S. Selwyn (2 March, p. 388) we also have been interested in the antibacterial activity of cephradine against *Haemophilus influenzae*, particularly in comparison with other cephalosporins. The results we have obtained for the minimum inhibitory concentration (M.I.C.) of seven cephalosporins for 36 strains of *H. influenzae* are shown in the table. From these results, none of the cephalosporins could be said to be very active against *H. influenzae*. The activities of the two orally administered derivatives, cephalexin and cephradine, are very similar and they are the least active. These findings are similar to those reported by Williams and Geddes.¹ We used an inoculum of 10^3 – 10^4 organisms and though the strains were not typed they were all isolated from the sputum of patients with exacerbations of chronic bronchitis or post-operative chest infections and therefore presumably 95% were non-capsulated.² We have not estimated M.I.C.s for *H. influenzae* known to be Pittman type band so cannot dispute the data of Dr. Selwyn. However, the most common indication for antimicrobial treatment of *H. influenzae* in clinical practice in Britain is chronic bronchitis, and non-capsulated strains are important here. Clinical cases involving *H. influenzae* outside the respiratory tract are much less common and it is doubtful if any cephalosporin could be recommended with confidence to treat, for example, *H. influenzae* meningitis.

Dr. Selwyn suggests that an inoculum of approximately 10^5 organisms should be used for testing *H. influenzae*. We have found that the M.I.C.s of all cephalosporins for *H. influenzae* are susceptible to inoculum size, and increasing the inoculum markedly increases the M.I.C. It is not unusual to find that a 100-fold increase in inoculum results in a 10-fold increase in the M.I.C. We cannot of course speculate on the relevance of this finding in the clinical context, but it must make comparison of results from different laboratories extremely difficult.

Though our in vitro results would suggest that cephradine would be an unsuitable drug with which to treat *H. influenzae* infection, the ultimate arbiter must be the clinical results, which are notoriously difficult to assess in chronic bronchitis. The only published work³ we can find on the use of cephradine in respiratory infection reported a fair or poor response in two of three patients with pneumonia and a poor response in one of three patients with

bronchitis. The in vitro activity of cephradine closely parallels that of cephalexin, and Pines⁴ reported poor clinical results with cephalexin in chronic bronchitis, so it is difficult to take an optimistic view of cephradine.

We would therefore agree with Dr. J. D. Williams and Mrs. J. Andrews (26 January, p. 134) that "oral cephalosporins are not indicated" in haemophilus infection.—We are, etc.,

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1 Williams, J. D., and Geddes, A. M., *British Medical Journal*, 1973, **2**, 613.

2 Turk, D. C., and May, J. R., *Haemophilus influenzae: its Clinical Importance*, p. 45. London, English Universities Press, 1967.

3 Mogabgab, W. J., *Journal of the Irish Medical Association*, 1973, **66**, no. 6 (suppl.), p. 18.

4 Pines, A., Macquet, V., and Lafitte, Ph., *Postgraduate Medical Journal*, 1970, **46**, (October Suppl.), p. 128.

Serological Tests for Amoebiasis

SIR,—Too much emphasis can be placed upon serological tests for amoebiasis (22 December 1973, p. 746). At the best serology can assist in diagnosis but certainly cannot replace the microscope. In Saskatchewan, using the indirect haemagglutination (I.H.A.) test, we have found that there is no full correlation between serological reactions and the presence or absence of parasites in the stools.¹ We have seen several children with negative serological results and *Entamoeba histolytica* parasites in the stools. Dr. G. R. Healy, of the U.S. Department of Health Education and Welfare,² suggests a correlation of 85%. Dr. Healy has performed I.H.A. tests on sera from this area of Canada since 1964 and has found several cases in which sera taken from children during the acute and convalescent stages of intestinal amoebiasis have given negative reactions.³

More success has been had with an intradermal reaction,⁴ where we have had 100% correlation with active intestinal amoebiasis and have shown it to be a reasonable indicator to the prevalence of disease in a community.¹ However, it has been found that, in its turn, the intradermal reaction tends to increase the I.H.A. titre at a subsequent test.⁵

Both intradermal and serological reactions have been investigated as a possible escape from the drudgery of the microscope, but I feel that both should be used with caution, maybe to select patients from whom stools should be examined. If there are any grounds for clinical suspicion, however, then a negative intradermal or serological reaction should be ignored and repeated stools examined. So far we have found the most successful treatment regimen to be a

Antibiotic Sensitivity of 36 Strains of *H. influenzae* to Seven Cephalosporins

Cephalosporin	Minimum Inhibitory Concentration (μg/ml)									
	25	12.5	6.25	3.12	1.56	0.78	0.39	0.19	0.09	0.04
Cefamandole	—	—	1	—	1	1	12	7	8	3
Cefazolin	—	1	3	2	8	10	4	2	3	2
Cephalexin	—	—	—	3	3	7	11	6	—	6
Cephadrine	—	—	—	7	10	8	5	—	1	5
Cephacetrile	1	10	1	2	8	4	2	5	2	—
Cephalexin	2	7	11	8	4	2	2	—	—	—
Cephradine	7	6	10	3	2	4	3	—	—	1

combination of metronidazole and diloxanide furoate, metronidazole by itself not providing a good parasitological cure.⁶—I am, etc.,

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¹ Miller, M. J., Scott, F., and Foster, E. F., *American Journal of Tropical Medicine and Hygiene*, 1973, 22, 331.

² Healy, G. R., paper presented to the American Public Health Association Annual Meeting, October 1967.

³ Scott, F., unpublished data.

⁴ Miller, M. J., and Scott, F., *Canadian Medical Association Journal*, 1970, 103, 253.

⁵ Meerovitch, E., and Scott, F., *Journal of Parasitology*, 1973, 59, 1134.

⁶ Eaton, R. D. P., Scott, F., and Meerovitch, E., *Canadian Journal of Public Health*, 1973, 64, monograph suppl., p. 47.

Anticomplementary Activity of Serum and Immune Complexes

SIR,—In the excellent report by Dr. M. J. Dillon and others on the outbreak of epidemic neuromyasthenia at the Hospital for Sick Children, Great Ormond Street (23 February, p. 301) I was astonished to read the statement, unsupported by any reference, that "it is now established that anticomplementary activity is a simple test showing the presence of immune complexes in the serum."

There seems little doubt that circulating complexes can be a cause of anticomplementary activity, but there are other causes. In view of the facts that (1) the anticomplementary activity of serum increases with ageing of the specimen, (2) denatured immunoglobulins are anticomplementary (and the extent of denaturation also increases with ageing), and (3) heating serum at 56°C for 30 minutes produces some denaturation of many proteins it would be naive in the extreme to assume that this simple test allows of a single interpretation.

It would be extremely valuable to have a reliable test for circulating complexes of antigen and antibody, but until such a test is available Dr. Dillon and his colleagues should be more cautious in invoking this important pathophysiological mechanism.—I am, etc.,

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General-practitioner Deputizing Services

SIR,—On the basis of a lop-sided experience of general practitioner deputizing services in one region Dr. Keith Wells condemns such services in general (9 March, p. 454).

His strictures would not in any case apply to the service in this town. All our deputies have to satisfy the local medical advisory committee that their experience is sufficient for the work that they will be required to undertake. The committee's criteria for admission of deputies are as follows: (1) they must have been registered for at least four years (three years or more on the permanent register); and (2) they must have had at least three months' general practitioner experience; or (3) they must be practitioners already established as principals in general practice. In fact the majority of deputies are

general practitioners already in practice locally.—I am, etc.,

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Shortage of Trainees in Adult Psychiatry

SIR,—Mr. F. S. A. Doran (16 March, p. 513) must be joking when he says there is a "large surplus" of trainees in adult psychiatry. This collective delusion of the Department of Health and Social Security was firmly refuted by Professor Gerald Russell last year.¹ However, the repeated statements in *Health Trends* and elsewhere that there was such a surplus may well have done a good deal of harm—for example, in discouraging doctors of high ability from entering the specialty.

Anyone who has served on a consultant appointment committee in adult psychiatry north of Potters Bar will agree that there has been an alarming fall in both numbers and quality of candidates over the past decade. Many posts are unfilled, particularly in the northern regions, and in many cases a committee is unable to recommend an appointment or even to meet because there are not enough candidates with even minimal qualifications. In other cases the majority of the committee reluctantly acquiesces in a rather dubious appointment in face of urgent pleas from the local hospital representatives that their service cannot carry on unless someone is recruited. As older consultants retire (or sometimes emigrate) there is an increasing danger of the quality of the psychiatric service going into a downward spiral of lowered standards and even poorer recruitment. It would be a tragedy if much of the progress of the past 20 years were to be lost in this way.

One factor which certainly contributes to this situation is the gobbling up of the most promising recruits to psychiatry by the few larger teaching centres. In spite of all this input, the output from them in terms of trained psychiatrists going out to work in regional hospitals is minimal. It is indefensible that such a richness of staffing and resources should make so small a contribution to the needs of our population. Therefore it is to be hoped that the D.H.S.S. will not retreat from its policy of redistributing training posts in spite of the anguished howls of protest that come from the teaching centres.—I am, etc.,

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¹ Russell, G., *News and Notes Suppl. to British Journal of Psychiatry*, 1973, (August), p. 8.

Consultant Discontent

SIR,—In your leading article (16 March, p. 467) you quote a common complaint of consultants that their rise in pay over the past 10 years has been less than that of general practitioners and junior hospital staff, the figures being 71%, 108%, and 150% respectively.

There are many G.P.s and junior hospital staff who would support a demand for a fair rate of pay and a better contract for consultants. This support will dwindle if statistics such as you have quoted are used out of

context to achieve their demands. There are many G.P.s who remember the appalling conditions of service, hours of duty, and pay when they did their house jobs which consultants in those days did little to improve; and many G.P.s who remember the humiliating conditions and pay structure in general practice from the 1950s to 1966. The statistics quoted above merely reflect the necessary action that was required to correct these conditions; they do not in themselves support a claim for consultants to have a particular percentage rise. There is great justice in the consultants' demand for a fairer contract and better pay; but let it be achieved not by stepping on the shoulders of their colleagues but by rational demands agreed among themselves, vigorously pursued, and supported I hope, by G.P.s and junior hospital staff.

There appear in the eyes of many G.P.s to be three grades of consultant—each determined by such criteria as length of training, responsibilities, skills, work load, etc. One grade is quite clearly superior to the average G.P.; the next equal to the G.P.; and a third, smaller, group possibly inferior to the G.P. But the consultants' methods of payment, their secret distribution of merit awards, and the vastly differing amounts of money they earn from private practice make their whole pay structure seem irrational and unfair—in short, a muddle. To increase their pay by a percentage figure without a complete restructuring is to perpetuate the present difficulties and to lead to intraprofessional rivalry and a win for the Government. Let the consultants organize themselves and put their house in order as the G.P.s and junior hospital staff have done and then they will have support from all the profession.—I am, etc.,

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SIR,—I would support Dr. Ronald Gibson's call for unity within the profession (9 March, p. 459) and would accept his statement that the general practitioners have built up a better negotiating machinery than have the hospital doctors. It should be remembered, however, that they have had state remuneration for twice as long. They also work largely from independent premises and could threaten to withdraw from the N.H.S. without great hardship to their patients. He does not mention these important facts and he does not tell us that the G.P.s had the support of the hospital staff and indeed of the whole profession, through the B.M.A., when they obtained their charter. We have yet to see evidence of similar support for the hospital staff from the G.P.s.

Of course the B.M.A. is not a G.P. Club, but they are far the strongest party and until recently had the major say in the Representative Body and the Council. I suspect that Dr. Gibson, whose ability I greatly admire, played his part in helping to improve this. I hope that he may now use his persuasion to gain for us the support of the G.P.s. I believe that if hospital doctors felt that they had the active support of their 70,000 colleagues behind them the majority would gladly return to the B.M.A.—I am, etc.,

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