eventually lead to a backlash which will destroy confidence and the necessary need for vigilance and referral of suspected cases of the syndrome. From its nature, this confidence can be built up only slowly, after years of painstaking work, but will be rapidly lost by a few injustices.—I am, etc.,

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Unusual Complication of Use of Sengstaken-Blakemore Tube

SIR,—We present, below, a previously undescribed hazard of the use of the Sengstaken-Blakemore tube.

The patient was a 55-year-old male alcoholic who presented with bleeding oesophageal varices. During the course of management a Sengstaken-Blakemore tube was passed and inflated with a Gastrografin and saline solution.^{1,2} The position of the tube was checked radiologically.

Despite sedation the patient was restless and succeeded in pulling the tube so that the oesophageal balloon was partly out of his mouth. The oesophageal balloon was immediately deflated, but no Gastrografin could be aspirated from the gastric balloon. The tube was passed back to the stomach and restored to the traction position. An x-ray (fig. 1) showed the gastric balloon still inflated and in position, but the distance between it and the now malpositioned oesophageal balloon showed that the patient's sudden traction had pulled the gastric balloon a considerable distance down the tube. Gastrografin injected via the gastric balloon's filling lumen did not increase the balloon's size, nor could it be reaspirated, showing that the balloon had been disconnected from its filling lumen.

FIG. 1.—X-ray showing position of balloons after traction by patient.

Later the patient again pulled on the tube and, despite the inflation of the gastric balloon, he succeeded in extracting it, fortunately without damage to the oesophagus. Fig. 2 shows that the gastric balloon had now been forced to the tip of the Sengstaken-Blakemore tube. Its original position is revealed by the two holes of the filling lumen just below the oesophageal balloon.

This case illustrates that the gastric balloon of the Sengstaken-Blakemore tube can be forced away from its filling lumen to a position where it can be neither filled nor deflated. This was done in this case by a restless patient, but there is a danger of the

FIG. 2.—Tube after removal by patient.

same complication arising from traction applied therapeutically to the tube—a procedure criticized on other grounds by Pitcher.³ This is potentially dangerous for in this situation there may be no way in which the gastric balloon can be safely removed without a laparotomy and gastrotomy.

To some extent the dangers of this complication may be minimized by light sedation, but too heavy sedation increases the risks of inhalation of secretions. The problem could be avoided by redesigning the tube with continuous rubber balloons on the lines of the Foley urinary catheter balloon.—We are, etc.,

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¹ Read, A. E., Dawson, A. M., Kerr, D. N. S., Turner, M. D., and Sherlock, S., *British Medical Journal*, 1960, 1, 227.

² McPherson, A. I. S., *Scottish Medical Journal*, 1964, 9, 243.

³ Pitcher, J. L., *Gastroenterology*, 1971, 61, 291.

Apathetic T-3 Toxicosis

SIR,—Drs. P. D. Fairclough and G. M. Besser (2 March, p. 364) rightly emphasize the occult nature of many cases of hyperthyroidism, be it due to thyroxine or triiodothyronine. Their patient is yet another example of the situation in which a prolonged period of ill health precedes a firm diagnosis of overt hyperthyroidism.

There is evidence that a prethyrotoxic state exists. In elderly subjects, in whom the cardiovascular system is relatively vulnerable, symptoms and signs referable to the heart and circulation may be due to this form of occult hyperthyroidism. The diagnosis can be suspected when only the serum protein-bound iodine is persistently raised and in eight such cases¹ each developed obvious hyperthyroidism. A similar syndrome may well arise with triiodothyronine, though chemically the diagnosis is even more difficult to establish, as Drs. Fairclough and Besser have shown.—I am, etc.,

CECIL SYMONS

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¹ Symons, C., Richardson, P. J., and Wood, J. B., *Lancet*, 1971, 2, 1163.

Patients' Attitudes to Medical Students in General Practice

SIR,—As a student I read with great interest Dr. H. J. Wright's observations (2 March, p. 372) and would like to add some of my own.

I have recently completed an initial course with a group practice in Manchester. Though this was apparently not recognized by its patients as a teaching practice, I very rarely felt that my presence was in any way resented by the patients. On the contrary, many expressed surprise that they might be expected to object (this discounts the small number of more senior patients who feared that their life-long doctor was being replaced by a young unknown entity and whose fears were rapidly dispelled). Nevertheless, I would have considered the courteous and correct opening to any consultation to be to introduce those present, if necessary explaining the capacity in which they are present, and to ascertain whether the patient objects to their presence. I should have expected such practice to be a common, indeed natural, procedure and was surprised that Dr. Wright thought it necessary to make such a recommendation.

My experience and that of other students, though somewhat limited, indicates that the manner of introduction may have a considerable effect on acceptance by the patient. The term "medical student" implies that the patient is being used as a teaching object, an idea which the presence of two or more students may potentiate, and any such feeling might become a barrier to doctor-patient rapport. The patient-doctor relationship is more personal in general practice than in hospital and therefore to allow the presence of a student at a consultation is an even greater privilege granted by the patient. I believe that the presence of more than one student is an abuse of this privilege.

The introduction of the student as a "student doctor" or, with a little poetic licence, a "young doctor," though meaningless terms, considerably aided acceptance by the patient and also helped to encourage participation by the student. While I was under the guise of a "young doctor from the Infirmary" my presence was not objected to during consultation or physical examination, nor indeed was my examination of the patient (which included an internal examination where indicated). The only occasions on which I encountered uneasiness involved patients with psychosocial problems wishing to discuss them with their own doctor; I believe the unease was due to intrusion by another individual rather than specific objection to a student. In such a situation I found it necessary to withdraw and become a very passive observer or to leave the consulting room altogether.—I am, etc.,

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Acute Pancreatitis and Diabetic Ketoacidosis in Hypothermia

SIR,—May we support Dr. R. E. G. Sloan's comments (9 February, p. 245) concerning the need to use arterial blood samples for all biochemical estimations made on hypothermic patients. This has always been our

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