tion by binding to ristocetin and not by an effect on the platelets or the ristocetin co-factor. There is no evidence that our observation reflects an influence of Haemaccel on the haemostatic mechanism in vivo. All evidence suggests that this inhibition is solely an in-vitro phenomenon.—We are, etc.,

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Tests for Lactose Malabsorption in Adults

SIR,—In your leading article, "When Does Lactose Malabsorption Matter in Adults?" (17 May, p. 351) a review of the methods introduced for the diagnosis of lactose intolerance was presented, and included broad screening tests such as stool pH, symptomatology after lactose ingestion, lactose-barium meal, and carbon-14 breath test as well as the more widely used 50-gram oral lactose tolerance test with measurement of the blood glucose rise—a technique requiring multiple blood samples. "Final proof of the diagnosis has to be obtained by measuring the lactase activity of a jejunal biopsy specimen." No mention was made in the leader of analysis of breath hydrogen (H_2) by gas chromatography following lactose ingestion. Calloway[1] and Levitt[2] first showed that breath H_2 concentration rises when malabsorbed lactose is fermented in the large bowel, and they suggested that breath H_2 may be used as a measure of lactose malabsorption.

We have recently completed a study[3] comparing intestinal lactase activity with symptomatology, blood glucose rise, and breath H_2 production in a group of patients with diarrhoea. We found breath H_2 to be as reliable as the blood glucose and better than symptoms in the diagnosis of hypolactasia. As it does not involve multiple venepunctures it is more acceptable to patients and it is also easier to apply from the technical point of view.[4] Furthermore, our experience agrees with that of Isokoski[5] and disagrees with your suggestion that symptomatology may be used as a reliable test of hypolactasia.

We would also argue against the suggestion that intestinal lactase activity is the final proof of lactose malabsorption. For example, in the coeliac syndrome jejunal lactase activity may be reduced but lactose absorption still occurs further down in the ileum. Thus the sugar can be said to have been malabsorbed only if it passes through to the caecum, whereupon its subsequent fermentation with evolution of H_2 may truly be claimed as a measure of its malabsorption.—We are, etc.,

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1 Calloway, D. H., Murphy, E. L., and Baver, D., *American Journal of Digestive Diseases*, 1969, **14**, 811.
2 Levitt, M. D., and Donaldson, R. M., *Journal of Laboratory and Clinical Medicine*, 1970, **75**, 937.
3 Metz, G. L., *et al.*, *Lancet*, 1975, **1**, 1155.
4 Metz, G. L., *et al.*, *Clinical Science and Molecular Medicine*, 1975. In press.
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Humidification of Inspired Air

SIR,—I read your leading article on this subject (26 April, p. 157) with pleasure. I beg you, however, to allow me to make one remark about the last sentence of the article.

Neither a jug of water nor "more effective humidifiers" will give appreciable improvement in humidification during cold periods if great care is not taken to pull thick curtains over the doors and windows. The relative humidity of the heated room depends on its coldest spot. Whatever amount of water evaporates in a room at a temperature of, say, 18°C will settle down on the window pane at, say, 0°C until maximum humidity at 0°C is reached. Since heating (above all central heating) usually leads to a very low relative humidity it is unrealistic to hope that one can really increase it with a simple jug of water or more sophisticated humidifiers as long as the fluid will only rain down on the cold window panes. This does not help breathing, nor does it save antique furniture.—I am, etc.,

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Arthritis of Hepatitis: the Tourniquet Test

SIR,—A serum-sickness-like illness manifesting as migratory polyarthritis, fever, and urticaria has been recognized as a prodromal phase of hepatitis both positive and negative for hepatitis B surface antigen (HBsAg). Fernandez and McCarty[1] reported a patient with hepatitis and arthritis in whom severe urticaria developed in the arm distal to the point of application of a tourniquet. We report here (1) the occurrence of three different skin lesions and (2) the effect of the tourniquet test in a documented case of HBsAg arthritis.

A 16-year-old non-addict male presented with a five days' history of malaise, headache, fever, and polyarthritis of the proximal interphalangeal and metacarpophalangeal joints. Itching and urticaria developed two days later. On examination, all the affected joints were swollen, tender, and hot. A scaly erythematous macular rash was evident over each of these joints. Giant urticarial lesions were present over the upper extremities. No icterus was detected. The liver was 2 cm below the costal margin, soft, and non-tender. Initial studies revealed a normal blood count, platelet count, E.S.R., and urine analysis. When the results of serum bilirubin (total 22·2 μmol/l (1·3 mg/100 ml), direct 7·7 μmol/l (0·45 mg/100 ml)) and SGOT and SGPT (400 and 450 Franklin units respectively) were available arthritis of hepatitis was considered. Further studies showed the presence of HBsAg in the serum, C_4 690 (normal 900-1500 mg/l) and C_4 100 (normal 100-510 mg/l). Haemoglobin Ab could not be detected in the serum. Total serum proteins, antinuclear antibody, L.E. cell, rheumatoid factor (latex), V.D.R.L., and heterophile antibody tests, and creatine, phosphokinase, and aldolase activities were normal or negative. He became jaundiced one week after the onset of the arthritis and shortly afterwards a petechial rash was noticed over the chest. The serum enzymes reached a peak on the 12th day of the illness (SGOT 1060, SGPT 2750 F.U.). When the tourniquet test was done itching developed distally but no skin lesions appeared. The subsequent course was uneventful. One month after the jaundice has appeared, serum hepatitis B antibodies could be demonstrated and the C_3s and C_4 levels became normal.

The acute onset of polyarthritis, fever, and urticaria along with hypocomplementaemia and HBs antigenemia is typical of HBsAg arthritis. Besides urticaria, erythema marginatum and papular, macular, and petechial lesions have been described.[2] Our patient is of interest in that three skin lesions developed. Scaly macular lesions over the involved joints of the hands, mimicking the lesions of dermatomyositis, and giant urticaria limited to the upper extremities were both present in the pre-icteric phase, and when jaundice appeared petechiae developed.

The arthritis of HBsAg hepatitis is thought to be due to the deposit of HBs Ag-anti-HBsAg complexes in the synovium.[3] The urticaria, which is an integral part of this syndrome, could also be due to such deposition in the dermis, with subsequent liberation of histamine. The induction of urticaria (or merely itching as in our case) by application of a tourniquet might be helpful as an early diagnostic sign in arthritis of hepatitis. We recommend that this simple test be performed in all cases of arthritis of hepatitis to appreciate its reproducibility and significance.—We are, etc.,

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1 Fernandez, R., and McCarty, D. J., *Annals of Internal Medicine*, 1971. **74**, 229.
2 Alpert, E., Isselbacher, K. J., and Schur, P. H., *New England Journal of Medicine*, 1971, **285**, 185.
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SI Units

SIR,—Dr. G. H. Hall (31 May, p. 502) complains that the opinion of clinicians has not been sought before a directive to adopt SI units in the N.H.S. was issued. Quite understandably he accuses "many pathologists" of wishing to introduce this change. He, and doubtless many others, may be interested to know that neither the main body of pathologists, nor biochemists, have been given chance to vote upon the topic by any properly conducted democratic means. Doubtless everybody thinks that because other specialties and other hospitals are changing to the new units, then they too must conform. It is the lack of an opportunity for us to express a collective opinion that allows us to be conquered.

I carried out a poll of opinion within this hospital department. Each member of the staff was asked to give his own personal opinion on the virtue of changing over to SI units. The result was as follows: in favour, three; against, four; "don't know," three; "the present proposals are unsatisfactory and should be reconsidered," 13. Most individuals felt that they would have liked an opportunity to express an opinion and were dissatisfied by the manner in which the directive to change had been given. Those who did not know had not studied the proposals.

There is a political decision to introduce metric units throughout the U.K., but how this should be interpreted in terms of units as applied to laboratory medicine was studied by a working party. Perhaps the current disagreement is not with the policy of metrication as such but rather with the interpretation of this adaptation. The

greatest cause of disagreement is over the use of units based on the mole when applied to substances of higher molecular weight. This seems to be a quite unnecessary complication and change to no great purpose and may well prove to be a cause of error and misunderstanding. It would be infinitely preferable to use mass concentration units in the same way as is recommended for proteins.—I am, etc.,

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Little and Big Bellyachers

SIR,—Many little bellyachers do grow up to become big bellyachers (leading article, 31 May, p. 459) because the cause of the bellyache is not treated.

All the bellyaches presenting initially as recurrent abdominal pain or irritable colon in children and later as dyspepsia associated with hiatus hernia, peptic ulcer, and/or gall bladder disease, and/or diverticulitis or colitis, and/or maturity-onset diabetes in adults are different syndromes of fat intolerance, a disorder of metabolism caused by excess dietary fat intake.[1] Fat intolerance is the consequence of family patterns of feeding; therefore it is not unusual to find that the mother and father and other relatives of the little bellyacher are big bellyachers. The disorder is not inherited; it is due to learnt feeding habits within the family and is a self-inflicted disease.

Sympathy, psychiatric treatment, or drug therapy is not a cure for the metabolic disturbance resulting from fat intolerance. The symptoms of the different syndromes and the metabolic abnormality can be cured by adhering strictly to the "fat free diet" as defined in my paper.[1] If this is done in the little bellyacher, he or she will not become a big bellyacher. There is now a series of 100 children and young adolescents in the Plymouth Clinical Area, symptom-free and developing normally and healthily on this diet. Many of these patients had failed to respond to the usual treatment for emotional disturbance or for irritable colon, or to appendicectomy. Many of my 2000 adult patients who are now cured of their big bellyache by strict adherence to the "fat free diet" started as little bellyachers. They continued to be bellyachers, off and on, all their lives until they adhered to the "fat free diet."

I support the statement in the leading article that there is a need for research and therapeutic trials. Big and little bellyaches are cured and can be prevented by reversion to a natural feeding habit. It is time that our profession looked dispassionately at the evidence and established the truth by means of appropriate research.—I am, etc.,

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1 Childs, P., *British Journal of Surgery*, 1972, **59**, 787.

Management of Depression

SIR,—In his excellent paper on the management of depression (17 May, p. 372) Dr. G. W. Ashcroft suggests that "adverse environmental circumstances" may prevent an adequate response to antidepressants or electric convulsion therapy. It is also important to recognize those physical illnesses such as hypothyroidism and vitamin B_{12} deficiency which may present as a resistant depression. The diagnosis is easily missed when psychiatric symptoms predominate and complaints such as weakness, anorexia, and constipation are misinterpreted. With specific treatment the improvement in the mental state is often dramatic.—I am, etc.,

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Screening Methods for Covert Bacteriuria in Schoolgirls

SIR,—May I comment on the interesting paper by Dr. Bridget Edwards and her colleagues (31 May, p. 463). I agree entirely with their observation that the MacConkey surface of the Oxoid dip slide suppresses the growth of the commensal organisms which reveal contamination, whereas these organisms grow well on the CLED surface. Use of the MacConkey surface, therefore, results in a number of false positive interpretations, as they describe. It is for this reason that, for the past three years, we have been using an Oxoid dip-slide coated on one side only with CLED agar; this medium supports growth of the known urinary pathogens, including micrococcus 3, which, though commoner in young women, does occasionally cause infection in children, both boys and girls. Contamination is indicated by a mixed growth which includes the vaginal and perineal commensals.

Originally we used the single-medium dip-slide for immersion in a midstream specimen[1]; more recently we have been using the dip-stream method for follow-up of boys who have had urinary infection.[2] The dip-stream method, especially in the patient's home, is likely to be more reliable if inoculation of only one medium is required. At present the manufacturers supply these slides at the same price as those coated with two media; a more widespread demand for a single-medium slide, however, might reasonably be expected to reduce still further the cost of the dip-stream method.—I am, etc.,

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1 Maskell, R., *Journal of Clinical Pathology*, 1973, **26**, 181.
2 Hallett, R. J., Pead, L., and Maskell, R., *Lancet*, 1974, **2**, 104.

Pay Beds and Professional Freedom

SIR,—I write in full and firm support of Mr. D. E. Bolt (7 June, p. 556). He lucidly expresses a point of view which is held by a large and growing number of hospital doctors, including many junior doctors. In the interests of the public and of the N.H.S., no less than those of the medical profession itself, it is vitally important to preserve pay beds and other facilities for private medical care in our hospitals. I have no doubt that consultants would be right in resorting to "work to contract" wherever pay beds are unjustly threatened or closed. Moreover, it would be wise for us to use every means in our power to resist any vindictive legislation which was pressed upon Parliament.

Mr. Bolt expresses the fear that his views are "without significant support among the leadership of the profession." Whether he is referring to the B.M.A. negotiators or the Hospital Consultants and Specialists Association I do not know, but the profession should need no reminding that, at its meeting on 15 October 1974, the Joint Consultants Committee (representing all the royal colleges and faculties, the B.M.A., the British Dental Association, and the junior doctors) clearly pledged its full support for the preservation of private facilities both within and outside the N.H.S. (26 October 1974, p. 241).

Writing as a member of both the B.M.A. and the H.C.S.A. may I emphasize how vital it is that the two organizations should work closely together? Internecine strife can only seriously impair the potency and efficiency of the profession's negotiators in their present confrontation with a particularly doctrinaire and intransigent Secretary of State. The enabling Act of 1946 always carried the seeds of total State monopoly in medicine; it is a pity that it has taken more than a quarter of a century for a majority of hospital doctors to recognize its dangerous implications for professional freedom.—I am, etc.,

REGINALD S. MURLEY

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Junior Hospital Staff Contract

SIR,—I am amazed at the vast number of letters in recent weeks which express reservations about the proposed 40-hour contract for junior hospital staff. Are their authors not those who, until two months ago, were giving at least vocal support to officers of the Hospital Junior Staffs Group Council? Who are in fact happy to allow their conditions of work to go unchanged, for the better anyhow? Who were least likely to take part in any form of sanction to improve their lot? Who in fact cannot see past the ends of their noses and visualize the Health Service in five years' time if things continue along their present course?

The 40-hour week, as I see it, is not just a method of increasing remuneration, and it certainly is not intended to reduce the quality or quantity of care given to the patient. It is to improve our ability to function as doctors practising medicine and therefore to use the medical manpower resources that are available as efficiently as possible.

Junior and senior medical staff will always complain while their patient:non-patient time ratio is low as long as it is not recognized that they work and will continue to work long hours. Freedom of the practitioner is an attitude of mind and not a number of hours on a piece of paper. The doctor who does stay on after hours does not stop doing this whether he has already worked 100 hours or 40 hours. Is the argument of some that we need to be told when to work?

I will give full support to efforts that will make our jobs more clinical. Hopefully the more time we are thus able to spend with the patient, then the more he will appreciate what we are trying to do.

For goodness sake let the administrators