tobacco has been heated to "cure" it, immediately inactivating enzymes and thereby leaving more unchanged hydrocarbon, while pipe tobacco and Spanish and Polish cigarette tobaccos are treated by the old method.

Our experiments showed that pipe tobacco made loosely into a cigarette[4] burned at a lower temperature than cigarette tobacco packed loosely into a cigarette. We also showed[5] significant lung cancer production in Swiss white mice compared with controls when smoke from manufactured cigarettes was used. We showed no cancer production in Swiss white mice when loosely packed pipe tobacco was used in the cigarette and, more significantly, no deaths at all in the experimental period (equivalent to nine years' smoking in man); also negligible bronchitis and no cardiac failure. Fifty-five per cent of the mice succumbed to cardio-respiratory illness when commercial cigarettes burning at high temperatures were used for the same time.

We also noted that draw-back was associated with less lung cancer in man than simple puffing[6] and pointed out a logical reason for this; simple puffing fills to the carina, while draw-back dilutes the advancing gas cloud quickly from the carina into the less susceptible alveoli. The nicotine content of the reduced-temperature-burning cigarette is similar to the nicotine content of the high-temperature commercial cigarette, so that smoking satisfaction on this level should be similar. Heterocyclic hydrocarbons producing satisfactory flavour may be reduced in the cigarettes with a lower temperature of combustion, but the smokers who smoked some 200 of these test cigarettes made the comment that the flavour was acceptable to a smoker.—I am, etc.,

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G.M.C. and Indian Doctors

SIR,—Dr. S. K. Roy (7 June, p. 562) appears to be very indignant about the new G.M.C. legislation whose aim is to prevent totally unsuitable doctors coming to this country from overseas and finding it difficult to obtain appointments. I find this indignation surprising as the new legislation can be nothing but advantageous to those overseas doctors, of whom Dr. Roy is obviously one, who have had an excellent training and are now well settled in this country.

Unfortunately the public, including, I fear, many of our own profession, do not seem to realize the vast difference that exists between the type of medical education that is given in the great centres such as Calcutta and Bombay and that which is given in the countless small medical schools which have neither the facilities nor the teachers to give a proper training. The whole economic situation in certain countries makes it quite impossible for a universal standard to be maintained and many doctors have in the past been reaching this country with pre-graduate knowledge that is so scanty that all the postgraduate training in the world will never make them competent.

By preventing doctors of this sort coming over here a different perspective towards the overseas doctor will be built up and the present feeling against them will surely disappear entirely.—I am, etc.,

ROBIN BURKITT

Ashford Hospital,
Ashford, Middx

Immigrant Doctors

SIR,—As the wife of a Commonwealth immigrant doctor I would like to make a few observations in connexion with the recent militancy and bad manners shown by immigrant doctors towards the B.M.A. by threatening mass resignations on the grounds that the B.M.A. has not represented their case adequately to the E.E.C. Commission.

I feel that the B.M.A. has done a lot for the cause of immigrant doctors. I quote one of your leading articles (4 November 1972, p. 249) which adequately clarified the attitude by the B.M.A.: "Racial discrimination, whether under the cloak of political, religious, or biological doctrines, is peculiarly repugnant to doctors . . . because it flatly contradicts the tradition and guiding ethic of their profession, by which they treat all men alike irrespective of their colour, creed, or nationality."

Asian doctors should be grateful to their host country, which has given them an opportunity of advancing their medical knowledge and acquiring decent careers which probably they would not be able to obtain in their own countries of origin.

I, for one, and my husband are not prepared to show ingratitude.—I am, etc.,

F. ALI

Basingstoke

Alternatives to the Fluoridation of Water

SIR,—Will Professor D. Jackson (5 April, p. 35) be good enough to tell us what agent is responsible for the "very infrequent" attacks of dental caries "beyond the age of 40" if, as he also claims, "the benefits of fluoridation of drinking water tend to fade with increasing age"?—I am, etc.,

WINIFRED M. SYKES
Hon. Treasurer,
National Pure Water Association

Bardwell, near Bury St. Edmunds,
Suffolk

*** We showed Mrs. Sykes's letter to Professor Jackson, whose reply is printed below.—ED., *B.M.J.*

SIR,—A brief description of the age-dependence of dental caries may help to answer the questions, both explicit and implicit, that are contained in Mrs. Sykes's letter.

In English non-fluoride communities the age-specific attack rate of caries in permanent teeth reaches its maximum between 12 and 15 years of age.[1] The attack rate then declines with advancing age, and beyond the age of 40 years relatively few attacks occur.[1] In Hartlepool, where the natural concentration of fluoride in drinking water is 1·5 to 2·0 p.p.m., the age-prevalence of dental caries is markedly lower than that in non-fluoride communities at all ages. However, the ratio of the age-prevalence of caries (Hartlepool: non-fluoride communities) is at its lowest in young people and it gradually rises to a plateau, or limiting value, of approximately 0·6 at around 40 years of age.[1] Thus, though substantial benefits from fluoridation are seen at all ages, they are most conspicuous in young people below the age of 15.

In my view the factors responsible for the infrequent attacks of dental caries beyond the age of 40 do not differ in *kind* from those that operate at lower ages. However, their *rate* diminishes with increasing age in adults. My collaborators and I have described in detail elsewhere[1-5] our interpretation of the somewhat complicated problem of the aetiology and pathogenesis of dental caries.—I am, etc.,

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Inhibition of Ristocetin-induced Platelet Aggregation by Haemaccel

SIR,—Most patients with von Willebrand's disease have a deficiency of the plasma co-factor required for ristocetin-induced platelet aggregation, and diagnostic tests using this antibiotic are rapidly gaining acceptance. We wish to report that the gelatin plasma expander Haemaccel (Behringwerke) inhibits ristocetin-induced platelet aggregation at concentrations that are achieved therapeutically. Thus an erroneous diagnosis of von Willebrand's disease could be made when bleeding patients have been treated with this agent.

We first discovered this effect in patients undergoing open heart surgery in the Academical Hospital Rotterdam-Dijkzigt. Ristocetin-induced platelet aggregation (platelet-rich plasma, ristocetin 1·5 mg/ml) was completely abolished or strongly diminished after the start of cardiopulmonary bypass. The cause was shown to reside in the plasma of the patient, since addition of platelet-poor plasma of the patient to normal platelet-rich plasma (1:1) abolished or diminished ristocetin-induced platelet aggregation. It is the normal procedure in this hospital to prime the heart-lung machine with a mixture of Haemaccel and fresh heparinized blood. Addition of Haemaccel to normal platelet-rich plasma (1:4 v/v), comparable to the intravenous administration of 500 ml of the plasma expander, abolished or strongly inhibited ristocetin-induced platelet aggregation.

Other experiments indicate that Haemaccel inhibits ristocetin-induced platelet aggrega-

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.,

J. STIBBE

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Philadelphia

E. P. KIRBY

Erasmus University,
Rotterdam

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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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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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