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 cigarette⁴ burned at a lower temperature than cigarette tobacco packed loosely into a cigarette. We also showed⁵ 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 puffing⁶ 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

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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

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** 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 elsewhere¹⁻⁵ 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 Haemacel

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 Haemacel (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 Academic 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 Haemacel and fresh heparinized blood. Addition of Haemacel 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 Haemacel inhibits ristocetin-induced platelet aggrega-