acting unfavourably with sympathomimetic amines or raising the blood pressure.—I am, etc.,

G. G. WALLIS

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Ilkley, Yorks

Classics and the Medical Student

SIR,—No doubt Dr. I. A. Olson and his colleagues (3 February, p. 282) expected to receive some interesting comments from the older generation in the profession on their categorical statement that "a qualification in Latin confers no advantage at all on the aspiring doctor." Perhaps it may be of interest for two young house surgeons to take issue with some of their conclusions.

Firstly, we feel many of the findings may have little general relevance since the first-year intake to a new medical school will not be comparable to that of an established institution. In particular, however, we consider that it would be very misleading to reject a knowledge of the classics, including Greek, by evaluating the use of Latin alone in understanding medical terminology. For in fact many medical terms have a Greek derivation, as do most words in the scientific vocabulary. (Note the origin of most of the words in level 6 of the appendix to Dr. Olson's paper. Latin was of course at one time the method of communication among doctors, aiding professional secrecy and keeping dangerous remedies out of the hands of the uninitiated. This is quite different from the value of Latin to the aspiring doctor today.

Since we have found that a knowledge of Latin and Greek has increased our interest and enthusiasm for many aspects of our medical course, we feel it is unjustifiable to discourage the interested school pupil from studying these subjects before taking a medical training.—We are, etc.,

R. HUGH MACDOUGALL
D. W. SINCLAIR

Royal Infirmary,
Dundee

Legalization of Cannabis

SIR,—It is stated in the press that the National Council for Civil Liberties is to attempt to get the use of cannabis made legal. Surely this should be strongly resisted. Firstly, although it is often denied, there seems no doubt that cannabis is addictive since why otherwise would those who smoke it heavily go all the way to Nepal, where it is freely available? Secondly, research, particularly in America, shows that it causes insidious damage to the brain with consequent deterioration of the personality; the addict loses all ambition and interest in hygiene and appearance. Thirdly, anyone who has treated addicts knows that a large number of young people try cannabis before proceeding on to heroin or lysergic acid.

When we look at the consequences of smoking tobacco and the fact that we have between half a million alcoholics in the country surely we should realize that we do not need any more legal drugs.

CLIFFORD ALLEN

Mold, Flintshire

Disseminated Intravascular Coagulation in Benign Tertian Malaria

SIR,—Disseminated intravascular coagulation has been demonstrated in severe cases of falciparum malaria^{1,2} but does not seem to be the usual cause of the thrombocytopenia observed in malaria of all types.³ In the case reported here, on the other hand, thrombocytopenia in a patient with *Plasmodium vivax* malaria was accompanied by evidence of disseminated intravascular coagulation.

The patient was a previously healthy 22-year-old Caucasian man who presented with episodic high fever, rigors, and slight splenomegaly. Blood films confirmed *P. vivax* malaria. Haemoglobin was 12.4 g/100 ml and platelet count 90,000/ μ l. Thrombin time was 13 sec (control 9 sec), cephalin-kaolin time 81 sec (control 38 sec), prothrombin time 17 sec (control 15 sec), and fibrinogen titre 1/128. Fibrin-fibrinogen degradation products were detected in a titre of 1/512 (equivalent to 640 μ g/ml of fibrinogen) on tanned red cell haemagglutination inhibition immunoassay. The coagulation abnormalities were present at the time of presentation and there was no evidence of deep vein thrombosis or other lesion to account for them. There was no evidence of intravascular haemolysis. No bleeding tendency was manifest clinically and only routine antimalarial treatment was given. The coagulation abnormality persisted for the first five days of the illness, then reverted to normal. The patient had also a low serum level of IgA (less than 10 mg/100 ml) and an elevation of IgG (2,500 mg/100 ml) and IgM (485 mg/100 ml).

It appears that although disseminated intravascular coagulation is not the usual cause of thrombocytopenia in malaria, nevertheless it may occur, though uncommonly, in benign tertian malaria as well as in falciparum malaria. The mechanism remains to be determined.—I am, etc.,

BARBARA BAIN

St. Mary's Hospital,
London W.2

- 1 Reid, H. A., and Nkrumah, F. K., *Lancet*, 1972, **1**, 218.
- 2 Dennis, L. H., Eichelberger, J. W., Inman, M. M., and Conrad, M. E., *Blood*, 1967, **29**, 713.
- 3 Beale, P. J., Cormack, J. D., and Oldrey, T. B. N., *British Medical Journal*, 1972, **1**, 345.

Airgun Pellets and the Eye

SIR,—I would like to lend support to Mr. D. I. Bowen and Miss Denise M. Magauran's important article concerning ocular injuries caused by airgun pellets (10 February, p. 333). There is no doubt that these injuries are increasing in number, and in a recent series from Birmingham,¹ despite the relative frequency of industrial injuries there and the relative rarity with which a pellet actually penetrates the globe, it was found that the airgun pellet is now the chief single cause for excision of an eye after intra-ocular foreign body.

There should indeed be legislation to raise the age limit for possession of an air weapon, and also stricter penalties for indiscriminate firing.—I am, etc.,

S. P. B. PERCIVAL

Scarborough

- 1 Percival, S. P. B., *British Journal of Ophthalmology*, 1972, **56**, 454.

SIR,—Mr. D. I. Bowen and Miss Denise M. Magauran (10 February, p. 333) are not quite right in stating that "no large series

of airgun pellet injuries of the eye has appeared in English." Though not so large as theirs, a series of 39 injuries due to airgun pellets out of a total of 614 ocular injuries in patients under 16 admitted to the Wolverhampton Eye Infirmary for two days or longer during a 10-year period was reported by Lambah in 1962.¹ Just over one-third of these airgun injuries resulted in blind eyes. It was also noted that over 90% of such injuries occurred in the 11-15-year age-group.

It is interesting to note that in the Liverpool area there has been only a small drop in incidence in the second decade of the period studied by Dr. Bowen and Miss Magauran. In my view, such important facts cannot be reported too often.—I am, etc.,

B. R. KESBY

Wolverhampton and Midland Counties Eye
Infirmary,
Wolverhampton

- 1 Lambah, P., *Lancet*, 1962, **2**, 1351.

Radioimmunoassay Follow-up of Hydatidiform Mole

SIR,—I found it difficult to understand from D. K. D. Bagshawe's letter (17 February, p. 414) just what treatment he, as an expert in the subject, advises for trophoblastic disease. His assertion that hysterectomy "has nothing to offer in terms of saving life" is a sweeping statement if its is based on only 16 patients who later "required treatment" in his extensive experience of 280 with hydatidiform mole who were followed up by regular radioimmunoassay. The reasons why they required treatment will doubtless be published later. Either a recurrent mole or carcinoma could be revealed by follow-up radioimmunoassay and indicate the need for treatment, but if confined to the uterus both would have been prevented by primary hysterectomy. But it is misleading to suggest that I advocated this as a routine treatment. A scientifically designed prospective study aims at collecting data from which to answer hitherto unanswered questions, which in this case would include an evaluation of the "preventive" value of hysterectomy. It was for that reason I proposed it.

Dr. Bagshawe agrees "that hysterectomy has a valuable place in the management of some of these patients," but does not define which ones they are. A prospective study might find the answer.—I am, etc.,

JOHN STALLWORTHY

John Radcliffe Hospital,
Oxford

Pathogenesis of Obesity

SIR,—Dr. J. Anderson (26 February, 1972, p. 560) discussing the pathogenesis of obesity, claims that the energy imbalance between intake and output is the initial cause of this disease. We are sorry to differ from him and wish to report our opinion on this question.

We believe that, in contrast with pancreatic diabetes mellitus, which is caused by hypo-sensitivity of the β -cells to a glucose load, obesity must be regarded as a disease of β -cell hypersensitivity. In the early stage of obesity we have demonstrated postprandial hyperinsulinaemia leading to latent hypo-

glycaemia.¹ This insulin-induced hypoglycaemia activates the corticotrophin releasing factor—corticotrophin—glucocorticoid axis. This permanent stimulation is afterwards responsible for the steady hypercorticism in obesity.

Glucocorticoids as well as other anti-insulin factors, involve tissue insulin resistance and hyperglycaemia which, by stimulating the β -cells result in their hyperplasia. Thus the chain of pathogenesis of obesity may be shown as follows: β -cells hypersensitivity to carbohydrates—postprandial hyperinsulinism—postprandial hypoglycaemia—hypercorticism—tissue resistance to insulin and hyperglycaemia— β -cell hyperplasia. The energy imbalance with predominant intake is a result of intensive lipogenesis resulting from hyperinsulinism.

Excessive insulin response to glucose load in obese subjects is well known. But for the successful prevention of obesity it is necessary to separate the "pre-obese" subjects. This group consists of the patients with normal weight and excessive insulin response to glucose load.—We are, etc.,

BORIS DRAZNIN
YURITH MARAKHOVSKII

Endocrinological Clinic,
Minsk, U.S.S.R.

¹ Draznin, B. N., and Marakhovskii, Yu. Kh., *Voprosy Endocrinologii (Minsk)*, 1973, 5. In press.

Cutaneous Polyarteritis

SIR,—Your leading article (6 January, p. 6) cites Borrie¹ as making a good case for regarding the cutaneous form of polyarteritis nodosa as a distinct clinical entity with a good prognosis, and you report his finding that a large majority of patients with livedo reticularis and nodules and a history of polyarteritis had no evidence of lung or kidney disease, though lesions in muscles and nerves with fever and a raised E.S.R. were rather common. Nevertheless, we saw a patient in whom the cutaneous manifestations of the disease were a mask behind which a multisystemic arteritis insidiously developed over a period of 30 months.

The patient, a man aged 41, was admitted on 17 November 1971 acutely ill with cachexia, fever, extensive muscle wasting in all limbs, sinus tachycardia, and a blood pressure of 200/120 mm Hg. He gave a history of purple macular and nodular lesions first developing in February 1969, when they were confined to the hands and feet, spreading later to the trunk and limbs. After 10 months blueish-purple eruptions with painful ulceration appeared from time to time. From February 1971 he began progressively to lose weight, and in August continuous fever developed. He had been admitted to various hospitals at various times and treated intermittently with small doses of prednisone for Schönlein's purpura. From the start of his illness in 1969 until the end of September 1971 his blood pressure, analysis of urine, E.S.R., and total blood cell count had always been normal. From October 1971 his condition deteriorated rapidly, with loss of appetite, weakness, epigastric pain, and paresis and hypoaesthesia of the legs.

When admitted to our hospital he presented the diagnostic picture of polyarteritis nodosa. There were signs of severe asymmetrical peripheral neuropathy. On the sixth day he developed irreversible haemorrhagic shock and he died. An enormous right perirenal haematoma was found at necropsy. The source of the bleeding could not be discovered. Macroscopically the kidneys were normal. The histology of the renal vessels and of the skin nodules were typical of polyarteritis nodosa and there were extensive throm-

botic and necrotic changes in the renal interlobar and arcuate arteries.

Motor and sensory neurological disorders are essential to the early diagnosis of polyarteritis nodosa.^{3,4} It was only after close questioning that we elicited from our patient that at the start of his illness he had had some joint pains, and in August 1969, six months after his cutaneous eruption first began, he had noticed weakness in his legs, with numbness, tingling, burning, and areas of paraesthesia, and also transitory pain and stiffness of the legs and hands. These were so slight that they were overlooked by both the patient and the doctors who treated him. Clearly any signs of peripheral neuropathy must be carefully looked for in cases of polyarteritis nodosa seemingly limited to one organ or system.—I am, etc.,

TADEUSZ DYK

Municipal Hospital,
Gdynia, Poland

¹ Borrie, P., *British Journal of Dermatology*, 1972, 87, 87.

² Dyk, T., and Piotrowski, M., *Polski Tygodnik Lekarski*, 1972, 27, 1279.

³ Richardson, J., *Connective Tissue Disorders*. Oxford, Blackwell, 1963.

⁴ Dyk, T., in *Internal Diseases*, ed. E. Szczeklik. Warsaw, Panstwowy Zaklad Wydawnictw Lekarskich, 1968.

Fluoride and Osteoporosis

SIR,—In our opinion, the findings of J. Jowsey and her colleagues¹ in treating patients with osteoporosis with fluoride, calcium, and vitamin D, referred to in your leading article (30 December, p. 748), are open to question.

A critical analysis of their paper shows that their 11 patients had histological evidence before treatment of osteomalacia and not of osteoporosis, since their mean osteoid border was significantly wider ($16.0 \mu\text{m} \pm 0.6$) than that of the aged-matched controls ($12.3 \mu\text{m} \pm 0.6$). Furthermore, their estimate of the correct dosage of fluorides (50 mg) is very arbitrary, as may be deduced from their statement that "the administration of less than 45 mg of sodium fluoride daily did not consistently increase bone formation, whereas the administration of 60 mg or more daily produced abnormal bone." If the dose range is so narrow monitoring is obligatory, especially since the patients who might benefit from treatment are of an age at which kidney function is reduced. Maes and colleagues² and Largent³ have shown that chronic renal sclerosis reduces fluoride elimination.

According to Jowsey and her colleagues¹ a 5% increase in bone formation may be expected with a daily dose of 50 mg of sodium fluoride. Since the errors of measurement in quantitative microradiography are about 10% in the same section and 20% in two different sections,⁴ this 5% increase in bone formation found with a dose of 50 mg fluoride falls within the errors of the technique. It would therefore be wrong at this moment to advocate large-scale uncontrolled trials of treatment of osteoporosis with fluoride, calcium, and vitamin D on the basis of the observations of Jowsey and her colleagues.—We are, etc.,

J. DEQUEKER
A. BURSENS

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Catholic University of Louvain,
Belgium

- ¹ Jowsey, J., Riggs, B. L., Kelly, P. J., and Hoffman, D. L., *American Journal of Medicine*, 1972, 53, 43.
- ² Maes, J., Dufaux, J., and Vandenbroucke, J., *Acta Clinica Belgica*, 1960, 15, 65.
- ³ Largent, E. J., *Fluorosis*. Columbus, Ohio State University Press, 1961.
- ⁴ Jowsey, J., et al., *Journal of Bone and Joint Surgery*, 1965, 47-A, 785.

Drugs in Infertility

SIR,—Dr. E. W. Barnes and others (27 January, p. 234) consider that their experience with the use of mesterolone in the treatment of eunuchoidal males is at variance with that which I mentioned briefly in my letter (18 November, p. 425). But is there all that much difference? I stated that of five eunuchoids treated with daily doses of 200 mg three failed to show an androgenic response while the other two responded satisfactorily, though for these two the corresponding daily maintenance doses of fluoxymesterone were 10 and 20 mg respectively. I concluded that mesterolone is a weak androgen and I said that, at maintenance dose levels, "suppression of endogenous testosterone production does occur." Perhaps I ought to modify the latter to read "depression of plasma testosterone levels can occur"—which may or may not be the same thing. I made no comment on the effects of mesterolone on urinary luteinizing hormone and follicle-stimulating hormone excretion.

Dr. Barnes and his colleagues observed "good clinical responses" in all four previously untreated eunuchoids given 200 mg of mesterolone daily—though I would not have regarded an "increase in shaving frequency from once to three times per week" as being entirely adequate after up to two years' treatment. A 30% drop in testosterone levels was noted in some of the patients. In a second group of eight patients previously on testosterone therapy, three found the 200-mg dose of mesterolone to be inadequate for maintenance.

I venture to suggest that there is little discrepancy between the Edinburgh findings and my own and that they confirm, rather than refute, the general statement I had made. Mesterolone would seem to have about one-tenth to one-twentieth the potency of fluoxymesterone when used for maintenance therapy in hypogonadal males; does not this justify the description of "weak androgen"?—I am, etc.,

G. I. M. SWYER

University College Hospital Medical School,
London W.C.1

Potential of Neuroleptics by Catecholamine Inhibitors

SIR,—There is much experimental evidence to support the view that neuroleptics of the phenothiazine or butyrophenone type block central catecholamine receptors.^{1,2} The synthesis and turnover of catecholamines are thereby increased, presumably owing to a receptor-mediated negative feedback mechanism, and the increased turnover seems to result in an increased sensitivity to inhibitors of catecholamine synthesis. Thus in animal experiments in our laboratory Ahlenius and Engel³ found that an inhibitor of tyrosine hydroxylase (the methyl ester HCl of DL- α -methyltyrosine) greatly potentiated the depressant action of haloperidol. In a pilot