anaesthesia and with liberal replacement of blood.—We are, etc.,

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Death during Dental Anaesthesia

SIR,—With reference to your medicolegal report (2 February, p. 207) entitled "Death during Dental Anaesthesia" I wish to make the following comments.

In evidence Mr. Alan Williams said regarding intravenous methohexitone, that "he had been instructed in its use at a course run by the Society for the Advancement of Anaesthesia in Dentistry." From this it might be inferred that the S.A.A.D. claimed to turn out trained anaesthetists after three days' tuition. This is far from the case, and I quote from my own lecture on the course: "If you expect to go away after three days, fully trained and fit to launch yourselves on your unsuspecting patients, think again please. In this short time, we can only give you the rudiments of the subject for you to build on by further study. We lay great stress on the complications that can occur, and tell you how to treat them, and more importantly, we try to show how to conduct your practice so that you prevent complications occurring. We lay down strict basic rules of safe anaesthetic practice and insist that these are followed completely in every case."

Regarding Mr. Williams's "six breaths of nitrous oxide"; during the course, in lectures and demonstrations, he was repeatedly warned of the dangers of anoxia. This point receives frequent mention in the *S.A.A.D. Manual*, 3rd edition, which he was advised to study. The following extracts are appropriate to the case. P. 2, para. 1.—"The rules of safety are clearly defined. Until they can be adhered to, the method must not be attempted." P. 9, Recommendations, no. 2—"Oxygen restriction below 20% is never needed and should never be tolerated. Any technique . . . in which there may be a temptation to restrict oxygen . . . should be carefully avoided." P. 16, Criteria, para. 2—"Oxygen restriction neither follows the administration of methohexitone nor is it necessary in any of the techniques employing it." P. 31, para. 3—"Many complications can occur from abuse of the method. At all times the fundamental principles of safety must be strictly followed." P. 38, Care and Control, para. 4—"The dangers of anoxia must be avoided by *never reducing the oxygen below 20%*. P. 40, Causes of Anoxia, no. 2—"Oxygen restriction during inhalation anaesthesia." P. 39, Causes of Hypotension, (b)—"Anoxia."—I am, etc.,

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Vaginal Cytology

SIR,—In reply to Mr. S. A. Way's letter (2 February, p. 201) it should be made clear that the Department of Health and Social Security has at no time stated that cervical cytological examination is unnecessary below

the age of 35. On the other hand, as the Registrar General's figures show, it is in those women aged 45 and over that the incidence of invasive cervical carcinoma is highest, and in situ carcinoma tends to precede the invasive lesion by 10 years.

The most urgent matter therefore has been to aim at comprehensive screening of women aged 35 and over, and to encourage this a fee for service is paid to general practitioners screening such women. This priority group has recently been extended to include selected higher-risk women below the age of 35. That general practitioners and others realize that younger women may be at risk is clear from the national figures, which show that about one-half of the two million smears taken annually in England and Wales come from women *below* the age of 35.—I am, etc.,

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Gamekeeper's Thumb on the Ski Slopes

SIR,—With reference to your leading article on this subject (9 February, p. 213), in addition to the mechanism described this injury appears to be more common on artificial training ski slopes, where the run is made of nylon mesh. In the event of a fall the thumb gets caught in the nylon mesh, and with the momentum of the body forced abduction ruptures the ulnar collateral ligament of the metacarpophalangeal joint of the thumb.—I am, etc.,

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British Academy of Psychopharmacology

SIR,—For many years the United Kingdom has lagged behind other countries in having no specific organization dedicated to psychopharmacology. Thus a number of other countries have established their own national organizations that are affiliated to the Collegium Internationale Neuro-Psychopharmacologicum (C.I.N.P.), one of the best known of these being the American College of Neuro-Psychopharmacology.

In view of the considerable contributions made by our country to psychopharmacology it appears to us that the present situation is anomalous and that the time has come to establish a national organization in Britain. To avoid confusion with the royal colleges we propose that this organization be called "The British Academy of Psychopharmacology."

We believe that there is a particular need to focus attention on the importance of drug treatment in psychiatry in the eyes of both the young clinician and the research scientist. Therefore the objects of the academy will be to further research in psychopharmacology, both clinical and experimental, and to improve the quality and standards of psychotropic drug evaluation. This can be accomplished by means of the spoken and written word through the media of meetings, study groups, and publications.

We propose to call a meeting in the near future of all those members of the profession who may have an active interest in psychopharmacology for the purpose of determining the constitution of the academy, to elect officers, and to consider the academy's policy, particularly in relation to the pharmaceutical industry. To this end we have drafted some suggestions and would be pleased to forward these to any interested member of the profession. We would also like to receive suggestions and comments concerning the establishment of the academy, and invite those interested to communicate with any one of the undersigned.—We are, etc.,

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MAX HAMILTON
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Home Graduates Only

SIR,—I heartily agree with Dr. T. K. Ghosh (16 February, p. 290).

I am a British subject now, but I am an Indian. I came to England in 1956 and have enjoyed every moment of my stay in this wonderful country. I was well aware of various attitudes in my medical colleagues but found that all these attitudes stemmed from a lack of communication by both parties concerned. When one appreciates British humour and their in-built sense of justice one soon realizes that the English as taught to us is vastly different from regional spoken English. When one masters this, one is no longer an outsider, no more so than a British missionary in, say, India can master the local vernacular and the local humour and still be an outsider. We are all colleagues, and if we would only "specialize" in the local English at the "local" (if you will pardon the pun), would not things be easier?—I am, etc.,

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Vitamin A, Pregnancy, and Oral Contraceptives

SIR,—Mrs. Jennifer Wild and her colleagues (12 January, p. 57) sought to show that oral contraceptives increase serum vitamin A levels and investigated the hypothesis that there is an increased teratogenic risk for mothers who become pregnant at such a time. However, their conclusions were

drawn from a series of measurements by a relatively non-specific technique.

Serum vitamin A has been measured by a variety of colorimetric methods (for example, those of Bessey *et al.*¹ and of Neeld and Pearson²) which consistently give a mean adult concentration of about 32 µg/100 ml (range 21–47 µg/100 ml).³ All are inaccurate when serum carotenoid levels are high. More recently, fluorimetric methods have been employed in an attempt to overcome this problem. Hansen and Warwick⁴ used a modification of Kahan's⁵ extraction procedure to obtain a mean adult serum vitamin A level of 133 µg/100 ml. After Thompson *et al.*⁶ had reported that phytofluene interferes with the fluorimetric estimation of serum vitamin A Garry *et al.*⁷ devised a method for separating the two compounds before measuring the fluorescence of the plasma extract. With this technique they reported that the mean serum vitamin A is reduced to a value of 34 µg/100 ml, and my colleagues and I, at the Institute of Child Health, Birmingham, have obtained values of the same order of magnitude (mean ± S.D. = 39 ± 15 µg/100 ml).⁸

Recently a method was developed for measuring levels of both vitamin A and phytofluene in the same 200-µl serum sample⁹ and these compounds were measured in serum from healthy individuals. No correlation was detected between the two sets of values. Phytofluene was present in all samples at a substantial though variable level.

The point I wish to make is that Mrs. Wild and her colleagues have used the method of Hansen and Warwick,⁴ which is inaccurate, for estimating vitamin A levels in the presence of phytofluene. In view of the findings of Bubb and Murphy⁹ their assumption that phytofluene is responsible for a small and constant contribution to this measurement is clearly inadmissible. Thus they have failed to prove that any of the serum vitamin A measurements in their subjects fell outside the normal adult range. In consequence their contention that a maternal serum vitamin A level of 94 µg/100 ml or more does not present a teratogenic hazard to the fetus remains unproved.—I am, etc.,

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¹²⁵I-Fibrinogen and Blocking of the Thyroid Gland

SIR,—In almost all papers concerning the diagnosis of venous thrombosis with ¹²⁵I-fibrinogen the blocking of the thyroid gland by 100 mg of sodium iodide or 150 mg of potassium iodide is described. The

iodide is given orally 24 hours before injection of ¹²⁵I-fibrinogen and the dose repeated daily for three weeks.

Radioiodine studies of thyroid blocking and recovery¹ have shown that the time taken to achieve the blocked state would appear to be about half an hour after giving 100 mg of iodine orally. At that dose the recovery from the blocked state to 1% of normal uptake is 24 hours and to 100% of normal uptake eight days. The 24-hour blocking time is a disadvantage in the ¹²⁵I-fibrinogen method. With no blocking time the investigation can start immediately.

Because of the relatively low fraction of free iodine in the labelled fibrinogen the amount of radioiodine taken up in the thyroid during the first two hours can be neglected. Thus if iodide is given orally together with the ¹²⁵I-fibrinogen, in practice the thyroid will be blocked as effectively as if iodide were given before injection of the ¹²⁵I-fibrinogen. As a suitable blocking dose I initially give 400 mg of potassium iodide orally and follow it with 200 mg daily for the next three weeks.—I am, etc.,

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- 1 Ramsden, D., Passant, F. H., Peabody, C. O., and Speight, R. G., *Health Physics*, 1967, 13, 633.

Anticonvulsant Osteomalacia and Vitamin D

SIR,—Dr. Claus Christiansen and his colleagues (22 December, p. 695), using photon absorptiometry, have demonstrated in their careful paper a decreased bone mineral content (B.M.C.) in the forearm bones of epileptic subjects compared with a normal control population. B.M.C. was lowest in those epileptics with hypocalcaemia and/or increased plasma alkaline phosphatase. The authors also demonstrated a significant increase in B.M.C. in epileptics when they were treated with 2,000 IU of vitamin D₃ daily. While we accept that photon absorptiometry may offer a new and sensitive method for studying abnormalities of bone mineralization, we feel we should attempt to qualify the enthusiasm with which the authors diagnose the presence of "anticonvulsant osteomalacia." They consider that the presence of any of the following—increased serum alkaline phosphatase, decreased serum calcium, and decreased B.M.C.—indicates the presence of "anticonvulsant osteomalacia."

One of us has previously demonstrated, using polyacrylamide gel electrophoresis, that the significant increase in plasma total alkaline phosphatase levels in adult epileptics is due to an increase in the hepatic isoenzyme.^{1,2} Mean levels of alkaline phosphatase of bone origin were no higher in adult epileptics than in normal adults. Also mean urinary levels of total hydroxyproline were no longer higher in adult epileptics than in normal adults (33.1 and 35.8 mg per 24 hours respectively). In this situation determination of plasma total alkaline phosphatase is of limited value in the diagnosis of osteomalacia.

Hypocalcaemia is common in epileptic subjects without other biochemical indications of osteomalacia and has been found in the absence of histological abnormalities in

bone.³ The lack of increase in serum calcium levels in epileptic subjects which these authors found after treatment with 2,000 IU of vitamin D₃ daily is in agreement with other studies; epileptic subjects with marginal hypocalcaemia were treated with even larger doses, either 10,000 or 50,000 IU vitamin D daily for seven months without effect upon plasma calcium levels.⁴ The presence of hypocalcaemia without other biochemical abnormality in epileptic subjects may therefore not always indicate osteomalacia but may instead reflect a second anticonvulsant effect upon calcium metabolism, as has been suggested by others.⁵ An association between epilepsy and osteoporosis has been recognized for some time⁶ and in our experience is appreciably more common than a finding of osteomalacia. A lowered B.M.C. has not been reported to distinguish between these forms of skeletal rarefaction.

Whatever the reason for diminished B.M.C. the effects of treatment with small doses of vitamin D on B.M.C. are at variance with the results of calcium balance studies, and better understanding of the relation between these measurements is clearly necessary. From their data the authors calculate that the change in B.M.C. is equivalent to a positive calcium balance of 300 mg daily. Such changes do not occur during the treatment of osteoporosis of any origin since the disease does not satisfactorily respond to treatment.⁶ While large positive balances certainly result from the treatment of osteomalacia with adequate vitamin D, "anticonvulsant osteomalacia" typically shows great resistance to the low doses (2,000 IU daily) which were used by these authors.^{7,9} We should therefore like to emphasize that the question of prophylactic vitamin D in the region of 2,000 IU daily merely a cost-benefit exercise, since doses of vitamin D in the region of 2,000 IU daily may prove insufficient either to heal anticonvulsant rickets or even to prevent its occurrence.⁹ We support the final conclusion of these authors concerning the necessity for close clinical supervision of all epileptic patients.—We are, etc.,

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Pyomyositis in London

SIR,—Colonel D. D. O'Brien (12 January, p. 78) is right in saying that many pyomyositis patients have a solitary muscle abscess, but in my letter (27 October, p. 235) I made no claim to the contrary. Having stated that pyomyositis was the formation of abscesses