be widespread I now accept the majority view that there should be a second person present to give the anaesthetic; but this would bring no gain in safety unless that person fully understood and kept constantly in mind the peculiar danger of anaesthesia in the dental chair. Whether this person needs to be qualified in medicine or dentistry is open to question. I think not and have suggested^{6,7} the establishment of an alternative grade.—I am, etc.,

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Depression and Organic Disorder

SIR,—On my return from abroad I have read the correspondence from Dr. G. Dodds (22 September, p. 638) and from Dr. Irene P. Rowlands and my colleague Mr. A. N. Smith (27 October, p. 234).

Dr. Rowlands interprets me correctly when she writes that I “merely intended that one must not lose sight of depression as the ‘writing on the wall’ in carcinoma occurring in later life.” It was indeed an off-the-cuff remark in closing the meeting as I noted that this feature had not arisen in the discussion at the clinicopathological conference. Nevertheless, Dr. Dodd’s remarks on the many other reasons why such a patient might well develop depression are perfectly pertinent and Mr. Smith has answered appropriately the question that he raised relating to the absence of this factor in the retrospective survey. In the event, my closing remark has had the advantage of greater publicity than it would have achieved if I had surrounded it with provisos.

I am unsure what Dr. Rowlands meant by my “usual flair for diagnostic slogans,” but as the tone of the letter is clearly friendly I shall take the liberty of assuming that flair compensates for slogans.—I am, etc.,

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Effect of Beta-receptor Stimulation on the Platelet Count

SIR,—The intravenous infusion of adrenaline causes an instant release of platelets from the exchangeable splenic platelet pool (E.S.P.P.), resulting in a simultaneous increase in the peripheral platelet count.¹⁻⁴ The mechanism behind this effect of adrenaline on the E.S.P.P. is, however, unclear. In view of the fact that adrenaline stimulates the alpha- as well as the beta-receptors it was considered of interest to study the possible effect of more selective stimulants on the peripheral platelet count. Therefore five healthy male volunteers aged 24-39 (mean 30) years were studied during an intravenous infusion of isoprenaline lasting 20 minutes (0.015 µg/kg body weight/min) on two occasions. Ten minutes before the second

infusion the subjects received 10 mg of propranolol intravenously at a constant rate over a 10-minute period. For determination of the platelet count venous blood was collected at five-minute intervals throughout the experiment. The last sample was drawn 20 minutes after the withdrawal of isoprenaline. The counting of platelets was carried out with a Coulter counter Model F, as described elsewhere,⁵ and the absence of platelet aggregates in the blood samples was confirmed by phase microscopy.

During the first experiment a progressive fall in the platelet concentration was observed in all subjects. After 15 minutes of isoprenaline infusion the mean platelet count had decreased to 82% of the baseline value. After the withdrawal of isoprenaline the platelet count referred to the baseline within 20 minutes. During the second study, when the subjects were pretreated with propranolol, there was no significant change in the peripheral platelet concentration in response to isoprenaline administration.

Thus while adrenaline causes an increase in the peripheral platelet count, there is a decrease in the peripheral platelet concentration in response to isoprenaline. This latter effect of isoprenaline can effectively be blocked if the subject is pretreated with propranolol, a beta-blocking agent. Since it is known that adrenaline exerts its effect on the peripheral platelet count by decreasing the E.S.P.P.¹⁻⁴ it may well be that isoprenaline exerts an opposite effect on the spleen. These effects might well be mediated by alpha- and beta-receptors.—We are, etc.,

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Anaemia in the Elderly

SIR,—Dr. J. H. Thomas (3 November, p. 288) reviewed the causes, types, and treatment of anaemia in the elderly and pointed out that a deficient vitamin C intake is associated with a normocytic and sometimes a macrocytic anaemia. Concomitant with the iron deficiency anaemia commonly found in the elderly, a deficiency of vitamin C is often encountered.^{1,2} The metabolism of iron is closely related to that of vitamin C.³ Iron absorption is enhanced in the presence of vitamin C,⁴ and its mobilization from transferrin to ferritin in the liver and vice versa is dependent on an adequate supply of ascorbic acid.^{5,6} Optimal haemopoiesis is possible only in the presence of iron and the maturation factors, vitamin B₁₂, folic acid, pyridoxine, and also ascorbic acid, in the bone marrow. In the presence of an adequate supply of the maturation factors, but with a deficiency of vitamin C, optimal haemopoiesis does not take place during adolescence^{2,7} or in the elderly.^{2,8} Adequate supplementa-

tion in a ratio of five parts of vitamin C to one of iron has been shown to enhance haemopoiesis in the elderly in the absence of deficiency of the other factors.^{3,8,9}

Dr. Thomas suggested a daily intake of an adequate amount of protein, 10-12 mg of iron, 2-4 µg B₁₂, 100 µg folic acid, 2 mg pyridoxine, and 35 mg vitamin C, to ensure an adequate supply of these haemopoietic factors for the daily needs of elderly people. Though a daily intake of more than 10 mg of vitamin C is adequate to prevent scurvy,¹⁰ 35 mg is not an adequate daily intake to satisfy haemopoiesis.^{2,8} Larger doses are necessary to satisfy other metabolic demands of a physiological¹¹ or of a pathophysiological nature.¹²⁻¹⁴—We are, etc.,

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E.S.R. versus Plasma Viscosity Readings in the Old

SIR,—Following Dr. F. I. Caird’s comprehensive account of laboratory findings in the old (10 November, p. 348) I would like to draw attention to some work relating raised erythrocyte sedimentation rate (E.S.R.) with normal or low plasma viscosity to either abnormally low packed cell volume or reduced plasma albumin level.¹ Albumin disperses rouleaux formation and therefore tends to reduce the E.S.R. whereas globulin enhances rouleaux formation, tending to accelerate the E.S.R. Harris¹ found that in some old people plasma albumin levels may be reduced in association with malnutrition or malabsorption in the absence of anaemia.

In our hospital group we have used plasma viscosity estimations in place of E.S.R.s for over eight years, the effects due to low packed cell volume or to changes in the red cell surface thus being eliminated.² Low plasma viscosity readings suggest hypoproteinaemia, normal readings virtually exclude inflammatory response from whatever cause, and readings raised above normal require investigation. The test has the advantage of being cheap (virtually the cost of the apparatus), it is rapid (20 seconds per test) reproducible (coefficient of variation < 1%), and suitable for postal samples (unlike the

E.S.R.), and it is probably less of a hepatitis hazard to technicians than the E.S.R.—I am, etc.,

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¹ Harris, G. J., *Journal of Medical Laboratory Technology*, 1972, 29, 405.

² Eastham, R. D., and Morgan, E. H., *Journal of Medical Laboratory Technology*, 1965, 19, 70.

Neonatal Jaundice and Maternal Oxytocin Infusion

SIR,—We agree with Mr. W. G. Mills and Miss M. C. R. Fisher (22 September, p. 637) that reliance on the mother's menstrual data can result in an inaccurate estimation of gestational age in the newborn infant. Nevertheless, this method, together with regular obstetric assessment, continues to be that most widely used in routine clinical practice and was that used in determining the maturity of infants in our study (1 September, p. 476). Each infant was examined by one of us (R.R.) and in instances when there were obvious discrepancies the scoring system of Dubowitz *et al.*¹ was used. This has recently been shown, in experienced hands, to provide an accurate assessment of gestational age.² Since the infants in all three groups were assessed in the same way it is improbable that the infants of mothers whose labour had been induced should be more gestationally immature than those in the remaining groups. This does not, however, eliminate the possibility that the increased bilirubin levels in these infants were the result of a functional immaturity of the hepatic enzyme systems.—We are, etc.,

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¹ Dubowitz, L. M. S., Dubowitz, V., and Goldberg, C., *Journal of Pediatrics*, 1970, 77, 1.

² Hancock, B. W., *Archives of Disease in Childhood*, 1973, 48, 152.

Norwegian Scabies and Monocytic Leukaemia

SIR,—Dr. W. D. Paterson and others (27 October, p. 211) report a case of Norwegian scabies in a patient under treatment with azathioprine and prednisolone, and briefly discuss the predisposing factors, quoting the patient of Dostrovsky *et al.*¹ who had chronic lymphatic leukaemia. The following report refers to a case of Norwegian scabies, megaloblastic anaemia and monocytic leukaemia, an unusual combination of diseases.

A woman of 82 years was admitted to a nearby hospital with a confusional state, anaemia, and a generalized pruritic eczematoid rash with some features of scabies, diagnosed by the late Dr. James Coburn as Norwegian scabies. A blood count showed haemoglobin 9.0 g/100 ml and white cells 3,000/ μ l (polymorphs 81%, lymphocytes 8%, eosinophils 1%). Bone marrow showed slight megaloblastic changes and approximately 25% monocytoid cells of different degrees of maturity. The serum B₁₂ level was 90 pg/ml

and the serum folate was 0.9 ng/ml. She was treated with B₁₂ and folic acid for the anaemia, and Eurax (crotamiton) and hydrocortisone for the skin condition, and later transferred to this hospital. Three weeks after her initial diagnosis the blood showed: haemoglobin 7.6 g/100 ml and white cells 11,000 per μ l (polymorphs 65%, lymphocytes 13%, monocytes 22%). A repeat bone marrow biopsy at this time showed normoblastic erythropoiesis, an increase of early granulocytes, and a similar number of monocytoid cells. Folic acid and B₁₂ were continued and the haemoglobin rose to 10.8 g/100 ml. She returned home.

Two years later she was readmitted with a recurrence of the skin disease. There was exfoliative dermatitis, particularly affecting the hands and feet, with lesions suggesting scabies. There was slight lymph node enlargement, but no enlargement of liver or spleen. Mites were seen in skin scrapings. Haemoglobin was 12.0 g/100 ml and white cells 8,900 per μ l (polymorphs 64%, lymphocytes 14%, monocytes 20%, eosinophils 2%). Bone marrow again showed an increase of promyelocytes, some large primitive blast cells, and approximately 20% of cells of the monocyte series, including 15% monoblasts. A diagnosis of subacute monocytic leukaemia was made. No specific anti-leukaemia treatment was given, but the skin was treated with a three-day course of benzyl benzoate and later a topical corticosteroid ointment. She returned home but died six months later in another hospital following a cerebral haemorrhage due to a fall. The hands and feet showed chronic hyperkeratotic changes. Haemoglobin was 11.4 g/100 ml. No other investigations were performed.

I am grateful to Dr. D. W. Dawson for the folate and B₁₂ results, and for letting me inspect the original marrow smears.

—I am, etc.,

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¹ Dostrovsky, A., Raubitschek, F., and Sagher, F., *Dermatologica*, 1956, 113, 26.

Lung Cancer and Smoking

SIR,—I write to express fundamental disagreement with Dr. R. C. Hobbs (24 November, p. 490), who suggests legislation to curb the use of tobacco.

It should never be the function of the medical profession to interfere with the liberty of the individual unless it can be proved (as in the case of drug addiction) that society as a whole is endangered. From this point of view the cigarette is much less a candidate for legislation than alcohol. The effects of cigarette smoking are essentially personal.

Many people smoke cigarettes with complete awareness of the hazards involved. For them a possibly shorter life with cigarettes is preferable to a longer life without. If that be their considered philosophy, no doctor has the right to interfere. It is no part of our calling to be nursemaids of the public. We should respect the personality of our patients, however inconsistent their actions may be with our preconceived notions of their welfare.—I am, etc.,

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Huddersfield

SIR,—Many doctors must have been perturbed by Sir George Godber's report on the nation's health.¹ The statements that more than 25% of deaths are due to ischaemic heart disease and 40% of cancer deaths are from lung cancer should surely give fresh impetus to all doctors and paramedical personnel to combat the smoking habit. But it seems that many members of the medical profession are unconvinced of the dangers of smoking.

If the medical profession is so little concerned about this problem there is nothing that health education of the public can do. However, if we can make a more concerted effort, then a start could be made by considering smoking in the same light as occupational hazards—that is, that protection has to be "imposed." I suggest that the following measures be considered: (1) In all public buildings administered by local government authorities no smoking should be permitted and a fine imposed for non-compliance (similar to the penalty for spitting). (2) At selection committees of key figures such as district nurses, health visitors, and public health doctors smoking should be discussed. (3) Smoking by public health staff while on duty should not be permitted. (4) Every effort should be made to discourage teachers from smoking during their training, and smoking should not be permitted in schools. (5) No smoking should be permitted in sports centres and swimming baths provided from public funds. Sports instructors, who are often objects of great admiration to young people, should not smoke while on duty.

It is extremely difficult to bring these matters to the fore because many of one's most congenial colleagues smoke and one is made to feel unconvivial and self-righteous by raising them.—I am, etc.,

THERESA E. WATTS

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¹ Department of Health and Social Security, *On the State of the Public Health, Annual Report of the Chief Medical Officer for the Year 1972*. London, H.M.S.O., 1973.

Medicine in the Common Market

SIR,—In his article on this subject (24 November, p. 478) Mr. P. R. J. Vickers seemed to be suggesting that our standards of specialist training should be reduced, otherwise we might expect to lose doctors to the other members of the European community. The articles from West Germany, Italy, and France, however, all emphasized deficiencies which are not present in the United Kingdom. Is it not possible that the rest of Europe might in some respects follow us, or are we always to adapt to them whatever the consequences?

It was a pity also that Mr. Vickers chose to illustrate his point by referring to radiology and anaesthetics and not some specialty of which he has more experience, where his criticisms of the colleges may have some substance. In both these disciplines, despite increasing additions to the syllabus and the complications of technical progress, the period of specialist training remains shorter than in many other specialties. It may also be of some interest that a radiologist without command of the language of the country in which he is employed is at a considerable