for a place for his son in a medical school, his son having passed "A" levels (not at his first attempt), with one B and two Ds. I have heard of several medical colleagues whose sons or daughters have failed to get into a medical school because they did not get high enough grades at "A" level.

What are we training doctors for? Are they all destined to become high-grade specialists or Fellows of the Royal Society? Doctors in general practice do not require an esoteric high-brow intellect, yet nearly half the graduates are destined for general practice. They require patience, humanity, common sense, and an ability for hard work in the field.

Hippocrates favoured the training of the sons of doctors in the profession and it used to be the practice in Britain. I suggest that it is high time that we returned to it.—I am, etc.,

N. GEOFFREY NICHOLSON

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SIR,—I most strongly disagree with the point of view expressed by Mr. Oliver Dearlove (10 November, p. 360). I do not consider that a medical home background is of any great benefit to one's training—indeed it all too often leads to the case of a son being expected to follow in his father's footsteps, even when he is not strongly motivated. This can lead to unfortunate failures and a wastage of valuable medical school places.

Furthermore, although "A" level results may not be the best guide, they are a good standard. I fail to see why, if somebody cannot get a place by normal methods—that is, the Universities Central Council on Admissions (where interviews usually are taken into account)—his father's position and willingness to finance advertising should entitle him to any advantages.—I am, etc.,

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Ambulance Personnel

SIR,—I read with satisfaction the letter from Dr. W. S. Parker (10 November, p. 359) concerning the Brighton ambulance men who participated in the mobile coronary care scheme. The attitude they and their union adopted typifies the professionalism we need to encourage in this important body of men; it does much to establish them as paramedical technicians—much more than the current regrettable industrial action. I have a deep interest in the status of ambulance men, being the medical representative of the Institute of Certified Ambulance Personnel on the Board of Registration of Medical Auxiliaries.

I would like to point out that there is provision for ambulance men who have obtained the Diploma of the Institute of Certified Ambulance Personnel to obtain admission to the National Register of Medical Auxiliary Services of the Board of Registration of Medical Auxiliaries at B.M.A. House. It is true that this is a small beginning, but all paramedical professions had to start somewhere and I believe that such a recognition of training and qualification will be greatly to the advantage of ambu-

lance men from a professional point of view. Physiotherapists, occupational therapists, chiropractors, and others who have comparably intimate contact with patients originally registered in this way but are now recognized on the Statutory Register of Professions Supplementary to Medicine. Such graduation is the essential function of the Board.

Doctors should encourage in ambulance men a professional attitude at all times; this begins by recognizing their importance and enhancing their pride in professional status.

—I am, etc.,

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Anaesthetic Safety Devices

SIR,—Dr. C. Burke (3 November, p. 229), no doubt unintentionally, gives the impression that common sense regarding the oxygen supply to anaesthetized patients might be the prerogative of anaesthetists in Canada and the United States. I can reassure him that fuel cell and oxygen electrode meters are readily available in Britain, albeit of American manufacture, with their advantages of displaying the inspired oxygen concentration received by the patient and giving an audible and visible warning if the concentration is above or below preset limits. In this way a failure of oxygen or nitrous oxide supply can be made known.

A fail-safe device as described by Mr. R. J. Davies (3 November, p. 229) is clearly an additional desirable feature, while an alarm to detect accidental disconnection of the patient from the anaesthetic circuit, particularly during controlled ventilation, would seem to be essential.

If these aids to the safety of anaesthesia are not generally in use it might suggest a surprising complacency by specialists in this field.—I am, etc.,

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Aerosols and Asthma Deaths

SIR,—It is usually assumed that the decrease in the number of deaths from asthma which followed the official warning about pressurized aerosols containing sympathomimetic drugs¹ was due to the stricter controls on the supply of these aerosols. This hypothesis has, however, been questioned by several authorities.²⁻⁵ Another possible explanation for the decrease in mortality after the warning was issued could have been a change in the attitude of general practitioners to the treatment of asthma, and this may have been the reason why more patients with asthma were admitted to hospital at that time,⁶ and why there was an increase in the number of prescriptions for corticosteroid drugs,⁷ which could both have contributed to the reduction in mortality. It is apparent from the recent correspondence in these columns that there is no convincing evidence to incriminate aerosol propellants as a major cause of death in asthmatic patients.⁸⁻¹¹

Though the reasons for the reduction in asthma mortality are not clear and may not be directly related to the decreased use of pressurized aerosols, it is obvious that we must constantly endeavour to ensure that our

patients do not overuse pressurized aerosols of any type. We must not, however, deny them this most efficient method of bronchodilator drug administration. Ever since the possible association of increased deaths from asthma and aerosols was suggested many doctors have been reluctant to prescribe pressurized aerosols because of the fear that they may be used indiscriminately. As a result, oral sympathomimetic amines are frequently prescribed, sometimes in combination and in massive doses, in an attempt to control asthmatic symptoms which could be more rapidly and effectively relieved by the same drugs in much smaller doses if given by aerosol. Pressurized aerosols are often reserved for patients with severe asthma because of their potential dangers, and patients with less severe symptoms are often treated with oral therapy. It is, however, wrong to withhold inhalers from patients with mild asthma as this is denying them the most efficient method of relieving their symptoms, and with such patients there is little or no danger of inhaler overuse as their symptoms are not likely to be distressing enough to encourage it.

The risks of toxic effects from oral sympathomimetic drug therapy are often overlooked. Salbutamol, the most widely used bronchodilator drug, is manufactured in two tablet strengths, 2 mg and 4 mg, and these are commonly prescribed in preference to the pressurized aerosol, which delivers 100 µg of salbutamol per dose. Surely it is irrational to prescribe any drug by mouth in a dose which is 20 or 40 times larger than the effective dose by inhalation. Even more difficult to justify is the concurrent use of sympathomimetic amines like salbutamol by mouth and by inhalation.

Dose for dose, inhaled salbutamol is cheaper, more rapid in action, more effective, and has less undesirable side effects than oral preparations. The medical profession has perhaps been alarmed by the continued debates about the potential dangers of aerosols in asthma and has tended to favour the use of oral sympathomimetic drugs in preference to aerosols. There should be, however, only one indication for the use of oral sympathomimetic amines in asthma—when, for any reason, a patient is unable to use a pressurized aerosol.—I am, etc.,

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Relapsing Polychondritis and Pulseless Disease

SIR,—With reference to your leading article on "Relapsing Polychondritis" (16 June, p.

627), all earlier workers failed to demonstrate specific antibodies in the serum of patients.¹⁻⁷ Even Hermans' group,^{8,9} as you mention, failed to detect antibodies to cartilage or chondrocytes using very elegant methods, but the increased lymphocyte transformation detected by them occurred in all of three cases of relapsing polychondritis and also in nine out of 12 cases of rheumatoid arthritis and in one of two cases of gouty arthritis among the control subjects, in the presence of the same antigens. Increased lymphocyte transformation by itself is not accepted as an indisputable index of cell-mediated immunity because a humoral or a cell-mediated mechanism, or both in combination, or a "non-specific" mitogenic effect could each transform lymphocytes.¹⁰ The possibility of the occurrence of a "non-specific" mitogen in the antigenic material used was not excluded. Further, as the increased lymphocyte transformation occurred in many cases of rheumatoid arthritis and one of gouty arthritis, the operation of a factor common to all these arthritides (including relapsing polychondritis) seems likely.

Hughes *et al.*¹¹ have recently demonstrated, by the immunofluorescent technique, positive human fetal cartilage staining in two out of three cases of relapsing polychondritis and in all of 12 cases of rheumatoid arthritis but no staining in 32 control subjects. Again, like Herman's findings, their results seem to indicate the occurrence of an antibody in the serum of patients with these chronic arthritides. We¹² have shown the presence of cell-mediated immunity to human laryngeal cartilage proteoglycan (kindly supplied by Dr. Helen Muir of the Kennedy Institute) by two in vitro methods—macrophage migration inhibition¹³ and lymphocyte transformation—in two patients with relapsing polychondritis, whereas nine control subjects suffering from a variety of arthritides gave negative results. Further, we were unable to show the presence of humoral antibodies to cartilage in the serum of these two patients by immunofluorescent staining (kindly done by Dr. G. Loewi, Dr. G. Johnson, and Mr. D. Kingston of this unit separately) or to proteoglycan by gel immunodiffusion. Subsequently we were in a position to study three other cases of relapsing polychondritis (to be published). The following results were obtained in them when the same four tests were applied. The first case showed no cell-mediated immunity and in the two humoral tests showed positive immunofluorescent staining of fetal cartilage but negative immunodiffusion with proteoglycan. The second case showed positive cell-mediated immunity and weakly positive immunofluorescent staining of cartilage but negative immunodiffusion with proteoglycan. The third case showed positive cell-mediated immunity but negative humoral tests like the two reported cases.¹² These findings are difficult to interpret. We feel that humoral immunity could be an early manifestation of the disease, while cell-mediated immunity could be a late manifestation. The interesting feature is the development of cell-mediated immunity against a normal biochemical constituent of the tissues affected in the disease process.

His-chemical studies indicate that the chondromucoprotein of the cartilage ground substance is affected in the pathological process of the disease. There is some evidence^{14,15} to suggest that the arterial walls may contain a similar substance and this could perhaps

explain the involvement of the arteries in the disease. One of the two patients under our care referred to above¹² (in fact, the first) presented as a case of Takayasu's syndrome, and over the years she developed both arthritis and finally chondritis. She showed a marked degree of cell-mediated immunity to human laryngeal cartilage proteoglycan. Another patient referred for our opinion had angiographically established Takayasu's disease but had no evidence of arthritis or chondritis clinically or radiologically. She showed a mild degree of cell-mediated immunity to the same proteoglycan antigen. It could well be that the arteritis of Takayasu's type and polychondritis of relapsing type are variants of the same disease or disease process.—We are, etc.,

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The Blind in Hospital

SIR,—Some years ago I spent three months in a surgical ward. During that time the sister spoke to me exactly once. Doubtless she was often in the ward, but I never knew because I am totally blind. It would have been a help because the nurses were continually changing. Over a period of two or three weeks I was asking nurses to ask Dr. X. to see me, without success. Eventually I phoned him from my bed and asked him if he had had a holiday. He was furious and said that he had been in the ward every day. The staff often forget that you are blind and completely fail to communicate. The situation was desperate for me and I would prefer to die in the gutter rather than go back.

If I am blind I never know who you are and rarely if you are there at all, because of background noise. People think I do not want to speak because I cannot speak first. Would it not be possible to hang up a little label saying, perhaps, "Blind but not deaf," or "Tell me you are here"?—I am, etc.,

R. SPENCE

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Clinical Experience with the Dalkon Shield

SIR,—We were interested to read Dr. J. S. Templeton's letter (8 September, p. 542) concerning our preliminary report on our clinical experience with the Dalkon Shield (21 July, p. 143) and would like to comment on some of the points that he has raised.

One of the prime objectives of our paper was to point out that in our hands the Dalkon Shield results were not as good as those reported by Davis¹ and his colleagues who had developed the intrauterine device (I.U.D.). We therefore presented our data in a similar way to that which they had done, using the Pearl index and the life table method.² It is possible that their results might be related to the use of a spermicide, which Davis recommends in the first few months of use. Furthermore, in the multicentre analysis reported by the family planning unit at the University of Exeter, there is no information on spermicide usage either.³ We quoted the use of spermicide in 5% of our cases. Variation in spermicide usage may be one of the factors related to the widely differing pregnancy rate in these 10-12 clinics, of which, incidentally, ours is one (the pregnancy rate in these clinics varies from 0.8 to 8.4%). We, too, have little doubt that valuable complementary data are derived from studies such as the multicentre trial quoted as well as experience in an individual clinic.

We have used the accepted definition of parity as a pregnancy which has gone beyond 28 weeks. Therefore our nulliparous patients were those who had been pregnant and had had a pregnancy terminated; thus they were able to accommodate the larger Dalkon Shield without any difficulty. It was in the two patients in whom the uterine cavity was small that we used the smaller Dalkon Shield.

As far as the training of doctors in the insertion of I.U.D.s is concerned, we accept that this may well contribute to a higher pregnancy rate, bearing in mind that if an I.U.D. is to be inserted in large numbers, it is to be expected that the skill of individual operators will vary.

We have since had our data analysed according to the currently recommended cumulative life table method⁴ and our pregnancy rate findings with this method of analysis were not dissimilar from those reported.—We are, etc.,

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One Thousand Vasectomies

SIR,—With reference to the report by the staff of the Margaret Pyke Centre (27 October, p. 216) I was interested to note that it was considered unreasonable to insist on sperm-free specimens before taking the responsibility of pronouncing sterility.

I have performed some 5,000 vasectomies