bladder cancer than for lung cancer such cases might be expected." I have two such patients whose abbreviated case histories are here reported.

A man born in 1902 smoked 85 g (3 oz) of tobacco per week, making his own cigarettes. He had a right pneumonectomy for squamous cell carcinoma in 1957. He remained fairly well and at work until 1966, when he was found to have a hiatus hernia. This was repaired surgically. In 1971 he developed haematuria which was shown to be due to a well-differentiated transitional cell carcinoma of bladder. This was treated with a radical course of radiotherapy, 6000 rads in 42 days, with an excellent result. He is still alive.

A man born in 1895 smoked 20 cigarettes a day from 1917 to 1938 and then a pipe. A right middle and lower lobectomy was done in 1961 for an anaplastic carcinoma of lung. In May 1971 he had haematuria and cystoscopy showed a carcinoma of bladder. A satisfactory biopsy specimen was not obtained. It was treated by cystodiathermy. In November 1973 new opacities developed in the right lung, the largest being 3·5 × 6 cm. These were considered to be metastases from the bladder carcinoma. They increased in size and he died in May 1975.

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Staffing in hospital laboratories

SIR,—Those of your readers who have not had an opportunity to study the policy statement of the Institute of Medical Laboratory Sciences on future staffing in the medical laboratory service will have gained a totally erroneous impression of the document from the letter you published from consultant pathologists in the Trent Region (12 February, p 441).

The implication of that letter seemed to be that the institute in its policy statement recognised no place for medically qualified staff; the institute's assumption, however, was that it was inappropriate to consider them in the same context as non-medical staff. As the first paragraph of the policy statement pointed out, medical staff were therefore excluded from the subsequent discussion. The statement did include, however, arguments for integrating the "scientific" and "technical" staff of laboratories as the distinction between the two is becoming increasingly artificial on grounds of either function or qualification. Those who work in medical laboratories will know just how trivial is the difference in function and, as the institute's evidence to the Royal Commission on the NHS pointed out, more graduates are now entering the "technician" class than are entering as "scientists." In these circumstances the distinction which the authors of the letter try to make between graduates and technicians does not bear examination.

Your correspondents choose to attack the policy statement as containing "many assumptions and inaccuracies" but fail to quote a single example. In contrast, inaccuracies can be demonstrated in their letter: two in the first sentence. This institute was formerly known as the Institute of Medical Laboratory Technology, not Technicians, and the title and content of our policy statement referred to staffing *in* the medical laboratory service, not *of* the service as your correspondents claimed. The institute observed this important distinction by deliberately refraining from comment on the *medical* staffing of hospital laboratories; the courtesy of reciprocation would have been appreciated.

There has been no evidence during the months since publication of the policy statement to justify its condemnation as "a divisive document which could sour the currently good relations built up over the years between all grades of laboratory staff"—perhaps because most readers did not misinterpret it. Good relations in the service will not be encouraged by a letter inciting others to inflammatory correspondence. In the interest of the medical laboratory service it would have been better left unwritten.

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Chemotherapy for varicella-zoster infections

SIR,—Dr R P R Dawber (29 January, p 286) makes a number of dogmatic statements which should not go unchallenged. MacCallum and I[1] recommended cytarabine and vidarabine in the treatment of severe zoster in our book five years ago. We did not refer to clinical trials because there had not been any at that stage. The drugs were quite new. In the case reports[2] that gave rise to your leading article (18 December, p 1466) I actually tried to measure the fate of the virus when it was treated with the antiviral drug and this, as well as recording the clinical course, is probably as much as one can hope to do in rare and life-threatening conditions like purpura fulminans in zoster. It is difficult or impossible to get enough patients with severe herpes simplex and varicella zoster manifestations to make a double-blind controlled trial possible. The best one can hope for often is to monitor the virus shedding. I have given examples of this elsewhere.[3] A double-blind controlled trial on the relative merits of cytarabine and idoxuridine in uncomplicated zoster is in progress at the moment.

I agree with Dr Dawber that the reason for treating even uncomplicated segmental zoster is not only to deal with the immediate problem of pain but with postherpetic neuralgia. I do not agree with his statements about the concentration of idoxuridine to be used. MacCallum and I[1] have already pointed out that a 35% solution of idoxuridine in dimethylsulphoxide (DMSO) is more effective than a 20% solution, and that in turn more effective than a 5% solution. I have treated in excess of 2000 patients and less than 5% get severe histamine reaction from the DMSO. In none have there been necrotic skin changes. If bought in bulk idoxuridine is not very expensive and the cost of treating the average patient with zoster by continuous topical application of a 35% solution, which I now use, varies between £15 and £30 depending on the extent of the lesion. The number of admissions for zoster to the infectious disease unit has dropped to about one-twentieth of what it used to be before there was an active treatment available, so the outlay on drugs is paid for many times over by keeping people out of hospital.

Dr P A M Walden and others (*BMJ*, 5 February, p 378) state that I have reported "a good clinical response in two out of three cases with herpes zoster infections.[2]" If the authors refer to the paper they quote they will see that it describes the use of vidarabine in two cases of severe chickenpox in adults and the use of the drug in that extremely rare condition purpura fulminans in zoster.

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1 Juel-Jensen, B E, and MacCallum, F O, *Herpes Simplex, Varicella and Zoster*. London, Heinemann, 1972.
2 Juel-Jensen, B E, *Journal of Antimicrobial Chemotherapy*, 1976, **2**, 261.
3 Juel-Jensen, B E, *Journal of Antimicrobial Chemotherapy*, In press.
4 Juel-Jensen, B E, and MacCallum, F O, *British Medical Journal*, 1974, **3**, 41.

Royal College of General Practitioners

SIR,—Many founder members of the Royal College of General Practitioners must have been very depressed by Dr I Capstick's Personal View (5 February, p 373). After two years in resident posts I entered general practice early in 1930 because I wanted to deal with "people" and not just to treat "cases" with various anatomical and physiological defects. After some years (seven as secretary of a BMA division) I realised that standards in general practice varied enormously and I was proud to join a professional body that was concerned solely with the standards and quality of work in general practice—a body that was able to contradict Lord Moran's statement that "GPs were the doctors who had fallen off the hospital promotion ladder." Our college has done this.

If general practice is to be recognised as a specialty in its own right our college must behave as the other royal colleges have done. No doctor can become a full consultant now without a higher qualification (FRCS, MRCP, MRCOG). Our college should press the authorities to rule that no doctor can become a full partner or unsupervised singlehanded GP without the MRCGP diploma.

Since retiring at the end of 1968 I have been saddened to see how mercenary the profession has become. The idea of doctors arguing like dockers about hours of work and overtime pay appals me. But if the younger generation want it that way we cannot stop them. They have been taking the DRCOG because it gave them higher fees in the NHS. If the MRCGP will do the same they will take that too. At least we shall know that they have achieved a certain standard of knowledge. But in the years to come I wonder if their old patients will send them Christmas cards and come down to visit them, as some of mine still do.

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SIR,—To judge from his writings in your columns and elsewhere Dr D R Cargill (19 February, p 508) does not always appear to view with detachment the postgraduate scene in relation to general practice. However, he has a point in emphasising the need to seek out the professional and informed views of general practitioners concerning vocational training.

This letter seeks to draw attention to a survey, conducted with support from the Royal College of General Practitioners and the Scottish General Medical Services Committee.

Together with colleagues in the University of Dundee I am completing an analysis of views of a one-in-eight sample of Scottish general practitioners concerning the consultation. From this it is clear that GPs (and a sizable proportion in the survey were not college members) do not share Dr Raine Roberts's view (p 508) on the consultation. There is very strong support for helping new entrants to general practice to readjust their approach to clinical medicine to suit the different circumstances of general practice.

May I take this opportunity to thank the 350 general practitioners who have assisted in the survey?

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Metabolic effects of salbutamol

SIR,—Intravenous salbutamol may produce significant metabolic effects[1 2] and the recent report by Mr A Neville and others (12 February, p 413) comparing the metabolic effects of aerosol and intravenous salbutamol draws attention to these. In particular, marked hypokalaemia and rises in free fatty acid levels have been observed following intravenous salbutamol. This is worrying since these changes have been implicated in the genesis of cardiac dysrhythmias and it is considered advisable to monitor the electrocardiogram when administering intravenous infusions of salbutamol in the acute asthmatic state. Indeed, Leitch *et al*[2] have shown that the cardiovascular effects of intravenous salbutamol are pronounced when hypoxia is combined with hypercapnia.

The metabolic changes following oral, intravenous, and aerosol salbutamol differ only in magnitude rather than effect and are a consequence of the differing metabolism of the drug when administered by these routes. Evans[3] has studied the absorption, metabolism, and excretion of salbutamol in detail. Salbutamol absorbed from the gut is conjugated in the liver to a metabolite with negligible adrenoreceptor activity, and this is excreted in the urine. Intravenous salbutamol tends to remain in the free form and only 27% of the drug excreted in the urine over 24 h has been metabolised. Most of the aerosol-administered salbutamol is swallowed, but if salbutamol is introduced directly into the lungs through a bronchoscope most of the dose in the plasma and urine is present as free salbutamol, suggesting that it is not metabolised in the lung. There is also a slower excretion rate for aerosol salbutamol which probably indicates its slow removal from the lungs.

Hence administering salbutamol intravenously produces higher plasma levels of the active drug with higher magnitudes of change in the metabolic and systemic effects of beta-2 agonism. However, oral salbutamol may produce changes in insulin, glucose, glycerol, and free fatty acid levels,[4] although hypokalaemia does not seem to occur,[5] and aerosol salbutamol can lower plasma potassium in certain hyperkalaemic conditions such as familial hyperkalaemic paralysis.[6]

Aerosol salbutamol has many advantages over oral and intravenous salbutamol. It produces negligible metabolic changes with a lower incidence of cardiovascular and other systemic side effects. More important, Hetzel and Clark[7] have shown that aerosol salbutamol produces a prolonged bronchodilator effect and is more potent than an equal intravenous dose.

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1 Goldberg, R, *et al*, *Postgraduate Medical Journal*, 1975, **54**, 53.
2 Leitch, A G, *et al*, *British Medical Journal*, 1976, **1**, 365.
3 Evans, M E, *et al*, *Xenobiotica*, 1973, **3**, 113.
4 Taylor, M W, *et al*, *British Medical Journal*, 1976, **1**, 22.
5 Deacon, S P, *Lancet*, 1976, **1**, 1302.
6 Wang, P, and Clausen, T, *Lancet*, 1976, **1**, 221.
7 Hetzel, M R, and Clark, T J H, *British Medical Journal*, 1976, **2**, 919.

Respiratory distress syndrome

SIR,—Further to your leading article (11 December, p 1403) I notice in your references you omit Geoffrey Dawes of Oxford,[1] who has made the single most important contribution to the prevention of respiratory distress syndrome this century. Surfactant and betamethasone are interesting research points, but simple physiology is more important.

When a child is born only 60-70% of its total vascular capacity is patent. Its blood volume at birth will also be 60-70% of what it should be, the remainder being in the placenta. If the cord is clamped before the lungs have expanded and before sufficient time has been allowed for the transfusion from the placenta to the baby to occur (limited by physical principles, diameter of blood vessels, length of cord, and negative pressure produced by inspiratory effort) this 60-70% of the total blood volume will suddenly be redistributed into the potential 100% vascular capacity. The "late arrivals" and most sensitive organs, the lungs, will suffer most and other vascular organs such as the liver to some extent. This will limit access, and possibly production, of the normal corrective enzymes to the pulmonary tissue. Respiratory distress syndrome will gradually develop.

In 1975 and 1976 we delivered 1754 babies in this unit (prematurity rate 3%, caesarean section rate 5%, forceps and ventouse rate 10%, induction rate 21%, and 6·5 of the mothers were aged 40 years or more). Our perinatal mortality rate was 8/1000 and our neonatal death rate 4/1000. The rule for all newborn babies here is, delayed clamping of the cord (if the cord is placed across the palm and four fingers gently applied to it its decompression with each vigorous inspiratory effort by the baby can be felt), thereafter immediate oxygenation, warmth, glucose via the intragastic route (to help the liver to do its work), and no handling or drips. We have one advantage over most units: the neonatal paediatrician is the obstetrician. The above approach was adopted on the basis of Dawes's teaching and I think he is right. Can you quote better results?

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1 Dawes, G S, *Foetal and Neonatal Physiology*, pp 161-2. Chicago, Year Book Medical Publishers, 1968.

Rising incidence of childhood diabetes

SIR,—The nation-wide interest in diabetes mellitus in childhood[1] is focused mainly on possible aetiological factors. It seems timely to draw attention to a considerable increase in the incidence of childhood diabetes. The numbers of newly diagnosed cases of diabetes in children under the age of 13 years seen at the Royal Hospital for Sick Children, Glasgow, in the past 44 years are given below, grouped into periods of four years.

1933-6:	27	1957-60:	39
1937-40:	41	1961-4:	62
1941-4:	17	1965-8:	48
1945-8:	25	1969-72:	66
1949-52:	19	1973-6:	113
1953-6:	25		

It is known that the mortality (and possibly the morbidity) from diabetes falls in time of war.[2] Our figures show that the morbidity in children certainly fell in the Glasgow area during the second world war and incidence did not return to the prewar level until the late 1950s. The incidence increased in the 1960s and exploded in the middle 1970s, so that we are now seeing about five times as many new cases as were seen 20-30 years ago.

Spot checks were made at two other hospitals. Stobhill Hospital in Glasgow is a large general hospital with a children's unit. In 1953-6 10 new cases were seen. In 1960-3 there were 12 and in 1973-6 20. Glasgow Royal Infirmary is a large general hospital without a children's unit. In the period 1961-4 2 new cases in children under the age of 13 were seen and in 1973-6 there were 12.

The rise in incidence at the two latter hospitals indicates that the rise in incidence at RHSC is not due to a change in fashion diverting children to that hospital but represents a true increase of morbidity in the population. Our figures apply only to Glasgow, but conversations with colleagues suggest that they may be relevant to the country as a whole. Should that be so practitioners would be well advised to consider the possibility of childhood diabetes if there is a history of polyuria and polydipsia, a history which can always be elicited in childhood diabetes if the right questions are asked.

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1 Bloom, A, Hayes, T M, and Gamble, D R, *British Medical Journal*, 1975, **3**, 580.
2 Himsworth, H P, *Lancet*, 1949, **1**, 465.

Salbutamol-induced acidosis in pregnant diabetics

SIR,—With the increasing use of beta-sympathomimetics (in particular salbutamol) in the interruption of threatened premature labour side effects are becoming apparent. In particular the administration of intravenous salbutamol to pregnant diabetic patients can be associated with the development of significant metabolic acidosis.

I have observed two diabetic women in premature labour at 33 weeks and receiving salbutamol infusion who developed severe metabolic acidosis. Qualitative tests for serum ketones were negative in each case. Betamethasone was given in conjunction with salbutamol to these women to promote fetal lung maturity. Two similar cases were reported recently and the acidosis attributed to the betamethasone.[1]

In order to elucidate further the metabolic effects of salbutamol eight normal non-diabetic patients in premature labour and receiving the