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. Evans³ 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 Clark⁷ 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 intragastric 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 childhood¹ 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