Correspondence

Fetal blood gas analysis at delivery

Sir,

In a recent article D'Souza *et al.*¹ note that it is simpler and more convenient to obtain umbilical venous than arterial blood to assess the acid base status of a newborn infant at delivery. They add, however, that sampling arterial blood would be preferable to venous blood as the former reflects more closely the metabolic condition of the fetus. This clinical dilemma can be resolved very easily by sampling blood from the fetal arteries that cross the chorionic plate of the placenta. As in the retina of the eye the arteries can be accurately identified as they cross over the veins.

For a number of years we have obtained fetal arterial blood in this manner to determine the degree of metabolic acidosis in infants born depressed or after an obstetric diagnosis of fetal distress. The method is easy, especially if the fetal blood is not permitted to drain before delivery of the placenta.

The measurement of base excess or buffer base is preferred to pH as the latter is rapidly altered by changes in maternal PCO_2 . For example, the presence of hypocarbia provides the explanation of the finding of an alkalosis in many of the infants studied by D'Souza *et al.* Lastly it remains debatable whether a maternal metabolic acidosis influences the acid base status of the infant at birth.²

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Drs D'Souza and Richards comment:

Obtaining a sample of blood from fetal arteries crossing the chorionic surface of the placenta may be feasible but we are concerned that this blood is less likely to reflect the pH, PO_2 , and PCO_2 in the infant at birth, since there is a delay in the delivery of the placenta. Several minutes generally lapse between birth of the infant and delivery of the placenta during which time fetal blood will not be circulating in the placenta after the cord is clamped. This raises some uncertainty about the relevance of pH, PO_2 , and PCO_2 measurements in fetal blood from the placenta.

In our study base excess was calculated from blood pH, PCO_2 , and haemoglobin. There is no way that base excess can be measured, it can only be calculated from pH, PCO_2 , and haemoglobin. One can either use the Siggard-Andersen alignment nomogram³ or one of the several equations, for example Siggard-Andersen⁴; both these are incorporated in the Radiometer slide rule⁵ and microcomputer chips incorporated into many modern blood gas machines (for example, Corning)⁶ but all demand a knowledge of the haemoglobin value. To follow Dr Woods's path necessitates measuring the 3 parameters,

especially haemoglobin, and measuring them quickly. Additional information obtained by calculating base excess is useful in that buffer base is altered depending on whether there is a metabolic acidosis, as in accumulation of lactate in the fetus, or a non-metabolic acidosis resulting from carbon dioxide retention.

Blood pH and gas tensions were not measured in our mothers during labour but we have referred to the results of other studies in which such measurements have been carried out. In newborn infants we found that blood pH, PO_2 , and PCO_2 measurements can be conveniently carried out when Astrup apparatus is available on the delivery unit. Lactate concentrations are another indicator of asphyxia at birth, but lactate measurements are less convenient since it is necessary to send blood specimens to the laboratory. Delay in obtaining results would reduce the usefulness of such measurements in the diagnosis and management of asphyxia at birth.

References

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Ventricular enlargement after periventricular haemorrhage

Sir,

Palmer *et al.*¹ have shown that in 25 preterm infants surviving periventricular haemorrhage, cerebral ventricular enlargement was significantly associated with a poorer outcome during the first year of life and that this effect was irrespective of the size of the initial haemorrhage. I have followed up 67 very low birthweight infants born in 1980 and 1981 who survived periventricular haemorrhage. Twenty eight showed ventricular enlargement, with ventricular width exceeding 5 mm at the level of the lateral sulcus on a coronal ultrasound scan via the anterior fontanelle. Follow up examination using a neurological examination, Denver developmental screening test, and Stycar tests of hearing and vision showed 26 infants to have a neurological deficit or developmental delay, or both at 18 months to 3 years. Eight of these infants have a ventriculo-peritoneal shunt inserted for progressive hydrocephalus.

Table Proportion of infants abnormal at follow up

Degree of periventricular haemorrhage	Subependymal haemorrhage	Intraventricular haemorrhage (No VPS)	Parenchymal extension (No VPS)	All	Subependymal/intraventricular haemorrhage only
Ventricular enlargement	1/3	4/9 (1)	14/16 (7)	19/28*	5/12†
No ventricular enlargement	3/28	3/10	1/1	7/39*	6/38†

* $\chi^2 = 15$, $P < 0.001$; † $\chi^2 = 2.2$, $P > 0.1$. VPS = ventriculo-peritoneal shunt.

Abnormal outcome is strongly associated with ventricular enlargement ($P < 0.001$). However, ventricular enlargement is also strongly associated with the degree of haemorrhage, in particular the presence of parenchymal extension of a bleed. Twelve of 50 babies with subependymal haemorrhage or simple intraventricular haemorrhage, and 16 of 17 with parenchymal extension of intraventricular haemorrhage (PAR H) had ventricular enlargement. Only 2 babies with PAR H appeared normal at follow up. It seems that the association of ventricular enlargement with poor outcome in this study is largely due to the bias introduced by the poor outcome of babies with large haemorrhages. If infants with subependymal or intraventricular haemorrhage alone are considered the relation between ventricular enlargement and outcome is no longer statistically significant. A similar bias is likely to have occurred in the study of Palmer *et al.* in that 12 of 14 babies without ventricular enlargement had small bleeds, while only 1 of 10 babies in the ventricular enlargement group had small bleeds ($P < 0.005$). Until larger numbers are available it will not be possible to allow for the effect of bleed size in analysis of the effect of ventricular enlargement on subsequent outcome. It is also not possible to conclude that ventricular enlargement has a greater effect than bleed size on subsequent development. This has implications for the rationale of conducting a trial of early intervention by cerebrospinal fluid drainage in posthaemorrhagic ventricular enlargement.

Reference

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Plasma terbutaline levels in asthma

Sir,

The reported safety of oral terbutaline in a dose of 250 $\mu\text{g/kg}$ in children prompts me to comment.¹ As a regular prescriber of this drug I have found a marked individual variation in response. Doses as high as 140 $\mu\text{g/kg}$ have caused tremor severe enough to render writing impossible and recently a dose of 330 $\mu\text{g/kg}$ (7.5 mg) has been reported to cause seizures.^{2 3}

It should be noted by your readers that Dinwiddie *et al.*

studied only 5 patients.¹ In a larger and more comprehensive study it has been shown that doses in excess of 100 $\mu\text{g/kg}$ produce side effects with no improvement in pulmonary function.²

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Dr Dinwiddie comments:

Our clinical experience of oral terbutaline, often given in the higher dose in children, is at variance with Dr Blumenthal's. We have found that children are generally more tolerant of this drug and that higher proportionate doses are often required to produce a therapeutic effect. It was this experience that prompted us to undertake the present study.

We agree that there is individual variation with this as with any drug and the dose must always be tailored to the patient and his response to treatment. Our small study does, however, show that children may be given doses of up to 250 $\mu\text{g/kg}$ with good response and usually no untoward side effects. We should point out, however, that the maximum dose of 5 mg as stated in our paper should not be exceeded.

Intracranial calcification and childhood medulloblastoma

Sir,

We read with interest the paper by Pearson *et al.*¹ on intracranial calcification in survivors of childhood medulloblastoma. They observed intracranial calcification in 3 of 5 survivors of medulloblastoma who had undergone surgery and radiotherapy but had not had chemotherapy.

We would like to report a boy, now aged 16 years, who developed intracranial calcification following cranial irradiation and partial surgical removal of a cerebellar