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Umbilical cord blood gas analysis at delivery: a time for quality data

JENNIFER WESTGATE *Lecturer/Honorary Senior Registrar*, JONATHAN M. GARIBALDI *Research Assistant*, KEITH R. GREENE *Consultant/Honorary Senior Lecturer*

Perinatal Research Group, Postgraduate Medical School, Department of Obstetrics, Derriford Hospital, Plymouth

ABSTRACT

Objectives To address the practical problems of routine umbilical cord blood sampling, to determine the ranges for pH, pCO_2 and base deficit and to examine the relationships of these parameters between cord vessels.

Design An observational study of umbilical cord artery and vein blood gas results.

Setting A large district general hospital in the UK.

Subjects One thousand nine hundred and forty-two cord results from 2013 consecutive pregnancies of 34 weeks or more gestation, monitored by fetal scalp electrode during labour.

Results Only 1448 (74·6%) of the 1942 supposedly paired samples had validated pH and pCO_2 data both from an artery and the vein; 54 (2·8%) had only one blood sample available, 90 (4·6%) had an error in the pH or pCO_2 of one vessel and in 350 (18%) pairs the differences between vessels indicated that they were not sampled from artery and vein as intended. Only 60% of the cases with an arterial pH less than 7·05 had evidence of a metabolic acidosis (base deficit in the extracellular fluid 10 mmol/l or more). Of all the cases, 2·5% had a venous–arterial pH difference greater than 0·22 units.

Conclusions Both artery and vein cord samples must be taken and the results screened to ensure separate vessels have been sampled. Interpretation of the results requires the examination of pCO_2 and base deficit of the extracellular fluid from each vessel as well as the pH. Confusion about the value of cord gas measurements may be due to the use of erroneous data and inadequate definitions of acidosis which do not differentiate between respiratory and metabolic components.

The 26th Royal College of Obstetricians and Gynaecologists' Study Group on Intrapartum Fetal Surveillance (1993) has recommended measurement of the acid–base status of the umbilical artery and vein cord blood after delivery as "a measure of the fetal response to labour". The cord vein carries oxygenated blood to the fetus whilst the two smaller arteries carry deoxygenated blood from the fetus to the placenta. Cord arterial blood normally reflects fetal acid–base balance, hence has a lower pH and pO_2 and a higher pCO_2 than the venous blood which reflects a combination of maternal acid–base status and placental function. Despite the College recommendation and the relative ease of the procedure, its value is still debated. Although cord acid–base assessment provides an objective measure of neonatal condition at delivery (Sykes *et al.* 1982, 1983; Low 1988; Goldaber & Gilstrap 1993), there is a lack of correlation with other measures of neonatal condition (Apgar scores, resuscitation, neonatal morbidity) and long-term outcome in some studies (Dijxhoorn *et al.* 1985; Ruth & Raivio 1988; Dennis *et al.* 1989) but not in others (Low 1988; Gilstrap *et al.* 1989; Goldaber *et al.* 1991).

In addition, there is no consensus definition of acidosis and the values of pH proposed to define acidosis range from 7·20 (Wible *et al.* 1982; Page *et al.* 1986) down to 7·00 (American Academy of Pediatrics 1986). Most studies refer to arterial pH values but some have used venous values because of reported difficulty in obtaining samples from the artery (Huisjes & Aarnoudse 1979), while others did not specify which vessel was used (Halligan *et al.* 1992). In addition, only a few studies have attempted to separate respiratory and metabolic causes of acidosis (Low *et al.* 1978; Sykes *et al.* 1982), despite the important and different pathophysiological implications of each.

The purpose of this paper is to resolve some of this confusion by reporting a detailed study of blood gas data

Correspondence: Mr K. R. Greene, Perinatal Research Group, Level 7, Maternity Department, Derriford Hospital, Plymouth, PL6 5DH, UK.

collected from 1942 labours. The aims of this paper are firstly, to address the specific practical problems related to routine umbilical cord sampling and to assess the reliability of the results, secondly, to determine the ranges for pH, PCO_2 and base deficit, and thirdly, to examine the relationships between pH and base deficit and between arterial and venous blood gas values.

Subjects and methods

During an 18-month period (June 1990 to December 1991) umbilical cord artery and vein blood samples were taken from all pregnancies of 34 weeks or more gestation who were monitored with a fetal scalp electrode. In the initial stages samples were collected by research personnel but after 100 cases had been collected samples were taken by midwives and nursing auxiliaries. The clinical staff were trained by researchers before the study and the training was regularly maintained for new personnel. All data analysis was performed after the completion of the trial.

A segment of cord (minimum recommended length 10 cm) was double clamped immediately after delivery and before the first breath, excised, placed into a kidney dish and passed out of the delivery room for sampling. Blood was taken first from the artery and then from the vein using preheparinised syringes. The syringes were identified by always taking a larger quantity of blood from the vein and the samples then placed through the analyser; artery first, then vein. Sampling within 10 min of birth was recommended. If this was not possible the cord was placed on an ice pack until the samples could be withdrawn. Analysis was usually achieved within 30 min of delivery; in no case was the delay longer than 40 min. Prior to the study, a serial analysis of 25 paired cord specimens, sampled over a 60-min period, established that no significant changes in cord blood gas parameters occurred during this time.

Blood will not clot in the cord, but will in the syringe or analyser unless heparinised. Therefore syringes were preheparinised on-site by adding one drop of liquid heparin (1:5000 units) from a 1 ml tuberculin syringe into a 2 ml plastic syringe, moving the plunger up and down and expelling any residual heparin before capping with a 21 gauge needle. All syringes were prepared by research personnel who were careful not to use more than one drop of heparin, as changes in PCO_2 and pH can occur if heparin makes up more than 10% of the volume of any blood gas sample (Duerbeck *et al.* 1992). Results from non-heparinised and heparinised syringes were compared in 30 cases before the study and no difference in results was found.

A Corning 178 blood gas analyser (Ciba Corning Diagnostics, Ltd) was used throughout the study. The machine is self-calibrating (one point calibrations every 30 min and two point calibrations every 2 hours). During analysis, a reading is taken from each electrode (pH, PO_2 and PCO_2) twice a second for a maximum of $2\frac{1}{2}$ min, or until 30 consecutive stable readings have been obtained. If stable readings cannot be obtained, two asterisks (**) are printed beside the value to indicate an unreliable result (Ciba Corning Diagnostics Ltd). The analyser was serviced daily by Medical Physics Department technicians. The analyser measurement error was checked by analysing 110 pairs of samples, drawn from 35 cords, and noting the difference in the two readings in each case. The standard deviations of errors of the pH, PCO_2 and PO_2 were consistent with the measurement errors reported in the CIBA Corning manual; pH 0·002 units, PCO_2 0·08 kPa, PO_2 0·05 kPa.

The Corning analyser provided base deficit values calculated from the blood compartment (BD_{blood}). It is more correct to use the base deficit calculated from the whole extracellular fluid compartment (BD_{ecf}) in the perinatal period as this value is less susceptible to the influence of high PCO_2 levels (Siggaard-Andersen 1971; Rooth 1988; Rosén & Murphy 1991). During the trial BD_{ecf} was calculated manually from pH and PCO_2 using a Siggaard-Andersen Acid Base Chart (Siggaard-Andersen 1971) but once the results were entered into a database, a formula was used automatically to calculate BD_{ecf} from pH and PCO_2 (Siggaard-Andersen 1976):

$$BD_{ecf} = -(1-0{\cdot}023Hb)(HCO_3^- - 24{\cdot}1 + (2{\cdot}30Hb + 7{\cdot}7)(pH - 7{\cdot}40))$$

where:

$$HCO_3^- = 0{\cdot}23PCO_2 \log^{-1}(pH - 6{\cdot}10) \text{ mmol/l}$$
$$\text{and} \quad Hb = 3{\cdot}7 \text{ mmol/l}.$$

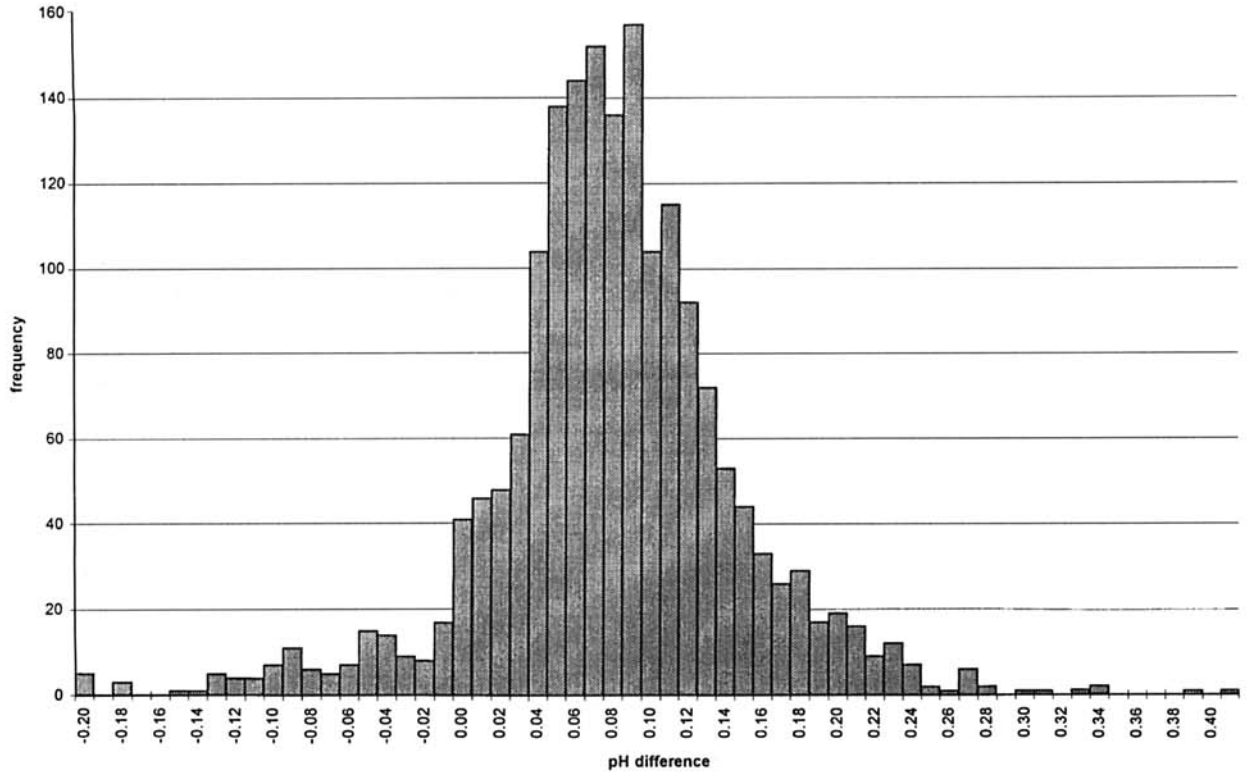
Results

Cord blood gas results were taken from 1942 of 2013 deliveries (96·5%) during the study period, with staff forgetting or being unable to obtain samples in the remaining 71 (3·5%). A single blood sample only was obtained in 54 cases (2·7%) and in another 73 cases (3·6%) an unreliable pH (as indicated by the blood gas analyser) was obtained from one or both vessels. This left 1815 paired results with a reliable pH in each sample. Of these, the blood gas analyser identified 17 unreliable PCO_2 readings (0·9%), leaving 1798 pairs of results with apparently accurate pH and PCO_2 values. Similarly there were 64 unreliable PO_2 readings (3·5%), leaving 1751 pairs of results with apparently accurate pH and PO_2 values.

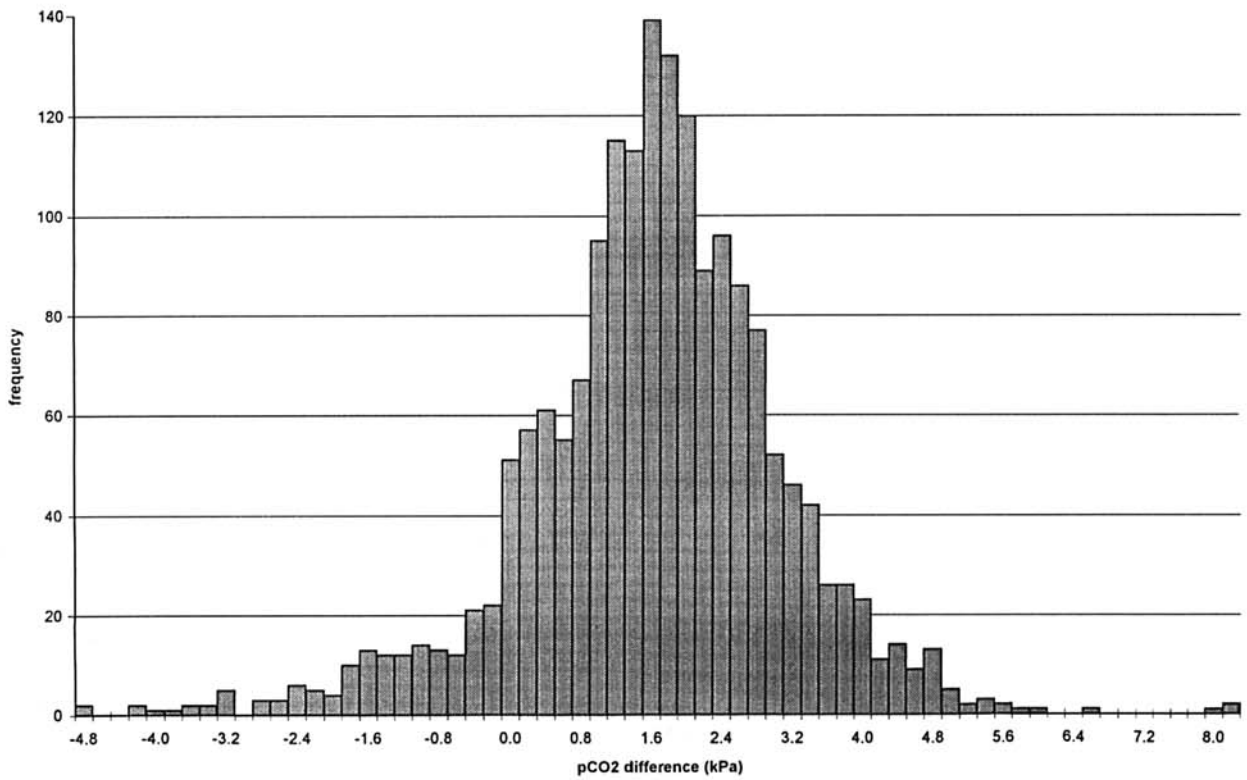
The venous–arterial differences for pH and PO_2 and the arterial–venous differences for PCO_2 for these results were examined in the frequency histograms shown in Fig. 1. There were a number of paired samples in which the differences were zero, very small, or opposite to the expected values (i.e. arterial pH/PO_2 higher than venous pH/PO_2, arterial PCO_2 lower than venous PCO_2). These results cannot be explained physiologically and the most likely causes were thought to be:

1. Inadvertent sampling from the same vessel twice (i.e. artery–artery or vein–vein instead of artery–vein); or
2. Transposing the vessels either when taking the samples or on introduction into the analyser (i.e. vein–artery instead of artery–vein).

The former would have caused a normal distribution of differences centred at zero (with a width governed by the size of errors of the parameter) and the latter would have caused a mirror-image of the real arterial–venous dis-



(a)



(b)

Fig. 1(a, b). For legend see facing page.

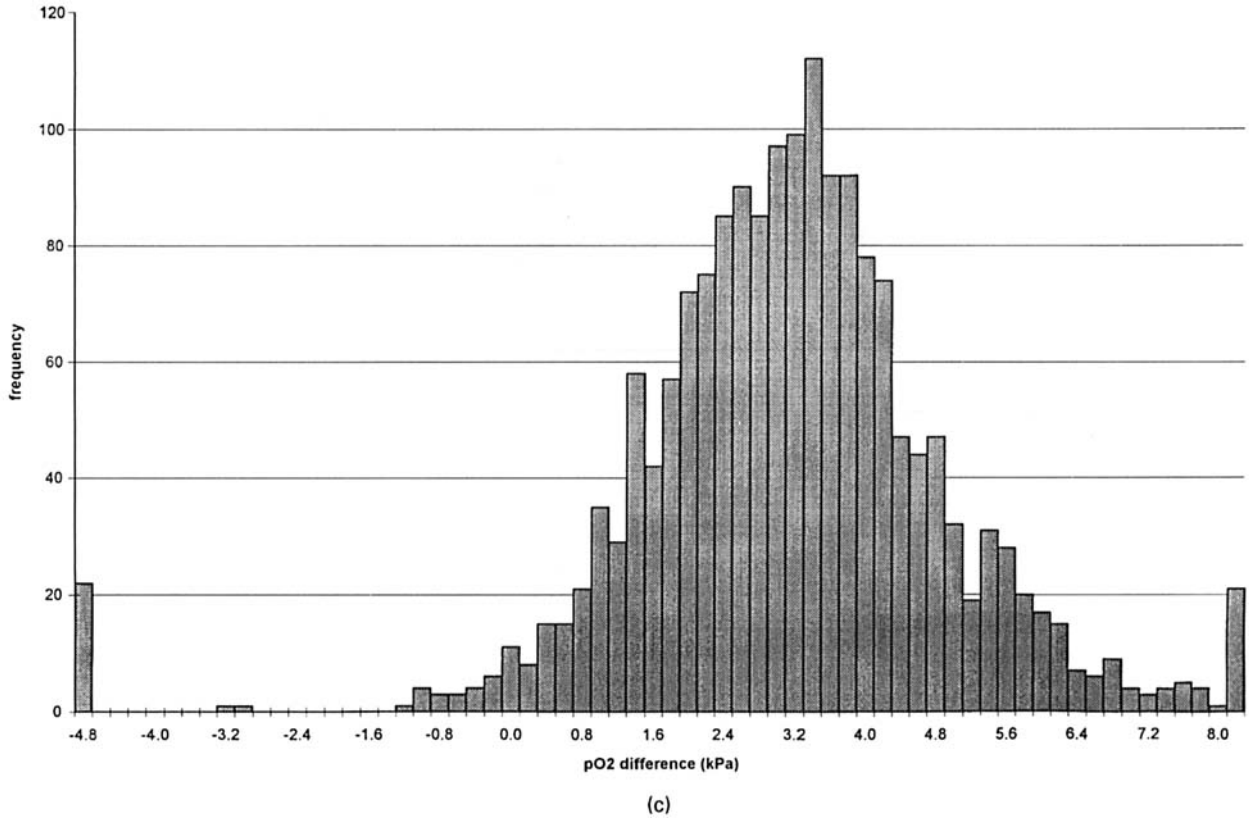


Fig. 1. Frequency distributions of: (a) venous–arterial pH differences in 1815 paired samples; (b) arterial–venous PCO_2 differences in 1798 paired samples; and (c) venous–arterial PO_2 differences in 1751 paired samples.

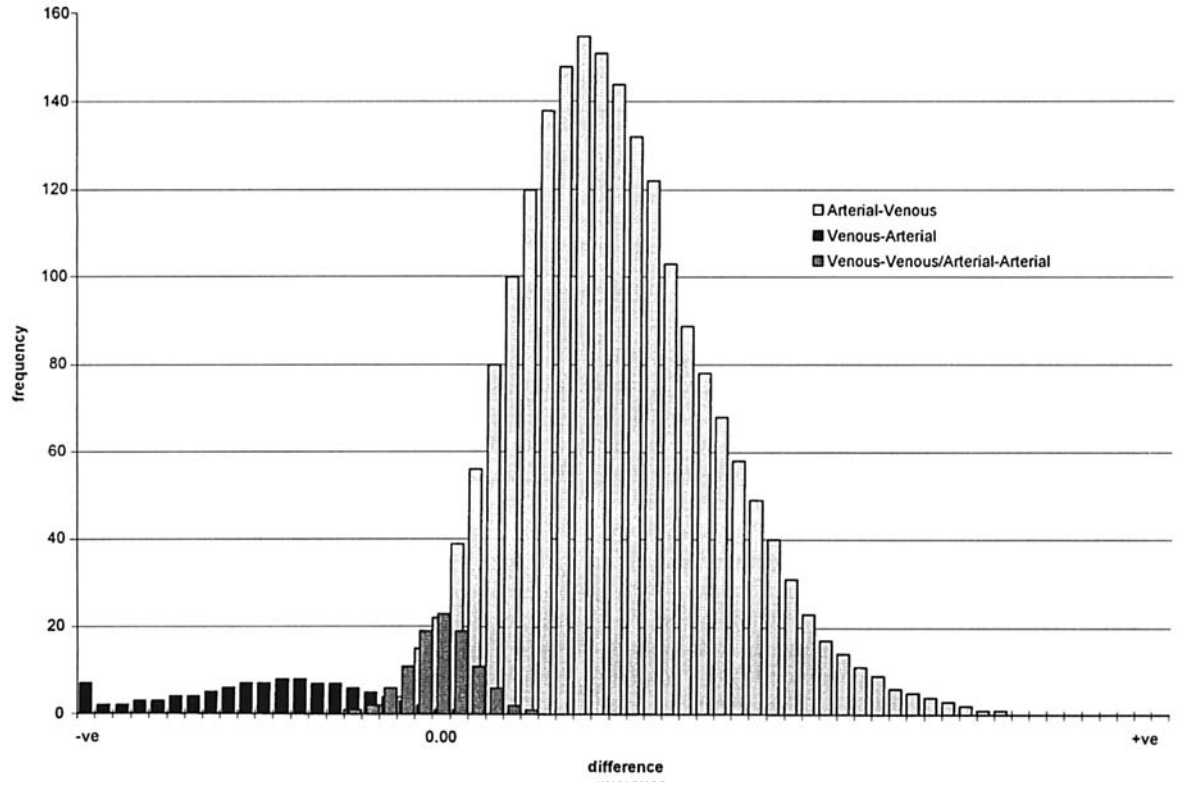


Fig. 2. Hypothetical distributions of differences in double samples.

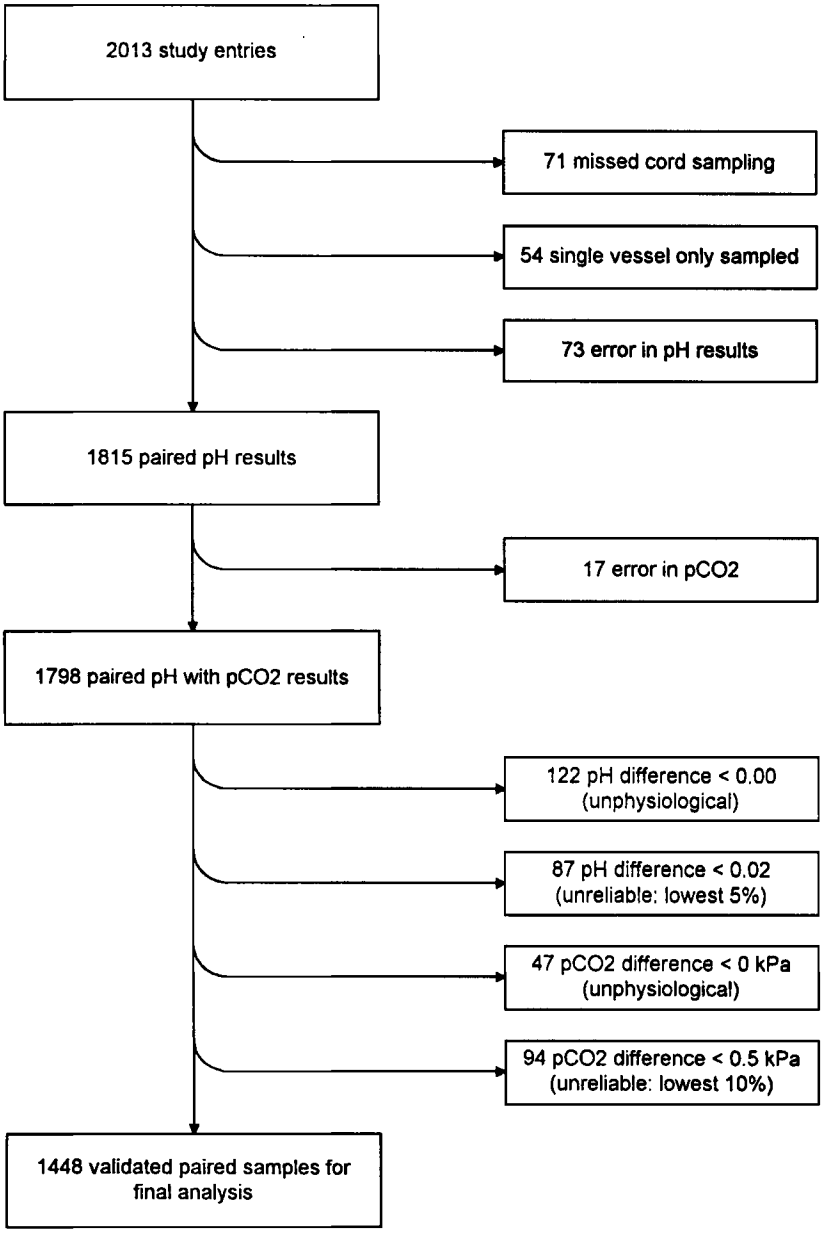


Fig. 3. A summary of the exclusions made to identify reliable paired arterial and venous cord samples.

tribution, reflected about zero. If, as seems probable, both occurred in some small proportion during the study, three distributions would result as illustrated in Fig. 2 which, summated, would form the actual distributions of Fig. 1. It is not possible, retrospectively, to separate each parameter's distribution into its three component parts and so rather than include possibly erroneous data, we sought to exclude such results from the further analysis. All results with negative pH or PCO_2 differences were deemed unphysiological (due to transposition) and rejected. Results with pH differences below the 5th centile (less than 0·02) or PCO_2 differences below the 10th centile (less than 0·5 kPa) were also excluded. The intention was to remove the results sampled from the same vessel, allowing for possible experimental error. The PCO_2 cut-off was higher because its measurement was less precise. Only the pH and PCO_2 differences were examined for exclusion purposes, as only these affect the BD_{ecf}.

Of the 1798 samples with paired pH and PCO_2 results, 209 (11·6%) had a venous–arterial pH difference less than 0·02 and 141 (7·8%) had a pH difference of 0·02 or more but a PCO_2 difference less than 0·5 kPa. These were therefore excluded from further analysis leaving 1448 validated pairs of results. A summary of the exclusions made is shown in Fig. 3. The frequency distributions for pH, PCO_2 and BD_{ecf} in the 1448 validated sample pairs are shown in Fig. 4. These frequency distributions are skewed and therefore statistical description of the results of the raw and validated data was made using median and centile values (Tables 1 and 2). The venous–arterial pH difference ranged from 0·02 to 0·49 units with a median of 0·09, the median arterial–venous PCO_2 difference was 1·9 kPa (range

Table 1. The median with 2·5th to 97·5th centile range in parentheses for cord artery and vein pH, PCO_2 and BD_{ecf} (raw data).

	Artery (n = 1798)		Vein (n = 1798)	
pH	7·27	(7·06 to 7·41)	7·35	(7·14 to 7·47)
PCO_2 (kPa) (mmol/l)	7·1	(4·2 to 10·6)	5·4	(3·6 to 8·9)
BD_{ecf}	2·7	(−2·4 to 11·0)	3·0	(−1·3 to 9·0)

Table 2. The median with 2·5th to 97·5th centiles range in parentheses for cord artery and vein pH, PCO_2 and BD_{ecf} (after exclusions as described).

	Artery (n = 1448)		Vein (n = 1448)	
pH	7·26	(7·05 to 7·38)	7·35	(7·17 to 7·48)
PCO_2 (kPa) (mmol/l)	7·3	(4·9 to 10·7)	5·3	(3·5 to 7·9)
BD_{ecf}	2·4	(−2·5 to 9·7)	3·0	(−1·0 to 8·9)

0·5 to 9·9 kPa) and the median arterial–venous BD_{ecf} difference was −0·8 mmol/l (range −11·8 to 9·7 mmol/l).

The relation between respiratory and metabolic components of acidosis was examined in a scatter diagram of arterial pH and BD_{ecf} (Fig. 5). The 2·5th centile for pH (7·05) and the 97·5th centile for BD_{ecf} (10 mmol/l) from Table 2 are indicated. It can be seen that 40% (14 of 35) of the cases with a pH less than 7·05 had a base deficit less than 10 mmol/l—a level associated with a mainly respiratory rather than metabolic acidosis.

The majority of cases with BD_{ecf} of 10 mmol/l or more also had a pH less than 7·05 but not exclusively so. Nine cases had normal arterial pHs (between 7·12 and 7·22), but had high BD_{ecf} values (10·0 to 14·1 mmol/l). A possible explanation for these results is the simultaneous occurrence of maternal hyperventilation (producing hypocarbia) and fetal hypoxia (producing a metabolic acidosis). Knowledge of maternal acid–base status at delivery would have been helpful in these cases but maternal blood was not taken. However, there was some evidence for fetal hypoxaemia; five cases had operative deliveries for an abnormal fetal heart rate pattern, three had a persistent bradycardia of eight to 10 min duration prior to delivery and in one case the fetal heart rate was not recorded for the last 15 min before delivery.

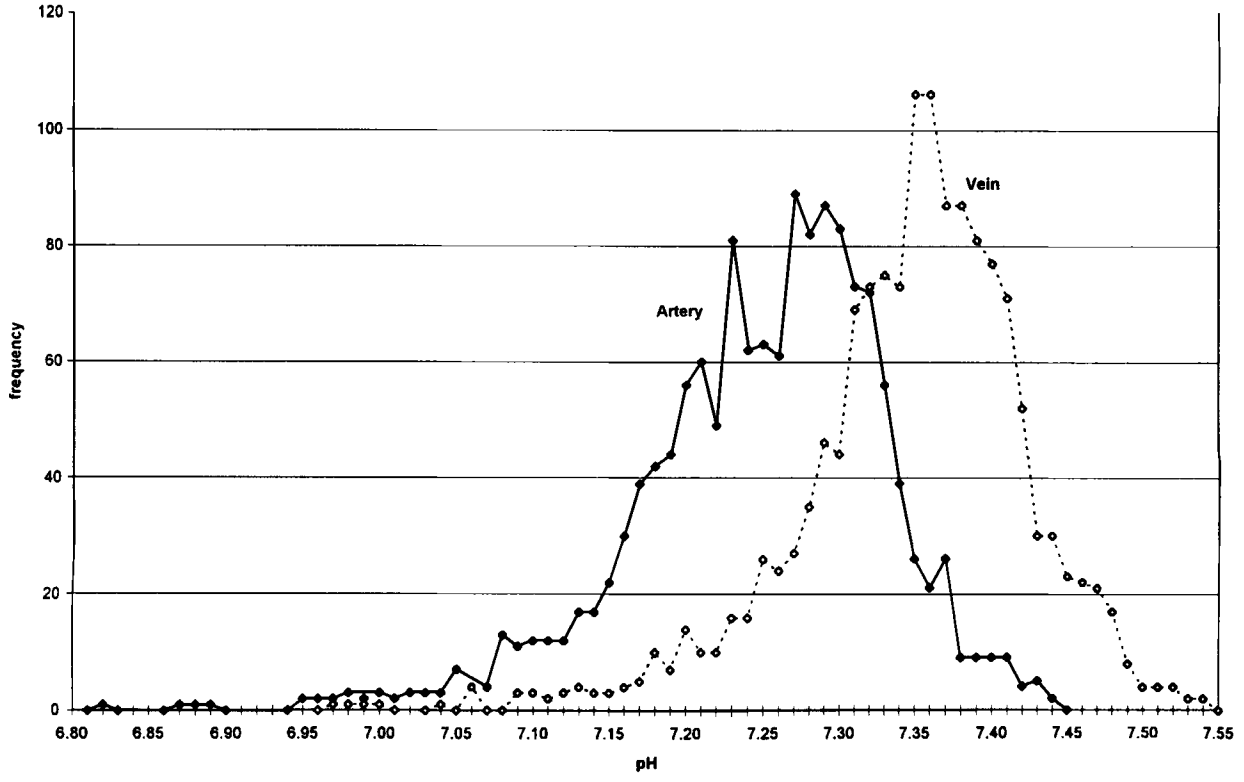
Discussion

This paper highlights several important aspects of cord blood gas analysis. The most critical is the necessity to take samples from both the artery and vein and to screen the results to ensure that separate vessels have been sampled. When this was done, approximately 25% of all paired samples in this study were rejected. Few studies have reported error-checking their results and those that did reported similar percentages of samples rejected (Huisjes & Aarnoudse 1979; Eskes *et al.* 1983; Yudkin *et al.* 1987). Many studies involving cord acid–base measures only report collecting arterial blood, which would have made error checking impossible (e.g. Steer *et al.*, 1989; Ruth & Raivio 1989; Goldaber *et al.* 1991). Neither midwifery staff nor doctors were very good at noticing sampling errors in our hospital, although this was the first time that cord sampling had been performed and error rates have decreased to 15% since then. Others have reported 10% error rates with routine sampling by midwives (Riley & Johnson 1993). We are investigating ways of improving this still further. The importance of adequate Blood Gas Analyser maintenance in obtaining accurate results also needs to be emphasised.

There is very little information on the normal range of differences between vessels. The minimum venous–arterial pH difference used by Rooth (1988) in his review of four studies was 0·04. Huisjes & Aarnoudse (1979) chose 0·03 units as a minimum allowable pH difference while Eskes *et al.* (1983) used 0·02 units, but neither group provided support for the levels chosen. One group even accepted negative venous–arterial pH differences (Egan *et al.* 1992). It has been a surprise to find that values for minimum differences have not yet been established and the most recent review of cord sampling managed to discuss sample collection and analysis without addressing this problem (Riley & Johnson 1993). The exclusion of results with small or negative differences had a minimal effect on the population statistics of Tables 1 and 2 because of the large study numbers. Despite this, such results should be excluded as, in the individual case, they are improbable physiologically and are most likely due to errors in sampling.

It is important to realise that very large arterial–venous differences can exist and that a normal venous result does not exclude the possibility of a significant arterial acidosis. Large arterial–venous BD_{ecf} differences are usually the result of cord entanglement (Johnson *et al.* 1990; Rosén & Murphy 1991) or a stasis of umbilical cord flow secondary to cardiac failure (Brar *et al.* 1988). The most notable example in our study had severe variable decelerations followed by a prolonged terminal bradycardia in the second stage as a result of a tight nuchal cord and unfortunately died (artery pH 6·88, BD_{ecf} 16 mmol/l, vein pH 7·38, BD_{ecf} 4 mmol/l). The relationship between cord gas values and outcome would not have been very obvious if a venous sample only had been obtained.

Most cases of fetal acidosis during labour are acute in onset (Johnson *et al.* 1990). With acute fetal hypoxaemia, there may not be enough time for fetal and placental blood to equilibrate before delivery and acids produced by the fetus may not be removed across the placenta. This will be particularly so for nonvolatile acids like lactic acid, for their placental transfer is much slower than for the volatile carbonic acid. As a result it will take some time for the placental extracellular fluid compartment to become saturated with lactic acid from the fetus. It is probable that large arterial–venous BD_{ecf} differences (high arterial BD_{ecf}, low venous BD_{ecf}) reflect an acute onset of fetal metabolic acidosis. In contrast, if both the artery and vein have a high BD_{ecf}, the fetal acid load has saturated placental buffering capacity and equilibration has occurred so the acidosis is not acute. Examples of two cases which illustrate the importance of these differences are shown in Table 3.



(a)

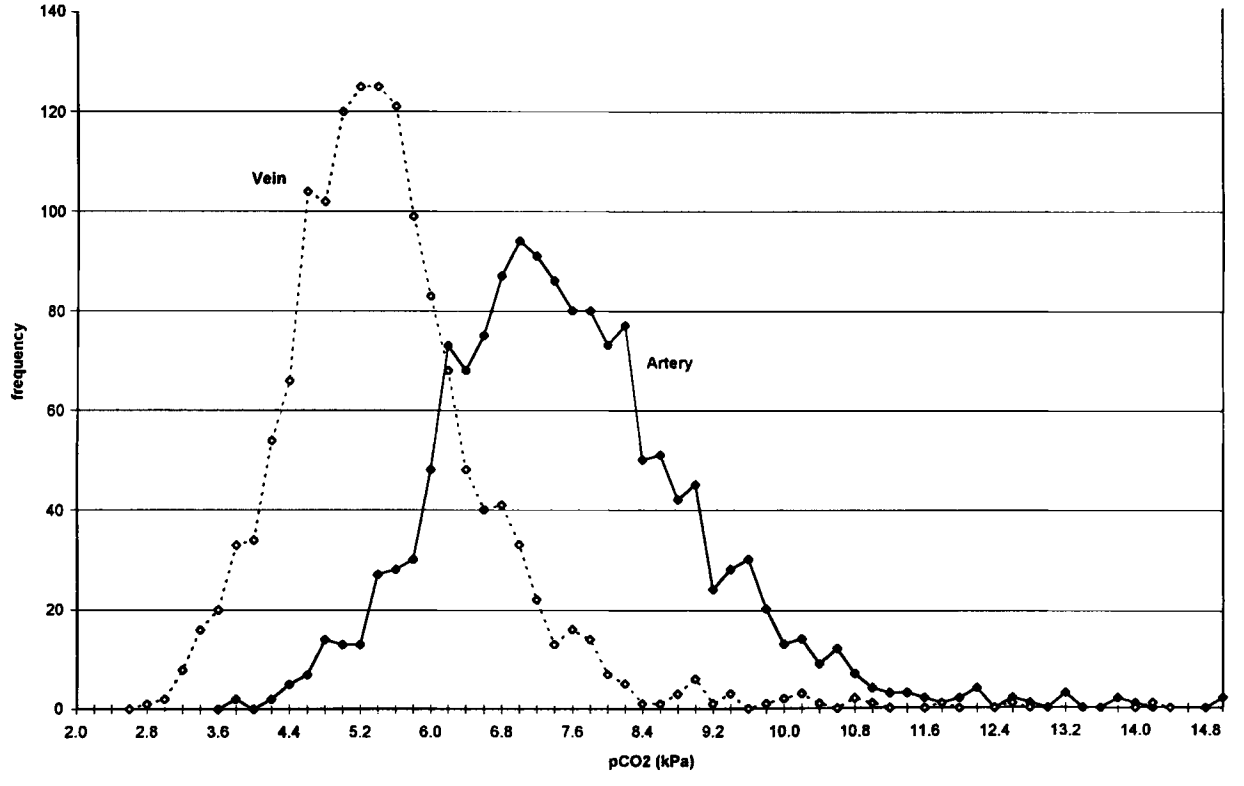


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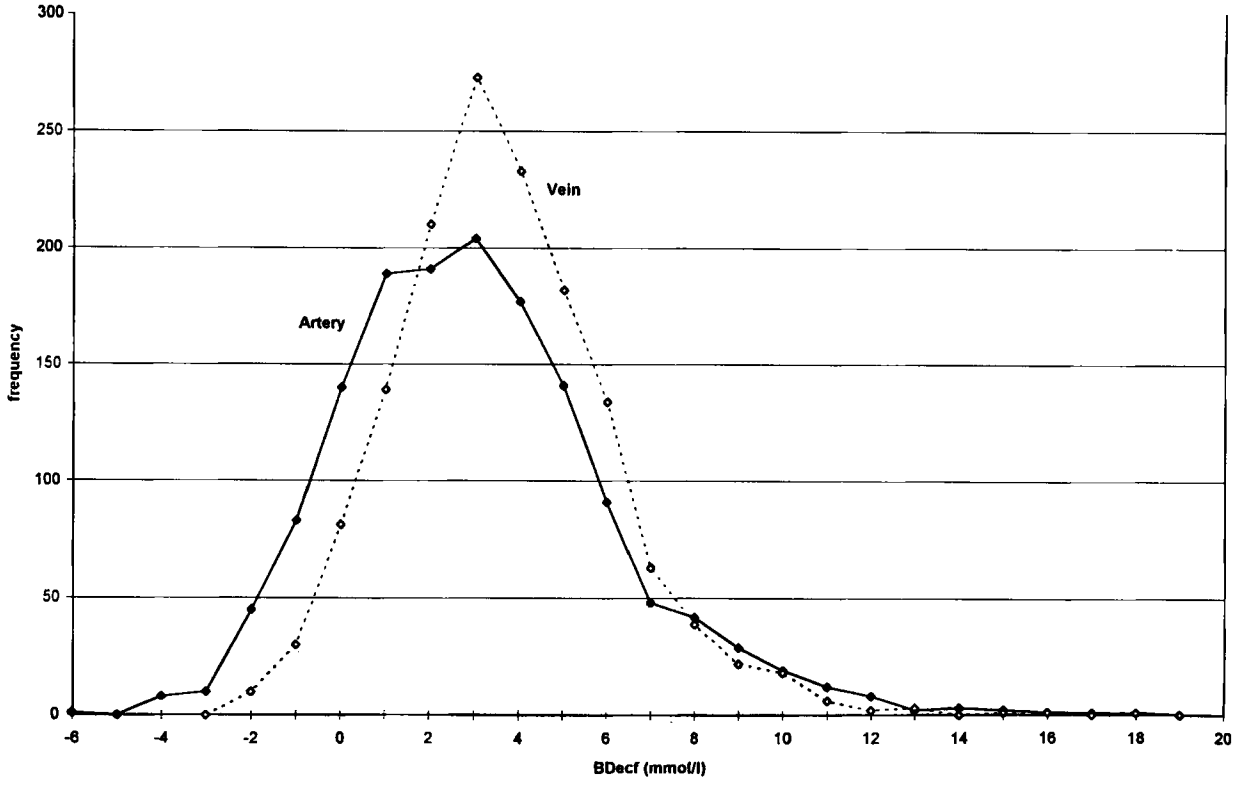
Fig. 4(a, b). For legend see facing page.



(c)

Fig. 4. Frequency distributions of cord artery and vein parameters in 1448 validated paired samples: (a) pH; (b) PCO_2; and (c) BD_{ecf}.

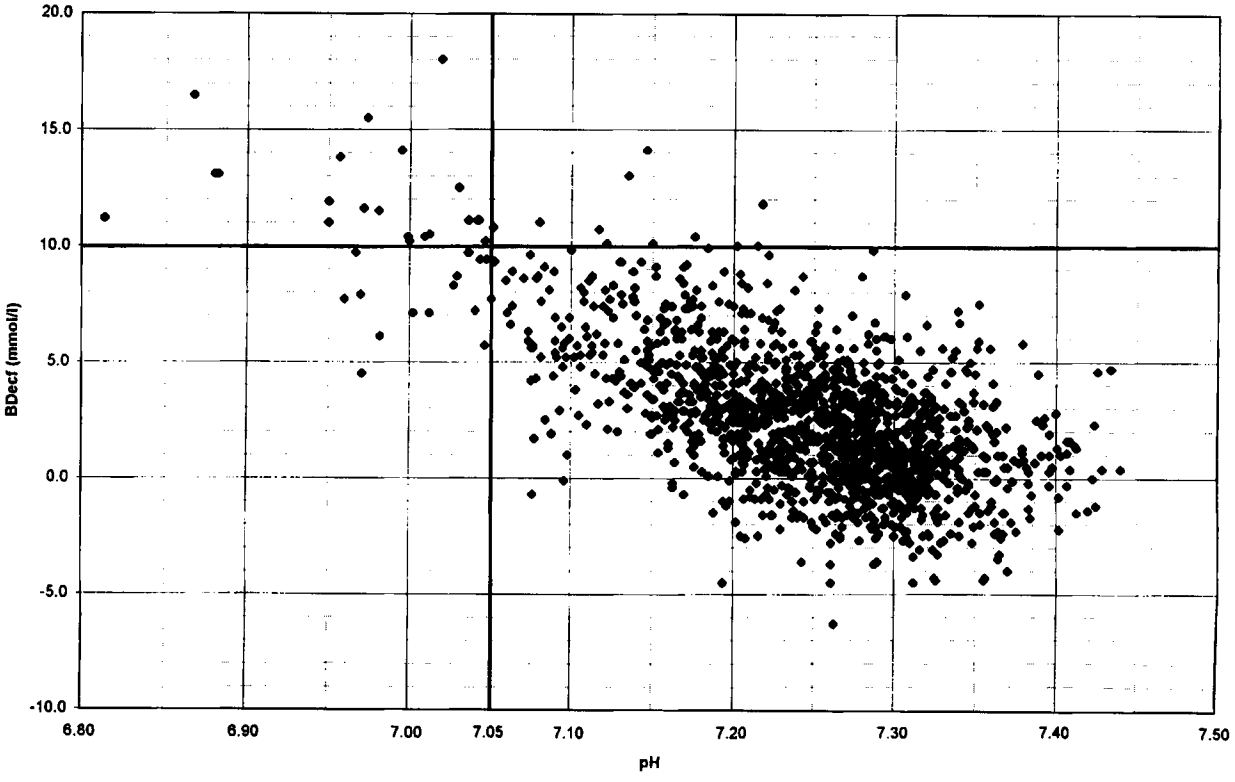


Fig. 5. Scatter diagram of cord artery pH and BD_{ecf} in 1448 validated paired samples.

Table 3. An example of two cases with similar arterial but different venous values. Case A required resuscitation at birth, was ventilated for 48 h and has cerebral palsy at one year of age. Case B had a 5-min Apgar score of 8 with no neonatal problems.

	Case A		Case B	
	Artery	Vein	Artery	Vein
pH	7·03	7·10	7·04	7·32
PCO_2 (kPa)	8·4	6·6	8·9	5·1
PO_2 (kPa)	0·9	2·6	1·8	4·5
BD_{ecf} (mmol/l)	12·5	12·6	11·2	5·5

The duration of metabolic acidosis is an important prognostic indicator (Low 1988). Differences in pH and BD_{ecf} can provide information about the time course of an acidosis and could more accurately identify babies at risk of neonatal complications. The cases shown in Table 3 illustrate the problem of using arterial results alone to predict outcome. As a result of discussions from this analysis of cord gas data, James Low has reworked his extensive database of paired cord blood samples and found that neonates with metabolic acidosis and narrow arterial–venous buffer base differences had a significantly poorer neonatal outcome than those with metabolic acidosis and large arterial–venous differences (Low *et al.* 1993). It is possible that analysis of a maternal venous or capillary blood sample at delivery may further aid interpretation of cord results in certain situations.

The definition of acidosis needs to be standardised. Statistically low (or high) levels should be defined by centile values rather than mean and standard deviation because the data are not normally distributed, but only Eskes *et al.* (1983) have done this. It does not necessarily follow that these statistically abnormal levels are physiologically significant. It is important to appreciate that the relationship between pH and $[H^+]$ is logarithmic and not linear. A 0·10 unit decrease in pH from 7·30 to 7·20 is associated with a rise in $[H^+]$ of 13 nmol/l, but a similar fall in pH from 7·00 to 6·90 increases $[H^+]$ twofold by 26 nmol/l. As the hydrogen ions are an important mechanism of tissue damage, there is a danger of failing to appreciate the physiological implications of low pHs. It is clear that the level of pH chosen to indicate acidosis in many studies was too high (Winkler *et al.* 1991) so, as might be expected, no strong relationship has been found between pH and neonatal outcome. However, it is also important to consider the aetiology of the acidosis. A respiratory acidosis caused by carbon dioxide accumulation is far less significant for the fetus and neonate than a metabolic acidosis which is caused by hypoxia during labour (Rooth 1988). In our study 40% of the cases with a low arterial pH (below 7·05) had a respiratory acidosis and would not be expected to have long-term sequelae unless hypoxia ensued in the neonatal period. Unless this distinction is made, the relationship between acidosis and outcome will also be blurred. There is increasing evidence from larger studies that acidosis is not significant until levels of pH below 7·05 and BD_{ecf} of 12 mmol/l or more are reached (Low *et al.* 1984; Gilstrap *et al.* 1989; Goldaber *et al.* 1991). These figures are close to those determined in this study on statistical grounds.

Paired cord blood gas analysis provides an objective measure of neonatal condition at delivery which can be used as an objective measure for the audit of intrapartum care (Eskes *et al.* 1983; Yudkin *et al.* 1987; Richards & Johnson 1993). It should encourage a physiological approach to the interpretation of cardiotocographs and fetal blood sampling, which is useful in training and education and may also protect against allegations of intrapartum asphyxia (Thorp *et al.* 1989; Gregg & Weiner 1993; Goldaber & Gilstrap 1993). But should cord sampling be routine (Thorp *et al.* 1989) or selective (Page *et al.* 1986; Duerbeck *et al.* 1992)? In our study, 27 (56%) of the 48 babies born with an arterial pH less than 7·05 had neither a fetal blood sample nor an operative delivery and would not have been detected by selective sampling. If full value is to be obtained from cord blood gas analysis, sampling will have to be performed for all deliveries and checked to be from both vessels. Furthermore, PCO_2 must also be measured to derive the base deficit of the extracellular fluid. We have had no difficulty in introducing this as routine clinical practice.

We support the RCOG recommendations for cord artery and vein acid–base assessment, but our study shows that this will only be valuable if the samples are correctly taken, correctly measured and the results correctly interpreted. Much of the previous work, especially on cord blood gases and outcome, needs to be repeated with a validated database of cord blood gas values, or at least re-evaluated using only studies where appropriate methodology has been used.

Acknowledgments

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