

K2 Medical Systems

Umbilical Cord Blood Sampling and Expert DataCare

- Protection from obstetric litigation and audit tool for labour management
- Expert interpretation of cord blood gas analysis
- Validation of paired cord sample
- Permanent database of results

Introduction

Assessment of the acid-base status of umbilical cord blood at birth provides a measure of the fetal response to labour and was recommended by the 26th Royal College of Obstetricians & Gynaecologists Study Group on Intrapartum Fetal Surveillance in 1993.³² In 1999, cord blood gas analysis was again recommended by the Royal College of Obstetricians and Gynaecologists and Royal College of Midwives in a joint consensus statement³⁹

An International consensus statement involving the Obstetric Colleges from USA, UK, Australia, New Zealand, Canada and others stated that cord blood gas analysis was essential for assessing the outcome of labour²⁶.

Despite the accessibility of umbilical cord after delivery, some people remain uncertain about the value of the procedure. Although cord acid-base assessment provides an objective measure of neonatal condition at delivery,^{10,16,24,47,48} there is a lack of correlation with other measures of neonatal condition (Apgar scores, resuscitation, neonatal morbidity) and long-term outcome in some studies^{3,4,41} but not in others.^{8,9,25}

In addition, there is no consensus definition of 'acidosis' and the values of pH used in various studies range from 7.20^{29,50} down to 7.00.¹ Most studies refer to arterial pH values, but some have used venous values because of difficulty in obtaining samples from the artery,¹⁴ while others have not specified which vessel was used.¹³ In addition, only a few studies have attempted to distinguish respiratory and metabolic acidosis,^{21,47} despite the important and different pathophysiological implications of each.

The aim of this monograph is to resolve this confusion by reviewing the background to cord sampling, the physiology of fetal oxygen supply, acid-base balance and fetal responses to labour.

Expert DataCare, an intelligent knowledge based computer system, has been developed with the experience of cord blood sampling of every delivery in our large obstetric unit. In particular, it addresses the important practical aspects of cord sampling and data quality, provides advice on interpretation of the results and records all results, including quality controls and calibrations, to database for audit and research. We believe it greatly enhances the value of cord blood gas analysis and eases its introduction into routine clinical practice.

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Physiology and Pathophysiology

Placental/fetal circulation

The placenta is the organ of gas exchange for the fetus (Figure 1). Oxygen diffuses from maternal blood into fetal blood across the placental membrane and is carried to the fetus in the single large cord vein. Deoxygenated blood from the fetus returns to the placenta in two smaller arteries and the waste products of fetal metabolism, including carbon dioxide (CO₂), are transferred or diffuse into maternal blood. Thus cord *arterial* blood normally reflects fetal acid-base balance while *venous* blood reflects a combination of maternal acid-base status and placental function.

Fetal oxygen supply

The fetus lives and grows down-stream from the maternal oxygen supply in a partial pressure of oxygen which is so low that the term 'Mount Everest in utero' has been coined to describe its supposedly perilous existence. Yet the fetus has a number of special adaptive mechanisms which enable it to balance oxygen supply and energy demand to maintain aerobic metabolism (Figure 2).

The amount of oxygen available to the fetus depends upon the blood flow, the partial pressure of oxygen in the blood (pO₂), the haemoglobin concentration, the type of haemoglobin and the oxygen saturation. The increased oxygen-carrying capacity of fetal blood is due partly to its higher haemoglobin concentration and partly to the greater affinity of fetal haemoglobin for oxygen. This enables it to become saturated with oxygen at low partial pressures of oxygen. The amount of blood flow to an organ will also determine its oxygen supply.

Fetal cardiac output is approximately four times higher than that of the adult per kilogram of body weight,^{2,31} with the majority directed to the placenta, so that the fetal organs are normally supplied with more oxygen than they require. This 'oxygen reserve' means that fetal oxygen extraction can be increased at the tissue if needed.

Oxygen requirement is determined by fetal size, fetal activity and essential fetal metabolic processes. Energy demand can be reduced by decreasing activity or growth. If oxygen supply and requirement are in balance the fetus has adequate oxygen to metabolise glucose aerobically to produce the energy required for organ function.

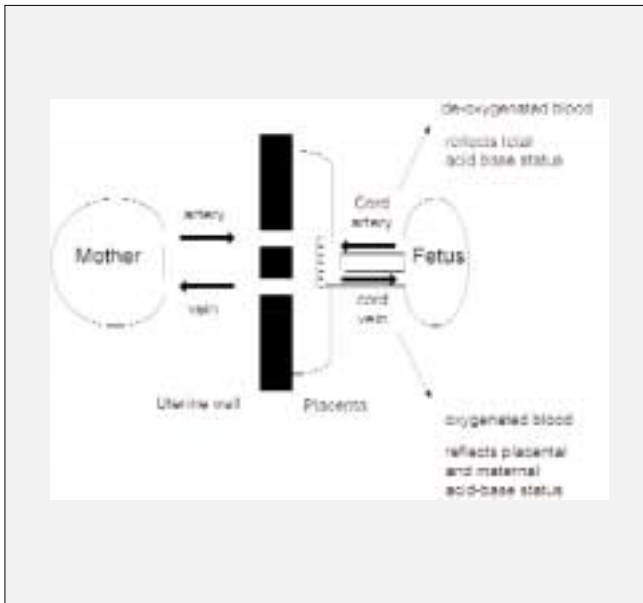


Figure 1. Placental circulation

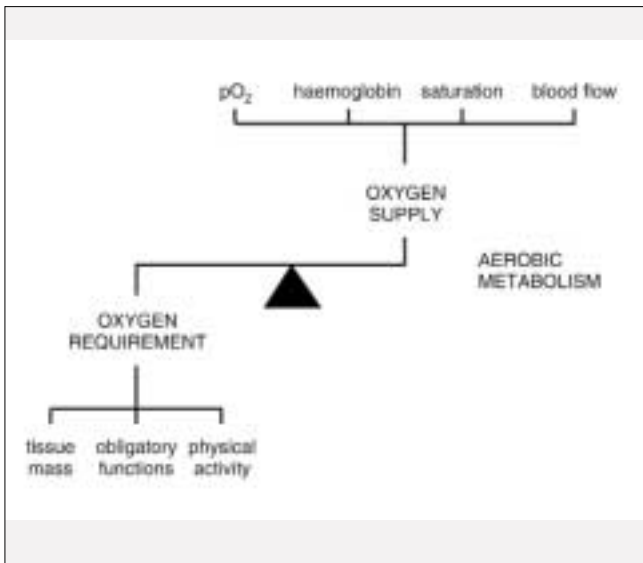


Figure 2. The factors which determine oxygen supply and requirement. Aerobic metabolism occurs when these are in balance.

Fetal responses to a reduction in oxygen supply

A number of mechanisms (Figure 3) can act both before and during labour to result in a reduction of fetal oxygen supply. The healthy fetus is very able to adjust to episodes of diminished oxygen supply by a synchronised response, which involves behavioural and cardiovascular adjustments.¹² The most important of these is the centralisation of blood flow to the heart and adrenals with increased oxygen extraction at the placental bed and tissues; these are largely mediated by beta-adrenergic receptors and the release of various humoral substances, particularly catecholamines from the fetal adrenal medulla.^{17,18,20,40}

If these adjustments fail to maintain adequate oxygen supply to central organs, aerobic metabolism is supplemented by anaerobic metabolism of glucose and glycogen to maintain cell and organ function.

The process of anaerobic metabolism produces lactic acid and releases hydrogen ions. The hydrogen ions are buffered primarily by bicarbonate ions, but also by haemoglobin and proteins. As the hydrogen ion concentration increases, these buffers are consumed and a progressive metabolic acidosis develops with a fall in pH and a rise in the base deficit of the extracellular fluid (Figure 4). Glycogen, the long term store of glucose, is depleted in this process.

Labour - a critical time for the fetus

Labour is a critical time for the fetus. During labour maternal blood supply to the placenta is normally interrupted during uterine contractions so that oxygen levels in fetal blood fall during contractions and recover once placental blood flow resumes (Figure 5). Thus a relative oxygen deficiency is a part of normal labour but most healthy fetuses have the physiological ability to sustain even stressful labour without handicap.

It is important to recognise not only that the time of onset, duration and severity of oxygen deficiency may vary, but also that the fetal compensatory mechanisms may be more proficient in one fetus compared to another. Thus the effect of an acute oxygen lack during labour in a healthy fetus is different from the same insult in a fetus that has already experienced chronic oxygen deficiency throughout pregnancy.

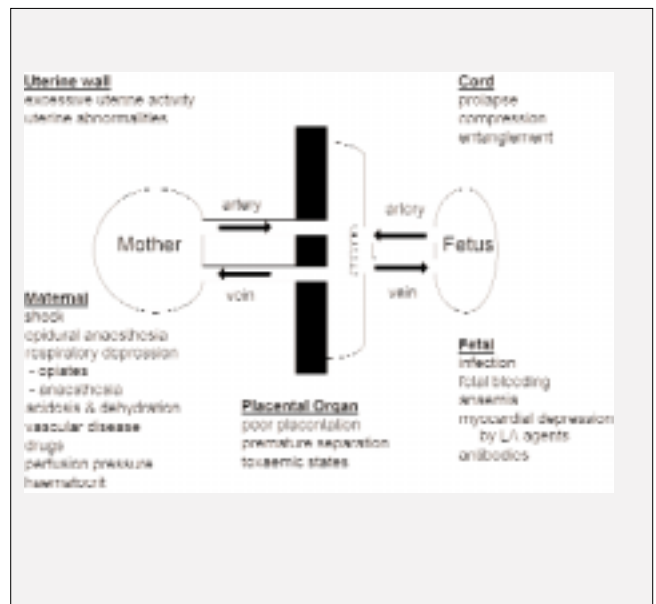


Figure 3.
Mechanisms by which fetal oxygen supply may be impaired.

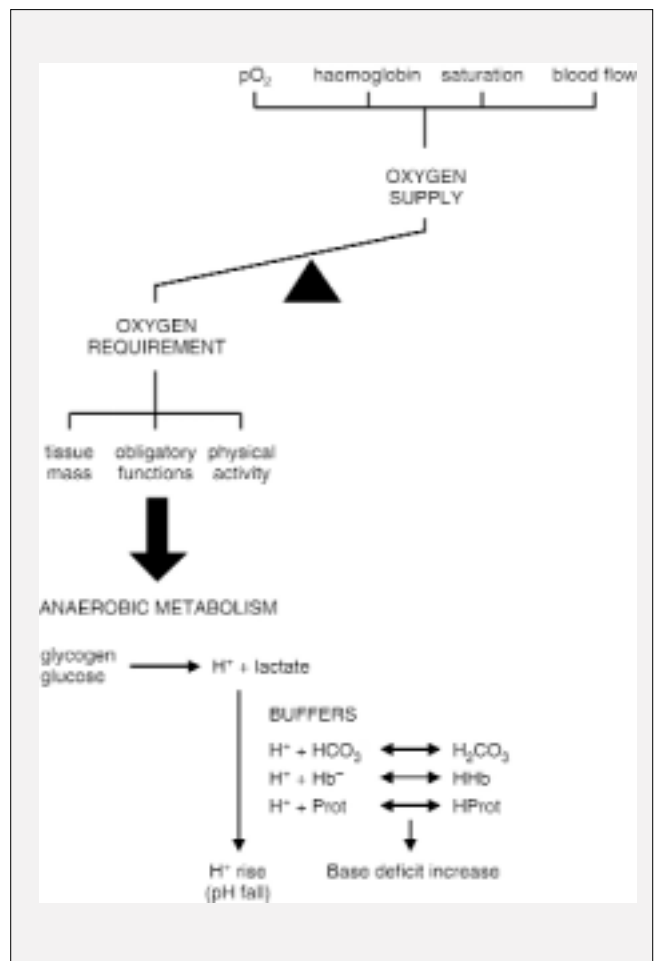


Figure 4.
Anaerobic metabolism. When oxygen supply is insufficient to meet requirements energy is produced by the anaerobic metabolism of glycogen and glucose.

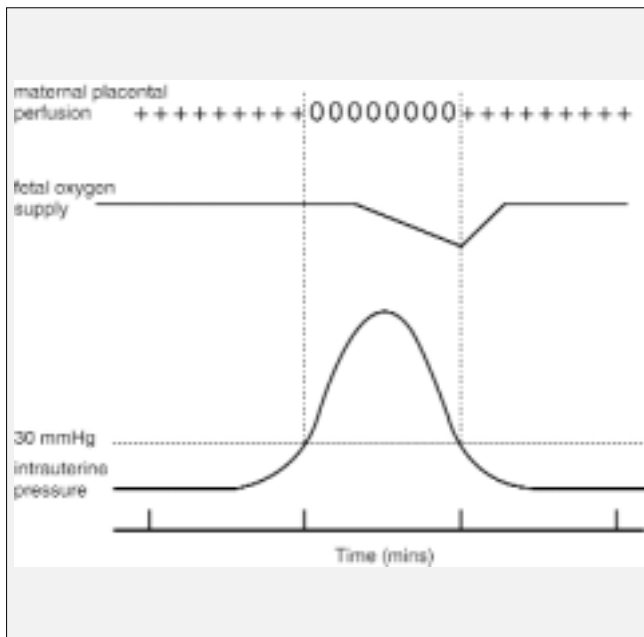


Figure 5.
The effect of uterine contractions on placental perfusion and fetal oxygen supply.

What is 'asphyxia'?

The word 'asphyxia' means 'without pulse', but in the fetus and neonate cardiac events are usually secondary and late sequelae to problems of blood gas transport. In the perinatal period **asphyxia** is strictly defined as the combination of oxygen lack and acidosis with impaired organ function.¹² It is important to realise that this only occurs after a sequence of events over time. These are:

1. **Hypoxaemia** - a reduction in oxygen carried in the blood as a result of decreased pO_2 and decreased oxygen content. This may be compensated for by increased blood flow and increased oxygen extraction to maintain oxygen supplies to the tissues.
2. **Hypoxia** - oxygen supply is insufficient for tissue energy requirements and aerobic metabolism becomes supplemented by anaerobic metabolism to maintain energy balance.
3. **Asphyxia** - if hypoxia continues, the process of anaerobic metabolism produces lactate and an increase in hydrogen ions so that oxygen lack is accompanied by acidosis. At some point in this process energy balance will no longer be maintained and organ failure will occur with a risk of permanent tissue damage.

For many years the diagnosis of 'intrapartum asphyxia' has been made primarily on the basis of the Apgar score. While a baby who has been severely hypoxic during labour can be expected to have low Apgars, asphyxia is not the only cause. Immaturity, infection, trauma, and congenital disorders are other possible causes. More recently, it has been suggested that neonatal encephalopathy is diagnostic of 'intrapartum asphyxia'. However, as with the Apgar score, hypoxia is not the only cause of encephalopathy; infection, hypoglycaemia, trauma and structural abnormalities can all be causes.^{15,28} Significant hypotension in the perinatal period may also cause cerebral underperfusion and cerebral injury.

Most would now agree that the diagnosis of 'intrapartum asphyxia' requires the presence of several, if not all, of the features listed above (Figure 6). The use of any single factor is likely to result in a heterogeneous population of neonates, only some of whom will have experienced significant intrapartum hypoxia. The loose use of the term 'birth asphyxia' should be avoided. Certainly the occurrence, description and timing of hypoxaemia and acidosis need to be more specific to avoid continuing confusion.

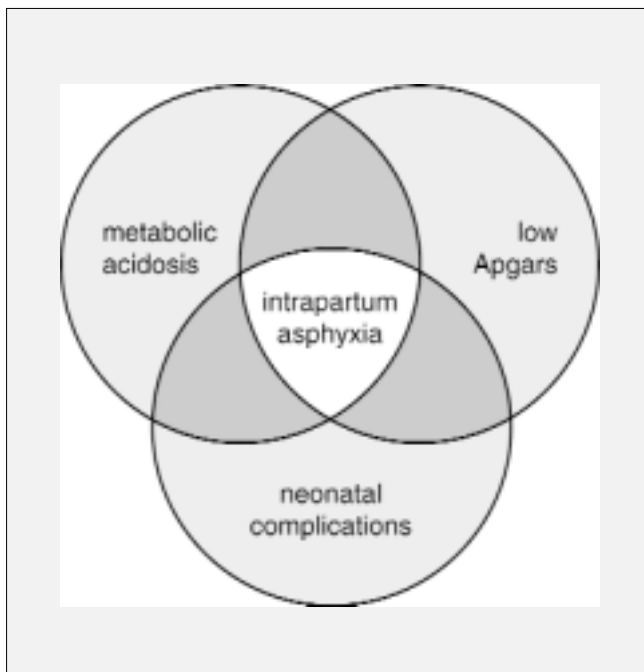


Figure 6.
A schematic representation of the combination of factors required to diagnose 'intrapartum asphyxia'.

Why don't Apgar scores correlate well with cord artery pH?

There is only a weak relationship between cord artery acidaemia and Apgar scores,^{5,8,41,44,46,47} but a strong association between the two should not be expected. The catecholamine surge which activates fetal defence mechanisms also has an effect on neonatal behaviour at birth. Catecholamines are an extremely important part of the normal adaptation to extrauterine life and produce a general neonatal arousal,²⁰ which will affect most components of the Apgar score. Thus it should be no surprise to find a high Apgar score where an appropriate response to hypoxia has occurred.¹⁹

The rationale for artery and vein cord blood gas assessment at birth

The analysis of blood from the umbilical cord at delivery provides objective information on the acid-base status of the fetus. It provides information on the occurrence, timing and possible causes of oxygen deficiency.^{37,51} A cord arterial metabolic acidaemia indicates a significant oxygen deficiency during labour; it does not, by itself, diagnose 'intrapartum asphyxia'.

Will all asphyxiated babies show a metabolic acidosis?

Can a fetus encounter a period of severe hypoxia during labour, sufficient to cause brain damage, but then recover to be born with normal cord gases and possibly even normal Apgar scores? Whilst it is possible that this may happen in the antepartum period, we have yet to see any evidence that it could happen during active labour. We believe it could not, as the recovery of responses and normalisation of acid-base balance would have to occur against the background of repeated episodes of hypoxaemia accompanying each uterine contraction.

Can a growth retarded fetus be 'asphyxiated' without showing a metabolic acidaemia because they do not have adequate energy stores to act as a substrate? This is unlikely as there is considerable evidence from cordocentesis studies that growth retarded fetuses have higher lactate levels and lower pHs than appropriately grown controls.^{27,30,33,45} Low et al²² demonstrated some time ago that decreased weight for gestational age was the best predictor of metabolic acidaemia and Schneider et al⁴² have also reported an increased incidence of arterial pH less than 7.10 in fetuses who were small for gestational age (10%) compared to appropriately grown fetuses (1%).

Practical aspects of cord sampling



Figure 7. An isolated segment of cord, kept intact with the placenta for illustration - normally the cord segment would be separated and removed prior to the third stage.

It is vital to ensure that the cord acid-base data obtained is reliable before attempts at interpretation are made. The important practical aspects of cord sampling are considered in this section. They are also covered in detail in chapter 1 of our computer based fetal monitoring training system.

A segment of cord must be isolated between two sets of clamps immediately after delivery (Figure 7). If this is not done the pH and pCO₂ in the cord artery will change as soon as the baby begins to breathe and therefore will not reflect acid-base status at delivery. We recommend a segment of cord (at least 20 cm) be separated from the rest of the cord and placenta and then passed out of the room for analysis. This allows the third stage to continue without further interruption. Too small a segment makes sampling difficult and it is important to ensure that the cord segment is full of blood, by 'milking' the cord from the placenta if necessary before clamping.

Time limits

Although changes in the pH, pCO₂ and pO₂ of cord blood occur with time as a result of cellular metabolism, these changes occur slowly. It has been shown that cord blood can be left at room temperature for up to one hour without significantly affecting the results.⁶ It can be kept for several hours if left on ice.

Choice of syringes

Blood does not usually clot while still in the cord, but we advise the use of heparinised syringes to take cord blood samples. If there is any delay in analysing the sample, clots may develop in the syringe and subsequently block the Blood Gas Analyser.

It is possible to pre-heparinise syringes on-site by adding liquid heparin to a 2 ml syringe. It should be noted, however, that liquid heparin can cause an error in the results if it makes up more than 10% by volume of the sample (heparin is an acid). The use of commercially prepared syringes containing lyophilised heparin will prevent measurement errors caused by varying amounts of heparin in each syringe.

Sampling

Blood must be taken from BOTH an artery and the vein and the results checked to ensure separate vessels have been sampled. Sometimes it can be difficult to obtain blood from the

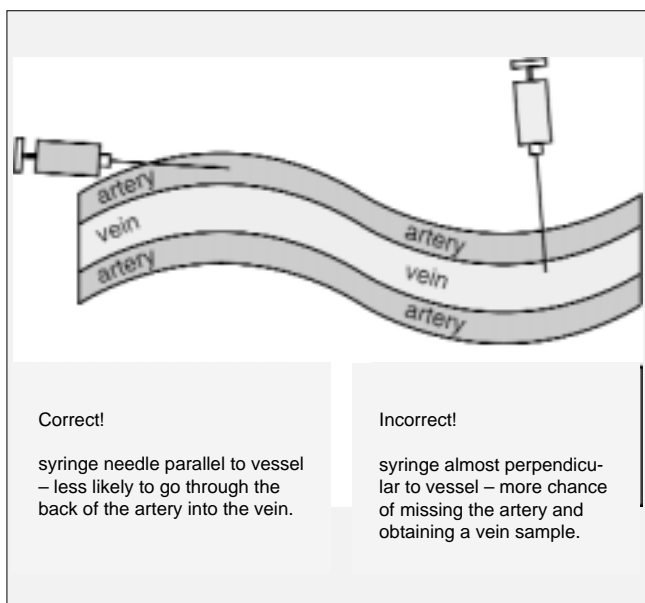


Figure 8. How to angle the needle when taking a cord sample.

small arteries and a venous or a mixed arterio-venous sample may be obtained by mistake (Figure 8). In a study done in Plymouth⁴⁹ we attempted to obtain paired cord samples from 1942 deliveries. However, in 7.4% of cases only one blood sample was obtained or the results from one of the vessels was identified as unreliable by the Blood Gas Analyser.

Figures 9a, 9b and 9c show the differences for pH, pCO₂ and pO₂ in the supposedly error-free paired samples. Note how in each case there are a number of results with negative differences and a cluster of results around a zero difference. Physiologically, arterial cord blood on return from the fetal tissue would be expected to have a lower oxygen content and a higher carbon dioxide content (thus also a lower pH) than venous cord blood. The most likely explanation for negative differences is that the samples were unwittingly reversed and for the nearly identical results is that the samples have been taken from the same vessel or are both mixed. A further 18% of samples were excluded to remove such possibly erroneous data.

In total, we had to reject just over 25% of our samples because they did not have full results from both artery and vein. With practice and feedback our sampling errors have been reduced to 15%, but even in very experienced hands the error rate is probably still around 10%.³⁶ Recently we have analysed cord samples collected from three hospitals during a multi-centre European study and found that only 40% of cases had full results from both vessels. It is very important to recognise that when sampling errors occur, venous samples can be mistakenly attributed to the artery. As we discuss later, arterial and venous values can be very different, so the incorrect assumption that an actual venous value is an arterial value would invalidate any attempt to correlate cord gas values with other measures of neonatal outcome. Unfortunately, many studies only report taking blood from the cord artery. Without a paired sample, showing the accompanying venous results, there would be no way of subsequently confirming this.

Blood Gas Analyser maintenance

Regular Blood Gas Analyser maintenance is essential if good quality results are to be obtained. A series of quality controls should be run through the machine daily and the calibrations throughout the preceding day should be checked.

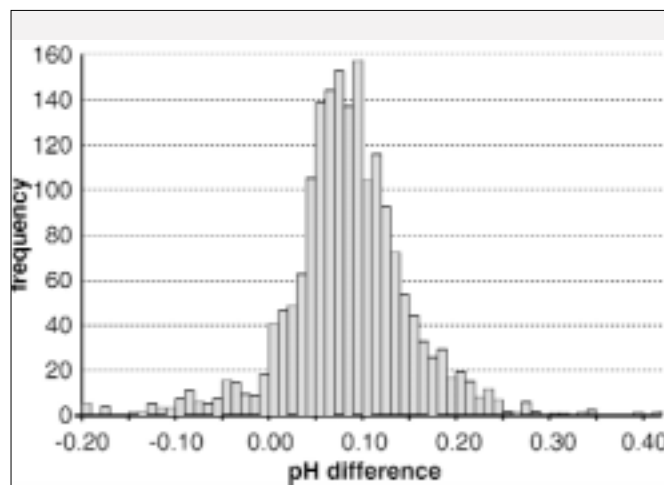


Figure 9a.
Venous - Arterial pH differences, n = 1815.

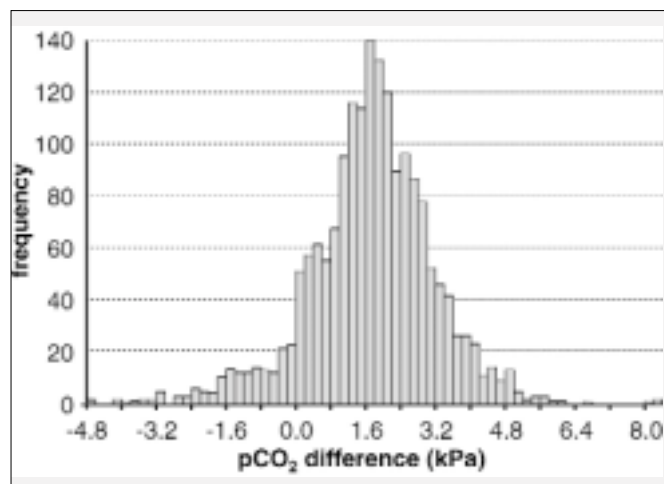


Figure 9b.
Arterial - Venous pCO₂ differences, n = 1798.

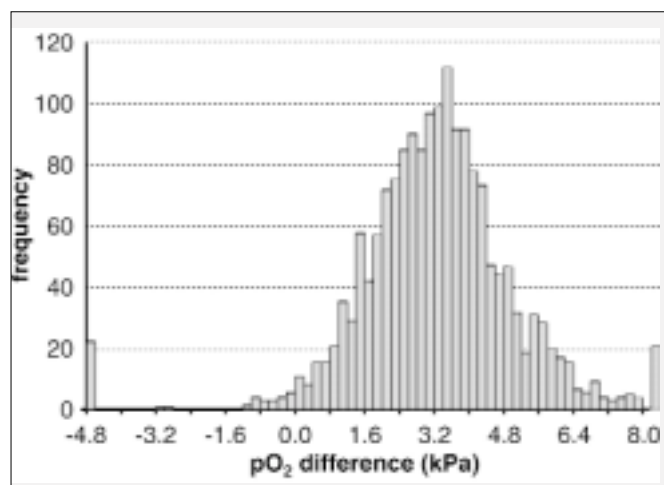


Figure 9a.
Venous - Arterial pO₂ differences, n = 1751.

Interpretation of cord results

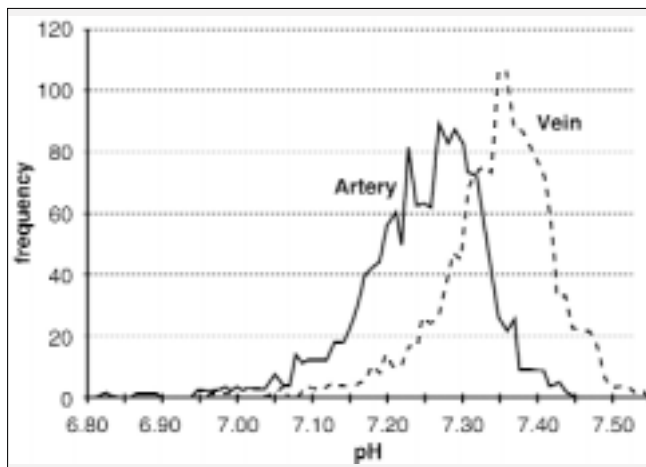


Figure 10a.
Frequency distribution of cord artery and vein pH, n = 1448.

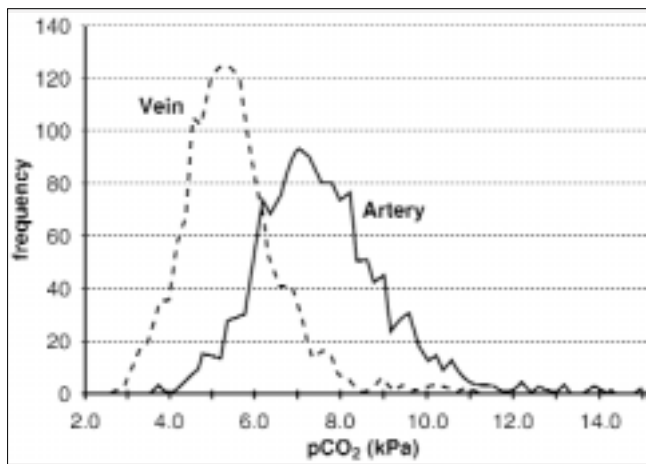


Figure 10b.
Frequency distribution of cord artery and vein pCO₂, n = 1448.

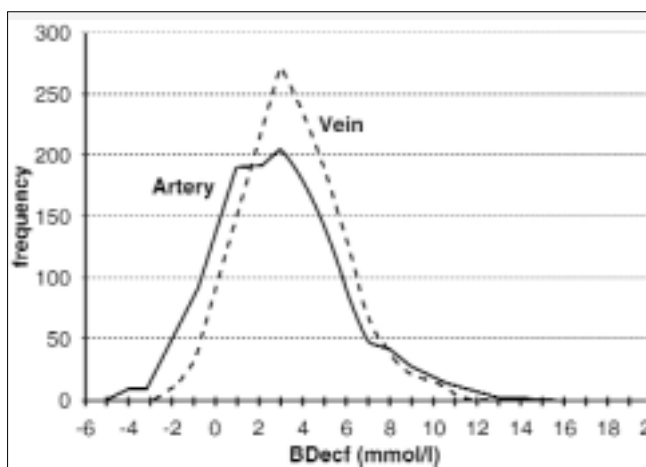


Figure 10c.
Frequency distribution of cord artery and vein BDecf, n = 1448.

Once validated cord artery and vein samples have been obtained, how should they be interpreted? What is the normal range of values? What is a metabolic acidaemia? In the literature, the values of cord artery pH used to define 'acidaemia' range from 7.20 down to 7.00, which is another reason why there is so much confusion about the significance of an 'acidaemia' at delivery.

The normal range of cord blood gas values

The frequency distribution of cord artery and vein pH levels in our 1448 validated pairs of samples is shown in Figure 10a. Note that these distributions are skewed and therefore any statistical description of these populations should be by centile values and not by mean and standard deviation.⁷ The same applies for pCO₂ (Figure 10b), pO₂ and base deficit (Figure 10c). Table 1 shows the normal ranges for these values obtained during the study in Plymouth.

What is base deficit?

As discussed previously, the process of anaerobic metabolism produces lactic acid, which dissociates to form lactate and hydrogen ions [H⁺]. pH is a logarithmic measure of free H⁺ ions, with the hydrogen ion concentration *increasing* exponentially with decreasing pH. H⁺ ions, which are toxic to tissues, are produced in large quantities by anaerobic metabolism, but the fall in the pH of blood is less than expected because the buffering systems shown in Figure 4 'mop up' many of the free H⁺ ions. Base deficit (BD) is a measure of how much base must be added to the system to return *both* the pH and buffers to normal. Thus base deficit is an indirect measure of the extent to which anaerobic metabolism has occurred.

Why measure base deficit?

The base deficit (derived from pH and pCO₂) allows a low pH caused by a build up of CO₂ (a respiratory acidaemia) to be distinguished from a low pH caused by a build up of metabolic acids secondary to anaerobic metabolism (a metabolic acidaemia). A respiratory acidaemia is usually corrected within minutes of birth with the establishment of adequate ventilation, as the excess CO₂ is blown off by the lungs. A metabolic acidaemia indicates that a significant period of hypoxia has occurred and is therefore a much more important finding.³⁸

Which base deficit measurement; BDblood or BDecf?

You will note that we referred to the base deficit of the extracellular fluid in Table 1. In adults BD is usually calculated from the blood compartment of the whole extracellular fluid (BDblood). This is appropriate in adults. It is not appropriate in the perinatal period as the fetus and neonate have a much larger extravascular fluid compartment, so that more of the buffering capacity is extravascular compared to the adult. This is particularly important when pCO₂ levels are high because a high pCO₂ will unduly influence BD values calculated from the blood compartment alone. The fetus is more likely to experience high pCO₂ values than the adult as a build up in CO₂ occurs commonly during labour. Siggaard-Andersen showed that the influence of a high pCO₂ could be avoided if the BD was calculated from the whole extracellular fluid (BDecf) and in 1971⁴³ he produced his Acid-Base Chart to allow this to be done. This can make a sizeable difference to BD calculation as can be seen from the examples of cord artery results in Table 2.

BDecf should therefore be used in the perinatal period to prevent the overdiagnosis of metabolic acidaemia.

What values of pH and base deficit are abnormal?

In Table 1 we showed the statistically defined range of blood gas parameters from a representative obstetric population. Is this approach justified? Is it physiological? Does a pH below the 2.5th centile for the population imply that it is the pH at which tissue damage begins to appear? We have already seen that pH alone does not distinguish between a respiratory and a metabolic acidaemia, so that both pH and BD values should be used to define a significant acidaemia.

In fact, the findings of several large studies indicate that the levels at which increased neonatal morbidity and mortality occur are a pH less than 7.05 or even less than 7.00,^{8,9,11,44,50} and a BDecf of greater than 10 or even greater than 12 mmol/l.²³ Experimental studies in sheep also indicate that a pH of 7.00 seems to be a critical level, associated with systemic hypotension and a terminal fall in cerebral oxygen consumption.³⁵ These levels (arterial pH less than 7.05 and BDecf greater than 12 mmol/l) are close to the 2.5th centile values for the population shown in Table 1.

	artery n=1448	vein n=1448
pH median	7.26	7.35
2.5 – 97.5th centiles	7.05 – 7.38	7.17 – 7.48
pCO ₂ median (mmHg)	55.0	40
2.5 – 97.5th centiles	37 – 80	26 – 59
pCO ₂ median (kPa)	7.3	5.3
2.5 – 97.5th centiles	4.9 – 10.7	3.5 – 7.9
BDecf median (mmol/L)	2.4	3.0
2.5 – 97.5th centiles	-2.5 – 9.7	-1.0 – 8.9

Table 1
Medians and centile ranges of blood gas values in 1448 validated cord artery and vein paired samples.

pH	pCO ₂ mmHg	pCo ₂ kPa	BDblood mmol/L	BDecf mmol/L
7.03	81.5	10.9	12.1	8.1
6.98	103.6	13.8	10.8	6.3
6.97	114.8	15.3	9.6	4.6

Table 2
A comparison of BDblood and BDecf calculations.

	Artery	Vein
pH	6.98	7.26
pCO ₂ (mmHg)	102.5	45.9
pCO ₂ (kPa)	13.7	6.1
pO ₂ (mmHg)	3.1	35.0
pO ₂ (kPa)	0.4	4.7
HCO ₃ ⁻	17.5	18.5
BDecf	6.5	5.6

Table 3.

A primigravida at term, spontaneous onset of labour. There were no fetal heart rate abnormalities in the first stage. Pushing commenced at 1320h; a normal delivery occurred at 1405h, with the cord tightly around the neck. The CTG showed frequent contractions with variable decelerations from 1350h, then a bradycardia (HR 60) for the last 7 minutes prior to delivery. The cord results are shown above; note the large A-V difference. The baby had Apgars 5 at 1, 7 at 5 and 8 at 10 minutes, required ventilation with bag and mask for 3 minutes (note the respiratory acidaemia with a high arterial pCO₂, but normal BDecf value).

	Case 1		Case 2	
	artery	vein	artery	vein
pH	7.03	7.10	7.04	7.32
pCO ₂ (mmHg)	63.0	49.5	67.5	38.0
pCO ₂ (kPa)	8.4	6.6	9.0	5.1
pO ₂ (mmHg)	7.0	19.5	13.0	33.5
pO ₂ (kPa)	0.9	2.6	1.7	4.5
BDecf (mmol/L)	12.5	12.5	11.0	5.6

Table 4.

Both babies had similar arterial results at birth but very different venous values. Case 1 required resuscitation at birth, was ventilated for 48 hours and has cerebral palsy at 12 months of age. Case 2 had a 5 minute Apgar score of 8 and no neonatal problems.

Are cord vein results useful?

Yes, for two reasons. Firstly, as already discussed, venous results are necessary to ensure that arterial blood has definitely been obtained. Secondly, they can provide important information about the time course and possible causes of any oxygen deficiency.

Cord arterial values reflect fetal acid-base status whereas those of the vein reflect maternal and placental status.

A normal venous result does not preclude the existence of an arterial acidaemia with a large arterial-venous (A-V) difference. This can occur when normal placental function and gas exchange are interrupted by an acute reduction in fetal blood flow, for example severe cord compression or profound bradycardia (see Table 3).

In contrast, an acidaemia in both artery and vein indicates that the hypoxia is not acute in onset. This is particularly so in the case of a metabolic acidaemia. Whilst CO₂ can diffuse rapidly across the placenta, H⁺ and lactate take much longer to equilibrate. With the onset of metabolic acidaemia, the H⁺ and lactate ions diffuse from the fetal blood into the placental extracellular fluid compartment until equilibrium is reached. This gradual process delays the appearance of metabolic acidaemia in the venous blood.¹²

As the fetus compensates for a reduced oxygen supply by increased blood flow to the vital organs and decreased blood flow to peripheral organs, a metabolic acidaemia occurs later in the central organs than in the peripheral organs. A venous metabolic acidaemia, indicating a non-acute event, therefore suggests that central organ metabolic acidosis is more likely to have occurred. We know that both the duration and severity of oxygen deficiency are important prognostic factors, so knowledge of both cord artery and vein status is valuable. The importance of both artery and vein sampling is illustrated by the cases shown in Table 4.

Why Expert DataCare?

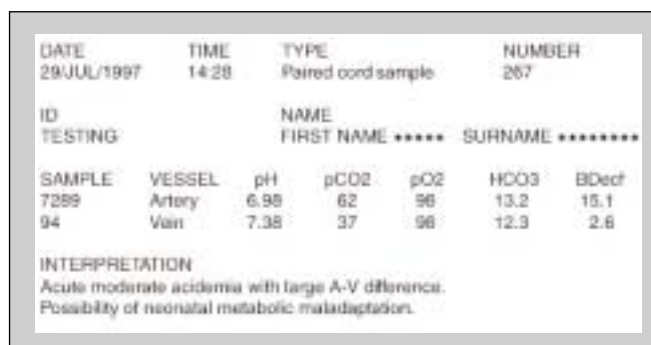
Hopefully it is clear from the preceding discussions that cord acid-base assessment involves much more than taking a blood sample from the cord and measuring the pH. Although cord blood gas analysis can provide very useful information, this will only be of value if the information is both correct and correctly interpreted.

Our experience has shown that the main difficulties are in recognising that the same vessel has not been sampled twice (or a mixture of arterial and venous blood has been obtained), recognising errors in analysis and distinguishing significant from non-significant results.

On a more practical level, it is important to ensure that each baby's results are recorded accurately and permanently in the appropriate notes (both mother's and baby's) and that the Blood Gas Analyser's calibrations and quality control results are logged. All these are then available for audit, research and medicolegal purposes.

Expert DataCare is an intelligent knowledge based computer system which has been designed by the Plymouth Perinatal Research Group to address these problems. Expert DataCare can connect to many of the leading manufacturer's blood gas analysers including, Bayer, Instrumentation Laboratory and Radiometer. Expert DataCare,

1. identifies whether the Analyser has noted any measurement errors,
2. identifies which vessel is which from an unlabelled paired sample,
3. identifies samples from the same vessel, giving the opportunity for immediate resampling,
4. checks to see if the results make physiological sense,
5. provides a brief interpretation of the results,
6. highlights any results which indicate fetal acidaemia and highlights possible neonatal consequences,
7. prints the patients name, hospital number, date and time of the sample, the results and a summary of the advice given onto the required number of sticky labels (figure 11)
8. records results onto a computer database for subsequent analysis, and
9. records all calibration and quality control data allowing the verification of Analyser performance.



DATE	TIME	TYPE	NUMBER			
29JUL/1997	14:28	Paired cord sample	267			
ID	NAME					
TESTING	FIRST NAME ***** SURNAME *****					
SAMPLE	VESSEL	pH	pCO2	pO2	HCO3	BDefc
7289	Artery	6.98	62	96	13.2	15.1
94	Vein	7.38	37	96	12.3	2.6
INTERPRETATION						
Acute moderate acidemia with large A-V difference. Possibility of neonatal metabolic maladaptation.						

Figure 11.
Expert DataCare printed label

The interpretations have been derived after discussion with several leading obstetricians, neonatologists and physiologists. On the basis of our experience and the evidence in the literature, the following levels are used as physiologically significant levels of pH and BDecf;

artery; pH < 7.05, BDecf > 10 and > 12 mmol/l,

vein; pH < 7.10, BDecf > 10 mmol/l.

The aim of an 'Expert System' is not to replace 'experts' or experienced clinicians but to use expert knowledge to support the actions and decision-making of the inexperienced and non-expert. Personnel with the knowledge and experience to check and interpret cord blood gas results are unlikely to be available in every labour ward 24 hours a day. A daily retrospective check of all cord blood gas results to ensure data quality and the accuracy of data transcription would be an onerous task but one which is easily accomplished on-line by the expert system.

The use of the **Expert DataCare** system provides a database of validated cord gas data which has many advantages for both obstetrician and paediatrician. This information is useful,^{16,34} as it;

1. provides objective information on fetal acid-base status at delivery which can be used as part of the overall assessment of neonatal condition,
2. provides an objective tool for the audit of intrapartum care,
3. encourages a physiologically-based approach to intrapartum fetal assessment,
4. aids the counselling of parents of babies who encounter problems in the neonatal period, and
5. helps in the defence of medicolegal cases to determine whether intrapartum asphyxia has been responsible for a long term handicap.

The Royal College of Obstetricians and Gynaecologists has recommended the analysis of cord blood gas samples, but this is unlikely to be of benefit unless they are taken for every delivery and correctly interpreted. The **Expert DataCare** system simplifies the analysis, provides the necessary interpretation, and eases the introduction of routine cord blood gases into clinical practice.

References

1. American Academy of Pediatrics. Use and abuse of the Apgar score. *Pediatrics* 1986;78:1148-1149.
2. Cohn HE, Sacks EJ, Heyman MA, Rudolph AM. Cardiovascular responses to hypoxemia and acidemia in fetal lambs. *Am J Obstet Gynecol* 1974;120:817-824.
3. Dennis J, Johnson A, Mutch L, Yudkin P, Johnson P. Acid-base status at birth and neurodevelopmental outcome at four and one-half years. *Am J Obstet Gynecol* 1989;161:213-20.
4. Dijkhoorn MJ, Visser GHA, Huisjes HJ, Fidler V, Touwen BCL. The relationship between umbilical pH values and neonatal neurological morbidity in full term appropriate-for-dates infants. *Early Hum Dev* 1985;11:33-42.
5. Dijkhoorn MJ, Visser GHA, Fidler VJ, Touwen BCL, Huisjes HJ. Apgar score, meconium and acidaemia at birth in relation to neonatal neurological morbidity in term infants. *Br J Obstet Gynaecol* 1986;93:217-222.
6. Duerbeck NB, Chaffin DG, Seeds JW. A practical approach to umbilical artery pH and blood gas determinations. *Obstet Gynecol* 1992;79:959-962.
7. Eskes TKAB, Jongsma HW, Houx PCW. Percentiles for gas values in human umbilical cord blood. *Eur J Obstet Gynecol Reprod Biol* 1983;14:341-346.
8. Gilstrap LC, Leveno KJ, Burris J, Williams ML, Little BB. Diagnosis of birth asphyxia on the basis of fetal pH, Apgar score, and newborn cerebral dysfunction. *Am J Obstet Gynecol* 1989;161:825-30.
9. Goldaber KG, Gilstrap LC, Leveno KJ, Dax JS, McIntyre DD. Pathologic fetal acidemia. *Obstet Gynecol* 1991;78:1103-1107.
10. Goldaber KG, Gilstrap LC. Correlations between clinical events and umbilical cord blood acid-base and blood gas values. *Clinical Obstet Gynecol* 1993;36:47-59.
11. Goodwin TM, Belai I, Hernandez P, Durand M, Paul RH. Asphyxial complications in the term newborn with severe umbilical acidemia. *Am J Obstet Gynecol* 1992;162:1506-1512.
12. Greene KR and Rosen KG. Intrapartum Asphyxia. In 'Fetal and Neonatal Neurology and Neurosurgery'. Eds Levene, Lilford, Bennett, Punt. Churchill Livingstone, London 1994.
13. Halligan A, Connolly M, Clarke T, Gleeson RP, Holohan M, Matthews T, King M, Darling MRN. Intrapartum asphyxia in term and post term infants. *Irish Med J* 1992;85:97-100.
14. Huisjes HJ, Aarnoudse JG. Arterial or venous umbilical pH as a measure of neonatal morbidity? *Early Hum Dev* 1979;3:155-61.
15. Hull J, Dodd KL. Falling incidence of hypoxic-ischaemic encephalopathy in term infants. *Br J Obstet Gynaecol* 1992;99:386-391.
16. Johnson JWC, Richards DS, Wagaman RA. The case for routine umbilical blood acid-base studies at delivery. *Am J Obstet Gynecol* 1990;162:621-625.
17. Jones CT, Ritchie JWK. The cardiovascular effects of circulating catecholamines in fetal sheep. *J Physiol* 1978a;285:381-394.
18. Jones CT, Ritchie JWK. The metabolic and endocrine effects of circulating catecholamines in fetal sheep. *J Physiol* 1978b;285:395-399.
19. Lagercrantz H. Asphyxia and the Apgar Score. *Letter. Lancet* 1982;i:966.
20. Lagercrantz H, Slotkin TA. The "stress" of being born. *Scientific American* 1986;254:100-107.
21. Low JA, Galbraith RS, Muir D, Killen H, Karchmar J, Campbell D. Intrapartum fetal asphyxia: a preliminary report in regard to long term morbidity. *Am J Obstet Gynecol* 1978;130:525-533
22. Low JA, Karchmar EJ, Broekhoven L, Leonard T, McGrath MJ, Panchar SR, Piercy WN. The probability of fetal metabolic acidosis during labor in a population at risk as determined by clinical factors. *Am J Obstet Gynecol* 1981;141:941-951.
23. Low JA, Galbraith RS, Muir DW, Killen HL, Pater EA, Karachmar EJ. Factors associated with motor and cognitive deficits in children following intrapartum fetal hypoxia. *Am J Obstet Gynecol* 1984;159:533-538.
24. Low JA. The role of blood gas and acid-base assessment in the diagnosis of intrapartum fetal asphyxia. *Am J Obstet Gynecol* 1988;159:1235-40.
25. Low JA, Galbraith RS, Muir DW, Killen HL, Pater EA, Karchmar EJ. Motor and cognitive deficits after intrapartum asphyxia in the mature fetus. *Am J Obstet Gynecol* 1988;158:356-61.
26. MacLennan A, for the International Cerebral Palsy Task Force. A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement. *BMJ* 1999; 319, October 16, 1054-1059.
27. Nicolaidis KH, Economides DL, Soothill PW. Blood gases, pH, and lactate in appropriate and small for gestational age fetuses. *Am J Obstet Gynecol* 1989;161:996-1001.
28. Nelson KN, Leviton A. How much of neonatal encephalopathy is due to birth asphyxia? *Am J Dis Child* 1991;145:1325-1331.
29. Page FO, Martin JN, Palmer SN, Martin RW, Lucas JA, Meeks GR, Bucovaz ET, Morrison JC. Correlation of neonatal acid-base status with Apgar scores and fetal heart rate tracings. *Am J Obstet Gynecol* 1986;154:1306-1311.
30. Pardi G, Buscaglia M, Ferrazzi E, Bozetti P, Marconi AM, Cetin I, Battaglia FC, Makowski EL. Cord sampling for the evaluation of oxygenation and acid-base balance in growth-retarded human fetuses. *Am J Obstet Gynecol* 1987;157:1221-1228.
31. Peeters LLH, Sheldon RE, Jones MD, Makowski EL, Meschia G. Blood flow to fetal organs as a function of arterial oxygen content. *Am J Obstet Gynecol* 1979;135:637-643.
32. Recommendations arising from the 26th RCOG Study Group. In 'Intrapartum Fetal Surveillance'. Eds Spencer JAD and Ward RHT. RCOG Press, London 1993.
33. Ribbert LSM, Snijders RJM, Nicolaidis KH, Visser GHA. Relation of fetal

- blood gases and data from computer-assisted analysis of fetal heart rate patterns in small for gestation fetuses. *Br J Obstet Gynaecol* 1991;98:820-823.
34. Richards DS, Johnson JWC. The practical implications of cord acid-base studies. *Clinical Obstet Gynaecol* 1993;36:91-98.
 35. Richardson BS. Fetal adaptive responses to asphyxia. *Clin Perinatol* 1989;16:595-611.
 36. Riley RJ, Johnson JWC. Collecting and analysing cord blood gases. *Clin Obstet Gynaecol* 1993;36:13-23.
 37. Roemer VM, Harms K, Buess H, Horvath TJ. Response of fetal acid base balance to duration of second stage of labour. *Int J Gynaecol Obstet* 1976;114:455-464.
 38. Rooth G. Perinatal acid-base balance. Studentlitteratur, Lund, Sweden 1988.
 39. Royal College Obstetricians and Gynaecologists & Royal College of Midwives - Towards Safer Childbirth - minimum standards for the organisation of labour wards. Report of a joint working party. February 1999.
 40. Rudolph AM. The fetal circulation and its response to stress. *J Dev Physiol* 1984;6:11-19.
 41. Ruth VJ, Raivio KO. Perinatal brain damage: predictive value of metabolic acidosis and the Apgar score. *Br Med J* 1988;297:24-27.
 42. Schneider EP, Stein JL, Verma VL, Tejani N. Umbilical artery acid base in SGA neonates. Society of Perinatal Obstetricians, Las Vegas, Nevada, Feb 3-6, Abstract 315, 1988. (Quoted in Goldaber & Gilstrap, 1993).
 43. Siggaard-Andersen O. An acid-base chart for arterial blood with normal and pathophysiological reference areas. *Scand J Clin Lab Invest* 1971;27:239-245.
 44. Silverman F, Suidan J, Wasserman J, Antoine C, Young BK. The Apgar score: is it enough? *Obstet Gynaecol* 1985;66:331-336.
 45. Soothill PW, Nicolaidis KH, Campbell S. Prenatal asphyxia, hyperlacticaemia, hypoglycaemia, and erythroblastosis in growth retarded fetuses. *Br Med J* 1987;294:1051-1053.
 46. Steer PJ, Eigbe F, Lissauer TJ, Beard RW. Interrelationships among abnormal cardiocograms in labor, meconium staining of the amniotic fluid, arterial cord blood pH, and Apgar scores. *Obstet Gynaecol* 1989;74:715-721.
 47. Sykes GS, Johnson P, Ashworth F, Molloy PM, Gu W, Stirrat GM, Turnbull AC. Do Apgar scores indicate asphyxia? *Lancet* 1982;i:494-496.
 48. Sykes GS, Molloy PM, Johnson P, Stirrat GM, Turnbull AC. Fetal distress and the condition of newborn infants. *Br Med J* 1983;287:943-945.
 49. Westgate J, Garibaldi JM, Greene KR. Umbilical cord blood gas analysis at delivery; a time for quality data. *Br J Obstet Gynaecol*, 1994;101:1.
 50. Winkler CL, Hauth JC, Tucker JM, Owen J, Brumfield CG. Neonatal complications at term as related to the degree of umbilical artery acidemia. *Am J Obstet Gynaecol* 1991;164:637-41.
 51. Yudkin PL, Johnson P & Redman CWG. Obstetric factors associated with cord blood gas values at birth. *Eur J Obstet Gynaecol Reprod Biol* 1987;24:167-176.

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