# Umbilical Cord Lactate: A preliminary study of 130 term babies

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# **ABSTRACT**

To determine the metabolic status of the newborn, pH and base deficit has been widely used. However, the correlation between neonatal outcome and pH or base deficit is poor. The measurement of umbilical cord blood lactate has been proposed as it is the end product of anaerobic metabolism which occurs during fetal hypoxia. A preliminary study on 130 term babies was undertaken to establish the normal range of umbilical cord lactate levels and to determine the relationship between the lactate levels, pH and base deficit. Of the 130 term deliveries, 12 were diagnosed to have non-reassuring fetal status from intrapartum fetal heart rate pattern. The mean lactate level in babies without evidence of fetal distress was 4.3mmol/l with a S.D. of 1.5, and 5.5 mmol/l with a S.D. of 2.1 in the fetal distress group. This study also showed that a definite correlation exists between the lactate level and the pH or base deficit in both the umbilical artery and vein in normal babies as well as those with fetal distress.

Keywords: lactate, pH, base deficit

# INTRODUCTION

Birth asphyxia has long been considered a major cause of perinatal mortality and morbidity, including the long-term neurologic sequelae in the newborn. It has been defined in many different ways. Low Apgar scores, tachypnoea, few minutes of assisted ventilation at birth due to the baby not crying immediately, are all classed as birth asphyxia. In recent years, acid-base balance has attracted special attention as a means of evaluating neonatal metabolic status at birth, since it mirrors hypoxic events during labour and at delivery. However, only about 40% babies depressed at birth are acidaemic<sup>1</sup>, and fetal acidosis and depressed

infants at birth represent two different entities that partly overlap. Sykes and colleagues² reported that, of those infants with umbilical artery acidaemia at delivery, only 14% had five minute Apgar scores of less than seven and conversely, of those with low Apgar scores (< 7 at 5 min) only 19% were severely acidaemic at birth. A number of other investigators have published data on the relationship between neurologic outcome and umbilical artery pH values, and with few exceptions, a poor correlation between pH values and the ultimate neurodevelopmental outcome was demonstrated³-9.

The phenomenon of low pH and good Apgar scores may be partly explained by the presence of transient respiratory acidaemia, which is not an uncommon occurrence during normal labour and delivery<sup>10</sup>. The correlation between cord artery acid-base balance and the presence of neonatal complications is poor, and significance is reached only when the pH level falls below 7.0<sup>11</sup>. In order to better define the metabolic status of the newborn, umbilical lactate levels have been measured. Lactate is converted from pyruvate during anaerobic metabolism, and it is well acknowledged that blood lactate is a useful systemic indicator of circulatory impairment and anaerobiosis. It has been shown that the accumulation of lactic acid

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in the brain in association with hypoxia causes oedema and tissue necrosis<sup>12</sup>. Intrapartum fetal hypoxia may result in fetal death or neurological damage. Blood lactate measurements may be used during labour and delivery as a measure of metabolic acidosis in the fetus and newborn<sup>13</sup>.

In view of the potential advantage of lactate over pH, a study was undertaken in the KK Women's and Children's Hospital with the aim to establish the normal range of lactate levels in the umbilical cords of our patients, and to determine the relationship between the lactate levels, pH and the base deficit.

## **MATERIALS AND METHODS**

A segment of umbilical cord 5 to 8 cm long was collected from term deliveries (37 to 42 weeks gestation). Measurements of lactate level, pH and base deficit were obtained from both the umbilical artery and vein. Using two pre-heparinised syringes, blood was first collected from the umbilical artery followed by the vein. About 5 ul of blood was obtained for lactate measurement. The lactate was determined using a portable Lactate Pro (KDK corporation, Kyoto, Japan). Lactate pro is a test strip microvolume lactate meter. It is an amperometric method using an enzymatic reation with lactate oxidase and potassium ferricyanide as an electron mediator. A meter measures the magnitude of the anodic current of the reduced mediator (ferrocyanide) by the enzymatic reaction and displays the lactate concentration. The pH and base deficit were determined using the standard blood gas analyzer (AVL 995) in the delivery suite. Measurements were made within one hour after delivery as previous studies have shown that the pH and lactate level do not alter significantly within the hour. The correlation between lactate and pH/base deficit was determined using the Pearson's product moment.

#### **RESULTS**

A total of 130 umbilical cords were obtained and analysed. The mean maternal age was 30.1 years (19 to 41 years) and the mean gestational age was 39.3 weeks (37 to 41.7 weeks). The mean birthweight was 3150g (2160 to 4095g). Of the 130 cords analysed, 12 were collected from babies diagnosed to have non-reassuring fetal status from intrapartum CTG changes. There was no significant difference in the maternal age, gestational age and birthweight between the two groups (Table 1). The results for pH, base deficit and lactate levels for the 2 groups are shown in Table 2. The mean lactate level in our term babies without evidence of fetal distress was 4.3 mmol/l with a standard deviation of 1.5, and 5.5 mmol/l with a standard deviation of 2.1 in the group with fetal distress.

A definite correlation was also noted between the lactate level and pH and base deficit in the umbilical artery and umbilical vein in both the fetal distress and non-fetal distress groups (Figs. 1 to 4). The correlation was stronger in the umbilical vein compared to umbilical artery in all the groups except that between lactate and base deficit in the fetal distress group. Similarly, the correlation was statistically significant in all the groups with the exception of the last group (Tables 3 to 6).

TABLE 1 Characteristics of non-fetal distress group and fetal distress group

	Non-fetal distress group (n = 118)	Fetal distress group (n = 12)	P NS
Maternal age (yr)	30	31	NS
Gestational age (weeks)	39.3	39.7	NS
Birthweight (g)	3196	3087	NS

NS: Not significant

TABLE 2
pH, base deficit and lactate in both umbilical artery and umbilical vein in the non-fetal distress group and fetal distress group

	Non-fetal distress group (n = 118) (mean ± SD)	Fetal distress group (n = 12) (mean ± SD)	р
Umbilical artery pH	7.21 ± 0.08	7.16 ± 0.10	< 0.05
Umbilical vein pH	7.28 ± 0.06	7.20 ± 0.08	< 0.05
Umbilical artery Base deficit (mmol/l	6.37 ± 3.35	8.64 ± 3.76	< 0.05
Umbilical vein Base deficit (mmol/l)	5.12 ± 2.64	6.78 ± 2.57	< 0.05
Umbilical artery lactate (mmol/l)	4.3 ± 1.5	1.5 5.5 ± 2.1 <	
Umbilical vein Lactate (mmol/l)	3.6 ± 1.4	4.7 ± 1.3	< 0.05

**TABLE 3**Correlation between lactate and pH (non fetal distress group)

	r	р
Umbilical artery	-0.479	< 0.05
Umbilical vein	-0.677	< 0.05

 TABLE 4

 Correlation between lactate and base deficit (non fetal distress group)

	r	р
Umbilical artery	0.571	< 0.05
Umbilical vein	0.674	< 0.05

**TABLE 5**Correlation between lactate and pH (fetal distress group)

	r	р
Umbilical artery	-0.745	< 0.05
Umbilical vein	-0.775	< 0.05

**TABLE 6**Correlation between lactate and base deficit (fetal distress group)

	r	р
Umbilical artery	0.663	< 0.05
Umbilical vein	0.494	< 0.05

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**TABLE 7** Comparison of the mean lactate level between various studies

	Lactate (mmol/l)	+2 S.D.
Smith et al ('83)	3.5	7.3
Suidan and Young ('84)	2.55	5.0
Ruth and Raivio ('88)	2.9	5.4
Nordstrom et al ('94)	3.7	6.1
Westgren et al ('95)	1.87	3.75
Watt et al	4.3	7.3

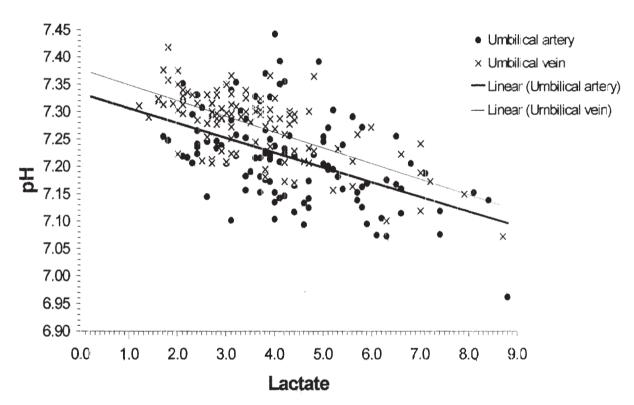


Fig. 1. Correlation between Lactate and pH (non-fetal distress group)

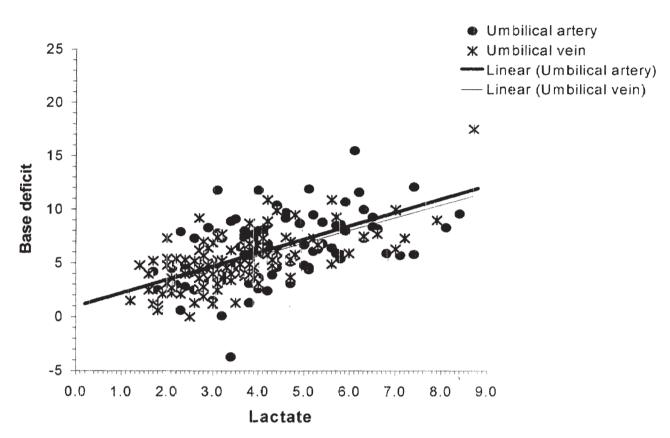


Fig. 2. Correlation between Lactate and Base deficit (non-fetal distress group)

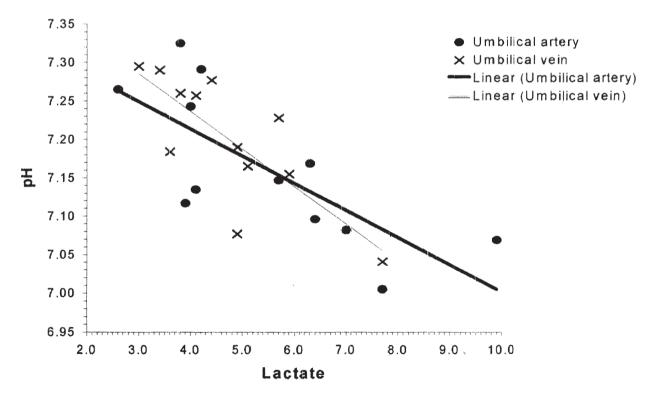


Fig. 3. Correlation between lactate and pH (fetal distress group)

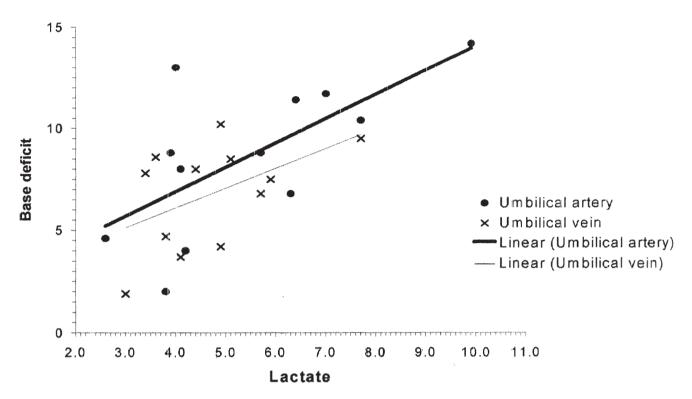


Fig. 4. Correlation between Lactate and Base deficit (fetal distress group)

# **DISCUSSION**

The mean lactate level in our term babies without fetal distress was higher than several other studies (Table 7). One reason for the difference could be related to the different method of lactate measurement. Furthermore, there may be inherent differences between the different populations studied.

The pH was lower and the lactate and base deficit higher in the umbilical artery compared to the vein. This was not surprising as the umbilical artery carried deoxygenated blood from the fetus while the umbiblical vein carried oxygenated blood to the fetus. The pH was also noted to be lower and lactate and base deficit higher in the group with fetal distress compared to the non-fetal distress group. This could be explained by the presence of a hypoxic state in the group with fetal distress.

The correlation between lactate and pH/base deficit was better in the umbilical vein compared to the umbilical artery. pH in the umbilical artery was contributed in part by respiratory acidaemia. Following gaseous exchange in the placenta, the respiratory component was eliminated. Hence the venous blood was affected mainly by the metabolic component. The strongest correlation was seen between the lactate level and pH in the group with non-reassuring fetal status.

Those lactate is a better predictor of the metabolic status, it has not found widespread use in clinical practice. This is because the older methods of measurement are cumbersome. A large volume of blood (about 1 to 2 ml) is required, and a special medium is necessary for transport to the laboratory where wet chemistry methods are employed. Often, there is a delay before results are available.

With the portable meter, lactate measurement has become much simpler. Only 5ul of blood is required compared to 50ul required for acid-base measurement. The test can be performed by the bedside, and the results available within 1 minute. The cost of measuring lactate is also cheaper compared to pH or base deficit measurement as it does not require an expensive blood gas machine or maintenance cost.

## CONCLUSION

This study has shown that a definite correlation exists between the lactate level and the pH/base deficit in both the umbilical artery and vein in normal babies as well as those with fetal distress. As lactate is simpler and cheaper to measure, it is a promising parameter that may be used in place of pH or base deficit in assessing the metabolic status of the newborn.

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## **REFERENCES**

- Ruth VJ, Raivo KO. Perinatal brain damage: predictive value of metabolic acidosis and the Apgar score. BMJ 1988; 297: 24-7
- Sykes GS, Molloy MP, Johnson P et al. Do Apgar scores indicate asphyxia? Lancet 1982; 1:494-6.
- 3. Dijxhoorn MJ, Visser GHA, Vidler 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; 93:217-22.
- 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.
- 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.
- Dennis J, Johnson A, Mutch L, Yudkin P, Johnson P. Acidbase status at birth and neurodevelopmental outcome at four and one-half years. Am J Obstet Gynecol 198; 161:213-20
- Fee SC, Malee K, Deddish R, Minogue JP, Socol ML. Severe acidosis and subsequent neurologic status. Am J Obstet Gynecol 1990; 162:802-6.

- Goldaber KG, Gilstrap LC, Leveno KJ, Dax JS, McIntire DD. Pathologic fetal acidemia. Obstet Gynecol 1991; 78: 1103-7.
- Winkler CL, Hauth JC, Tucker JM, Owen J, Brumfield CC. Neonatal compl,ications at term as related to the degree of umbilical artery acidemia. Am J Obstet Gynecol 1991; 164: 637-41.
- Ingemarsson I, Aurlkumaran S. Fetal acid-base balance in low-risk patients in labour. Am J Obstet Gynecol 1986; 155: 66-9
- Goodwin TM, Belai I, Hernandez P et al. Asphyxial complications in the term newborn with severe acidemia. Am J Obstet Gynecol 1992; 167(6):1506-12.
- Myers RE, Wagner KR, Courten GD. Lactic acid accumulation in tissue as cause of brain injury and death in cardiogenic shock from asphyxia. In: Lauersen NH, Hochberg HM, eds. Clinical perinatal biochemical monitoring. Baltimore: Williams & Wilkins, 1981:11.
- Nordstrom L, Ingemarsson I, Persson B, Shimojo N, Westgren M. Lactate in fetal scalp blood and umbilical artery blood measured during normal labor with a test strip method. Acta Obstet Gynecol Scand 1994; 73(3):250-4.

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