STUDY OF MAJOR METABOLITIES OF CARBOHYDRATE AND LIPIDS IN HUMAN LABOUR AND THEIR RELATION TO FOETAL WELL BEING

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SUMMARY

Glucose, lactic acid and free fatty acid levels were determined in maternal blood at term and during different stages of normal and abnormal labour and also in cord blood at the time of delivery to evaluate their relation between their maternal and foetal levels and their effects on foetal well being. All these three fuel metabolites in maternal blood were found to show a progressive rise during various stages of labour in all cases of normal vaginal deliveries. In the cord blood the glucose concentration corresponded to maternal concentration at the second stage of labour, lactic acid level was about two times higher than its maternal blood concentration, whereas cord free fatty acid concentration was much lower than in the maternal blood.

Raised maternal lactic acid concentration was found to be an important index of foetal distress, whereas, no such correlation was observed between glucose and free fatty acid levels and condition of baby at birth.

Introduction

The process of human labour invokes stress in mother and foetus and demands more energy. The energy demand in the body is met mainly by the major metablites of carbohydrates and lipids viz. glucose, lactic acid and free fatty acids. The effect of stress on these metabolites during labour has been studied by various workers (Dahl, 1928, Peters et al 1951, Hendricks, 1957, Burt 1960, Burt et al 1962, Whaley et al, 1966 and 1967, Fairweather 1971). But most of these works are limited in scope and are confined to the study of either glucose, lactic acid or free fatty acid levels at the last stage of labour.

The present work was undertaken to study these major fuel metabolites together, in maternal blood at term and during different stages of normal and

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abnormal labour and also in cord blood at the time of delivery to evaluate their relation between their maternal and foetal levels and their effect on foetal well being.

Material and Methods

Seventy four pregnant women from S.R.N. Hospital and Kamala Nehru Memorial Hospital, Allahabad were selected for this study. Diabetic patients were excluded. These cases were divided into the following groups:

A. Normal labour at term	30 cases
B. Normal pre-term labour	14 cases
C. Postmature induced labour (other than by oxytocin infusion)	12 cases
D. Forceps delivery	8 cases
E. Caesarean delivery	10 cases

Sample collection

Venous blood samples were collected from mothers at term, during first, second and third stages of labour and cord blood samples were collected from umbilical artery of the new born babies. In cases of caesarean delivery the samples were collected during first stage of labour before intravenous glucose infusion and then during delivery of the baby.

Blood glucose was estimated by the method of Asatoor and King as described by Varely (1965), lactic acid by the method of Barker and Summerson (1941) and serum free fatty acid by the method of Datta and Chakrabarty (1969).

Results

The results of glucose, lactic acid and free fatty acid levels found in maternal blood and cord blood of different groups are summarised in Table I.

It was found that glucose, lactic acid and free fatty acid levels in the maternal blood show a progressive increase from the term period to first, second and third stages of labour in all groups. No significant difference in the pattern of increase of these metabolites was found among different groups. However, the increase in blood glucose level was found to be more marked in cases of caesarean delivery than other groups.

The mean glucose level in cord arterial blood in all cases was found close to its maternal blood level of the second stage of labour. The cord blood lactic acid concentration was found to be about 1.5 to 2 times higher than its maternal concentration in all groups, whereas free fatty acid level in cord blood was found to be about 2-3 times decreased as compared to the maternal blood levels.

The period of gestation and parity of mother were not found to affect the levels of these three metabolites in maternal as well as in foetal blood during labour.

The condition of baby at birth was found to be related to the lactic acid concentration during delivery. In general, cases associated with foetal distress and low Apgar score of newborns showed appreciably increased lactic acid concentration. No such correlation was observed between glucose and free fatty acid levels and the condition of baby at birth.

Discussion

The present work was carried out to study the changes in carbohydrate and lipid metabolism during stress of labour. The blood levels of their main metabolities viz. glucose, lactic acid and free fatty acids were determined at term, during different

TABLE 1
COMPARATIVE VALUES OF BLOOD GLUCOSE, SERUM LACTIC ACID AND SERUM FREE FATTY ACID CONCENTRATIONS (MEAN + SD; MG/100 ML) IN MATERNAL VENOUS AND CORD ARTERIAL BLOOD DURING LABOUR IN DIFFERENT GROUPS

Labour stage	*Normal labour (A)*			*Normal preterm labour (B)*			*Postmature induced labour (C)*			*Forceps delivery (D)*			*Caesarean delivery (E)*		
	Gluc	*L.A.*	*F.F.A*	*Gluc*	*L.A.*	*F.F.A.*	*Gluc*	*L.A.*	*F.F.A*	*Gluc*	*L.A.*	*F.F.A*	*Gluc*	*L.A.*	*F.F.A*
At term	69.9 ±6.5	12.9 ±1.9	0.717 ±0.126	-	-	-	69.1 ±6.0	13.6 ±1.3	0.954 ±0.284	79.0 ±8.2	13.2 ±1.3	0.856 ±0.120	74.6 ±5.4	12.1 ±1.0	0.877 ±0.122
First	73.1 ±7.2	13.9 ±2.2	0.932 ±0.247	70.0 ±8.0	13.3 ±2.8	0.770 ±0.199	72.7 ±4.6	14.2 ±1.1	1.011 ±0.100	81.8 ±8.7	14.9 ±1.5	0.997 ±0.151	80.4 ±5.7	14.2 ±1.4	1.167 ±0.252
Second	81.8 ±8.7	17.1 ±2.6	1.130 ±0.267	78.4 ±9.0	14.6 ±3.3	0.998 ±0.110	80.0 ±6.0	16.2 ±1.6	1.239 ±0.273	90.5 ±7.6	20.4 ±3.2	1.606 ±0.398	110.1 ±12.6	15.4 ±1.4	0.997 ±0.171
Third	87.6 ±8.7	17.6 ±2.6	1.320 ±0.285	87.0 ±9.0	15.4 ±3.6	1.044 ±0.104	85.1 ±7.8	16.7 ±1.6	1.259 ±0.267	95.7 ±5.7	22.0 ±2.8	1.368 ±0.390	-	-	-
Cord	80.7 ±8.5	27.1 ±4.4	0.388 ±0.155	79.7 ±7.9	24.4 ±6.4	0.347 ±0.064	80.0 ±6.0	23.4 ±1.6	0.351 ±0.091	87.2 ±8.7	43.5 ±3.7	0.546 ±0.217	105.0 ±12.8	27.0 ±2.7	0.338 ±0.067

Gluc = Glucose
L.A. = Lactic acid
F.F.A. = Free fatty acid.

stages of labour and in the cord arterial blood at the time of delivery to evaluate the changes and their significance in relation to the foetal well being.

During labour the progressive rise of blood glucose was observed in all the cases of different groups. The rise was more marked in cases of caesarean delivery than other groups. The mean blood glucose concentration in umbilical cord was found to be close to the level of maternal blood glucose at the second stage of labour. This shows that there is no significant difference between maternal and foetal blood glucose concentration at the time of delivery. Increased glucose concentration has also been reported by Dahl (1928), Dancis (1962), Whaley et al (1967), Fairweather (1971) and Kashyap et al (1976).

Various factors have been suggested for the rising trend of blood glucose level in maternal blood during labour. Dahl (1928) attributed the rise to a reflex from the uterus, cervix and vagina, which by Claude Bernard's centre and splanchnic nerves causes glycogenolysis in liver resulting in a rise in blood glucose concentration. Ketteringham and Austin (1938) suggested that this rise may be influenced by other factors such as rate of secretion of insulin and epinephrine, severity of stress of labour, available glycogen store and the previous diet. In our opinion the stress of labour which undoubtedly affects various hormones responsible for the maintenance of blood glucose level in the body seems to be the main factor for the progressive rise of glucose level.

The lactic acid concentration in maternal blood was also found to show an increasing trend during different stages of labour in all the cases. The rise was more marked in cases of forceps delivery having foetal distress. In cord arterial blood at the time of delivery the level was found to be about two times higher than its concentration in maternal blood, it was more so in cases of foetal distress. Increased level of lactic acid in maternal and cord blood is also reported by other workers viz. Bell (1928), Eastman and Melane, (1931), Loeser (1932) and Hendricks (1957). Different sources for the increased production of lactic acid have been suggested by different workers. Bell (1928) suggested that foetus is the main source of increased lactic acid. Loeser (1932) held placenta to be responsible for it. Hendricks (1957) assumed that increased maternal formation, increased formation by uterus and/or its contents and delay in re-conversion of lactic acid back to glycogen by maternal structures may be responsible for such a rise. In his "uterine flush out studies" he concluded that with a vigorous uterine contraction blood supply to uterus is temporarily shut off, resulting into a transient hypoxia to the foetus, which changes its metabolism from aerobic to anaerobic thus causing an increase in lactic acid formation. He further observed that lactic acid concentration was found to be higher in cord arterial than cord venous blood, indicating that foetus rather than placenta is the source of increased lactic acid formation during labour. Weil et al (1965) suggested that increase in blood lactic acid in mother is the consequence of simultaneous increase in blood glucose and free fatty acid concentration brought about by adrenaline.

The maternal serum free fatty acid concentration during labour was found to increase in all cases of normal vaginal

deliveries who did not have glucose infusion. There was a progressive rising trend of maternal free fatty acid concentration through various stages of labour, an observation also noted by other workers (Peters et al, 1951, Burt 1960, Kashyap et al 1976).

This rise in maternal serum free fatty acid levels during labour have been attributed to various factors such as the stress of labour causing increased secretion of epinephrine (Weil et al, 1965, Whaley et al 1967, De Alvarez et al 1967). Sabata (1963) described starving during labour to be a factor causing a rise in serum free fatty acid levels in the mother.

Randle et al (1963) and Drazancis and Stavlenic (1971) found higher free fatty acid levels in diabetic pregnant women than others suggesting that free fatty acid concentration depends upon glucose metabolism.

A free fatty acid mobilizing substance of placental origin has been detected in the blood of pregnant women (Bleicher et al 1964). This substance has been thought to aid in the release of free fatty acid. It reduces maternal blood glucose consumption by increasing plasma free fatty acid concentration and/or by directing inhibition of glucose uptake, allowing a preferential shunting of glucose to foetus. It's amount is directly proportional to the concentration of maternal and foetal blood glucose concentration.

In the present study the cord arterial blood was found to have much lower cocentration of free fatty acid than its concentration in maternal blood at the time of delivery. It was about one third of its maternal blood concentration during second and third stages of labour. Fairweather (1965) also found the maternal and foetal free fatty acid ratio between 1.7 : 1 and 3.9 : 1 at the time of delivery. In his opinion this decrease in foetal free fatty acid and concentration could be due to its utilization by conversion into fat, keeping its turnover rate slower than glucose which is the main source of energy in the foetus.

As regards the relation of foetal well being to the levels of these three biochemical parameters it was found that blood lactic acid levels of mothers having babies with a low Apgar score were higher than those having babies with a score towards normal. No such correlation was observed between maternal blood glucose and free fatty acid levels and foetal well being. It may be concluded from this study that a rising level of maternal blood lactic acid concentration during labour could be an important parameter for the assessment of foetal distress.

References

1. *Barker, S.B. and Summerson, W.H. : J. Biochem, 38 : 535, 1941.*
2. *Bell, Blair : Birt. Med. J. 1 : 126, 1928.*
3. *Bleicher, S.J., Maldow, C.F., Scherrer, J. and Goldner, M.G. : Metabolism, 13 : 583, 1964.*
4. *Burt, R.L. : Am. J. Obstet. Gynaec. 80: 965, 1960.*
5. *Burt, R.L. Leake, N.H. and Dannenburg, W.N. : Am. J. Obstet. Gynaec. 84 : 1081, 1962.*
6. *Dahl, P. : Acta Obstet. et. Gynaec. Scandinav, 7 : 363, 1928.*
7. *Dancis, J. : Am. J. Obstet. Gynaec. 1749, 1962.*
8. *Datta, C.K. and Chakrabarti, B.K. : Indian J. Med. Res. 57 : 1309, 1969.*
9. *DeAlverez, R.R. Goodell, B.W. and Zighelboim, I. : Am. J. Obstet. Gynaec. 97 : 419, 1967.*
10. *Drazancis, A. and Stavlenic, A. : Am. J. Obstet. Gynaec. 109 : 666, 1971.*
11. *Eastman, N.N. and McLane, C.M. : Bull John Hopkins Hosp. 48 : 261, 1931.*

12. *Fairweather, D.V.I. : J. Obstet. Gynaec. Brit. C.Wealth 78 : 707, 1971.*
13. *Hendricks, C.H. : Am J. Obstet. Gynaec. 73 : 492, 1957.*
14. *Kashyap, M.L. Sivasamboo, R. and Sathy B.P. : Metabolism 25 : 865, 1976.*
15. *Ketteringham, R.C. and Austin B.B. : Am. J. Med. Sci. 195 : 318, 1938.*
16. *Loesey Alfred : Arch. Gynak, 148 : 118, 1932.*
17. *Peters, J.P. Heinemann, M. and Man, E.B. : J.Clin. Invest 30 : 383, 1951.*
18. *Randle, P.J. Garland, P.H., Halen, C.N. and New Sholme, E.A. : Anaesthesia 785, 1963.*
19. *Sabata, V. : Cesk, Gynekol, 28 : 466, 1963.*
20. *Varley, H. : Practical clinical biochemistry, 4th Ed. Arnold Heinemann Publishers (India) Pvt. Ltd. 1965.*
21. *Weil, R., Hua, P.P. and Attzzuler, N. : Am. J. Physiol 208 : 287, 1965.*
22. *Whaley, W.H. Zuspan, F.P. and Nelson, G.H. : Am. J. Obstet. Gynaec. 94 : 419, 1966.*
23. *Whaley, W.H., Zuspan, F.P., Nelson GtH. and Ahlquist, R.P. : Am. J. Obstet. Gynaecol, 97 : 875, 1967.*

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