

ORIGINAL PAPER

# A Comparative Study of Blood Glucose Level Amongst the Normal Birth Weight and Low Birth Newborns

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## ABSTRACT

The aim of the study is to estimate serum glucose level in normal birth weight or adequate for gestational age (AGA) and low birth weight or small for gestational age (SGA) newborns and to observe incidence of hypoglycaemia in SGA newborns within first 24 hours after birth. A cross sectional study was carried out for 1 year. 32 LBW (SGA) newborns and 49 NBW (AGA) newborns were taken for the study. Newborns weighing <2.5kg were taken as LBW. Preterm baby, baby of diabetic and toxemic mother, twins and baby of birth weight <1.5kg were excluded. Blood sugar investigation was done at different time period for the first 24 hour. Four sample of each newborn was taken for investigation. A little amount of umbilical cord blood obtained from the cut end of the cord, was the first sample taken at zero hour. Then next sample was taken at 2<sup>nd</sup> hour after the initial feed, then again before feeding at 6<sup>th</sup> and 24 hours after birth respectively. The method used is Glucose Oxidase method. To make a comparison of blood glucose level in both NBW and LBW newborns, the P values are calculated by using Z test. The operational threshold for hypoglycaemia is currently believed to be a blood glucose value of <40 mg/dl, From my study it is found that in both the groups of newborns, lowest blood glucose value is found at the 2<sup>nd</sup> hour and the highest number of newborns having low blood glucose value is found at the 2<sup>nd</sup> hour. The mean serum glucose values of SGA babies were less than that of AGA babies at zero, 2<sup>nd</sup>, 6<sup>th</sup> and 24<sup>th</sup> hour after delivery and the difference is significant. From my study it can be concluded that determination of blood glucose in those newborns which are at greater risk for developing hypoglycaemia in first

24 hours of life is very important as prolonged neonatal hypoglycaemia is associated with a risk of long term neuro-developmental sequelae.

**Keywords:** Hypoglycaemia, adequate for gestational age, small for gestational age, glycogenolysis, gluconeogenesis

## INTRODUCTION

Glucose is the major source of energy for foetus and neonate. It is an immediate source of energy. A total 38 moles of ATP is formed for each mole of glucose metabolized by the cells.<sup>1</sup>Upto 90% of total glucose used is consumed by the brain. The usual rate of glucose utilisation is 4-8 mg/kg/minute.<sup>2</sup> Glucose passes to the interior of the cells by the mechanism of facilitated diffusion. The amount of glucose that can diffuse to the inside of the most cells of the body in the absence of insulin, with the exception of liver and brain cells, are far too little to supply the amount of glucose normally required for energy metabolism.<sup>1</sup> It means cerebral glucose uptake is dependent on blood glucose concentration, not on insulin. Glucose regulatory mechanisms are sluggish at birth. Hypoglycaemia is said to develop when serum

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glucose concentration in full term infant is <30 mg/dl as the plasma glucose concentration observed in relatively large population of healthy full term infants, >1.7 mmol/L (>30 mg/dl) in the first 24 hours of life and >2.5 mmol/L (>45 mg/dl) after 24 hours.<sup>3</sup> But the operational threshold for hypoglycaemia is currently believed to be a blood glucose value of <40 mg/dl. Small for gestational age babies are the risk factor for neonatal hypoglycaemia. SFD or LBW babies develop hypoglycaemia due to decreased tissue glycogen stores, decreased gluconeogenesis, hyperinsulinism, increased glucose needs of hypoxia, hypothermia and large brain. Chronic foetal glucose deficiency in IUGR foetuses leads to cell cycle arrest of the pancreatic  $\beta$  cells and less secretion of insulin.<sup>7,8</sup> Chronic glucose deprivation in the IUGR foetus produces competing metabolic changes of increased capacity for glucose utilization and a tendency to hypoglycaemia. Hypoglycaemic infants may not always be symptomatic. Therefore routine monitoring for at risk infants is mandatory.

**Screening:** Low birth weight babies are prone to develop hypoglycaemia especially in first 24 hours of life, as transition from intrauterine glucose regulation to extra uterine adaptation develops in first 24 hours of age.<sup>10</sup> So blood test for glucose level is done in zero, 2<sup>nd</sup>, 6<sup>th</sup> and 24 hours after delivery.

## MATERIALS AND METHOD

A cross sectional study was carried out in the department of physiology of Gauhati Medical College. A total of 81 term newborns were taken out of which 49 were NBW and 32 were LBW. Birth weight is defined as normal birth weight with weight between 2.5 kg and 3.9 kg and low birth weight with weight < 2.5 kg. The term AGA and SGA were defined according to intrauterine growth curves based on percentile of birth weight.<sup>11</sup> Thus newborns were taken between 1.5 – 3.9 kg body weights and delivered between 37 completed weeks and 42 weeks of gestation. A simple random sampling method was followed. Newborns were taken from obstetrics and gynaecology, and paediatric department.

**Exclusion Criteria:** Preterm baby, baby of diabetic and toxæmic mother, twins and baby of birth weight <1.5 kg were excluded.

A detailed history including maternal history and history on delivery was taken. Examination of baby was done to

see the weight and for signs of hypoglycaemia.<sup>12</sup> Blood sugar investigation was done at different time period for the first 24 hours. Four venous blood samples of each newborn were collected. The first sample which was taken from the cut end of the umbilical cord immediately after delivery was taken as zero hour. Then next sample was taken at 2<sup>nd</sup> hour after birth, after the initial feed, then again before feeding at 6<sup>th</sup> & 24<sup>th</sup> hour after birth. The method used is Glucose Oxidase Method. Sample blood was allowed to clot at room temperature, undisturbed for half an hour. When the blood firmly clotted, and started to retract, the sample may be left in a refrigerator overnight at four degree centigrade, so that the clot retraction was complete and unfavourable for the growth of bacteria. If there is delay in refrigeration then the glucose level of the sample may drop at a rate of 15 -20 mg / 100 ml /hr. In some samples the clot fails to retract, it was then gently detached from the sides of the vial with a platinum wire. Extreme care was taken to prevent haemolysis. Serum obtained was straw coloured, undiluted and was transferred to fresh plastic vial. It was then subjected to centrifugation at 3000 rpm for 15 min. The samples obtained were strictly labelled; hence it was ready for final glucose estimation by Glucose Oxidase Method.

## RESULT AND OBSERVATION

**Table 1** shows the difference in serum glucose level in different time period. From the table it is seen that serum glucose level is lowest in 2<sup>nd</sup> hour and the mean glucose values (mg/dl) of 81 newborns are within 95% confidence interval.

**Table 1** Comparison of serum glucose level (mg/dl) of newborns according to different time period

Time Period (Hours)	Serum glucose level (mg/dl)		
	Mean	S.D.	95% C.I.
0	65.06	14.40	36.26,- 93.86
2	48.67	13.19	22.29, 75.05
6	54.78	13.57	27.64, 81.92
24	73.89	16.75	40.39, 107.39

**Table 2** shows the lowest blood glucose level in different time period in first 24 hours of life. Overall prevalence of hypoglycaemia is 70 out of 81 term newborns in first 24 hours of life. From this table it is seen that the maximum number of low blood glucose value, which is < 40 mg/dl is seen after 2<sup>nd</sup> hour of delivery.

**Table 2** Number of newborns having lowest blood glucose level during the first 24 hours

Time period (hours)	No. of newborns	
	Blood glucose values <40 mg/dl	Blood glucose values <30 mg/dl
0	6	1
2	42	1
6	22	0
24	No	No

**Table 3** shows the difference in mean serum glucose level between NBW (AGA) and LBW (SGA) by different time period. From this table it is seen that, the mean serum glucose value of LBW babies at zero, 2<sup>nd</sup> and 6<sup>th</sup> hour is less than that of NBW baby, which is statistically significant as  $P < 0.05$  but it is highly significant after 2<sup>nd</sup> hour.

**Table 3** Comparison of mean serum glucose levels amongst the newborns of AGA and SGA by different time periods

Time period (hour)	Serum glucose level (mg/dl) of AGA of babies (n=49)		Serum glucose level (mg/dl) of SGA of babies (n=32)		z-value
	Mean	S.D.	Mean	S.D.	
0	72.65	16.60	53.44	10.40	6.40
2	55.77	15.90	37.80	7.59	6.81
6	60.35	15.40	46.26	10.40	4.91
24	80.46	17.90	63.84	15.09	4.49

## DISCUSSION

Neonatal hypoglycaemia is a common metabolic disease<sup>13</sup> because of inability to maintain glucose homeostasis. The overall prevalence depends on birth weight, gestational weight, and intrauterine growth retardation. Symptomatic hypoglycaemia is not common (1- 3 per 1000 live births) as against chemical hypoglycaemia (67% in preterm small for gestational age to 4% in term appropriate for gestational age).<sup>14</sup> Undiagnosed hypoglycaemia can have long term neurological consequences; thus the emphasis is on prevention and early detection along with treatment of asymptomatic hypoglycaemia. From table 1, it has been observed that serum glucose level is 48.67 mg/dl in 2<sup>nd</sup> hour of life, which is the lowest level in the first 24 hours. So, from my study it can be said that in 2<sup>nd</sup> hour of life glucose production in the body is low.<sup>15</sup> Studies using radioisotope tracer techniques in humans<sup>16</sup> have demonstrated that

the fatal glucose supply is derived from the mother with no endogenous glucose production in the fetus. The time of birth is marked by an abrupt change from a high carbohydrate low fat diet to a high fat low carbohydrate diet.<sup>17</sup> So the neonate must make the transition from an environment of continuous glucose supply from the placenta to one of the intermittent periods of feeding and fasting.

From **Table 2**, it has been observed that the number of newborns having blood glucose value < 40 mg/dl is 6, at zero hour, which reflect maternal blood glucose level, 42 at 2<sup>nd</sup> hour before feeding, and 22 at 6<sup>th</sup> hour and no case of hypoglycaemia found after 24 hours. It means maximum number of low glucose value is seen in 2<sup>nd</sup> hour. After 2<sup>nd</sup> hour blood glucose level gradually increases. So from my study it can be assumed that low blood glucose level in 2 - 3 hours of life is physiological<sup>18,19</sup> and that the newborn has unique physiologic adaptation to low blood glucose level.<sup>20,21</sup> This metabolic adjustment occurs, when the blood glucose value falls to a level, which is unable to fulfil body's requirements. The body makes glucose available in the fastest state by counter regulation, which occurs either by glycogenolysis or gluconeogenesis. Counter regulatory hormones become important if blood glucose level continue to fall: glucagon release is stimulated followed by release of epinephrine, cortisol<sup>22</sup> and growth hormone.<sup>23</sup> Epinephrine and Glucagon both increase cAMP in hepatocytes which activate protein kinase A, which causes increase glycogen breakdown and also inhibit glycogen synthesis.<sup>24</sup>

**Table 3** shows that mean serum glucose value in LBW (SGA) baby is lower than NBW (AGA) baby at zero, 2<sup>nd</sup>, 6<sup>th</sup> and 24 hours of delivery. Several studies show that SGA infants when compared to AGA infants have increased plasma concentration of lactate and alanine with in first 24 hours of life.<sup>25</sup> After that these substrates fall and become lower than AGA infants. The elevated concentration of gluconeogenic substrates reflect delayed maturation of gluconeogenic pathways in SGA infants due to delay in the development of a rate limiting hepatic gluconeogenic enzyme.<sup>26</sup> SGA newborns have greater brain weight.<sup>27</sup> In SGA babies liver weight is much reduced whereas brain weight remains within normal limits, so that the ratio of brain weight to liver weight is > 5. This discrepancy in the size of the utilize that is brain and liver that is liver glycogen of glucose causes low glucose values leading to hypoglycaemia.

## CONCLUSION

So, from my study it can be concluded that determination of blood glucose in newborns which are at greater risk for developing hypoglycaemia in the first 24 hours of life, mainly LBW (Small for date baby) is very important. Others who are also at risk for developing hypoglycaemia are preterm babies, moderate to severely asphyxiated babies, babies of diabetic mothers and toxemic mothers. Asymptomatic hypoglycaemias which develop in the first 24 hours of life are mainly physiological. Management of hypoglycaemia by feeding is widely used for asymptomatic hypoglycaemia which is usually successful. The most effective method of preventing hypoglycaemia is early breast feeding. Early intervention by early diagnosis of hypoglycaemia subsequently prevents brain damage or, any neurological complication decreasing the number of physically disabled children in our country. So, an approach aimed at reliable detection of hypoglycaemia at risk, prevention and appropriate treatment is very essential.

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**Ethical clearance:** Taken

**Conflict of interest:** None

**Contribution of Authors:** We declare that author(s) named in this article did this work and all liabilities pertaining to claim relating to the content of this article will be borne by the authors. The study was conceived and designed by Dr. Chinmayee Sarma Bhargav, who also collected and analyzed the data. Dr. Biju Dutta Choudhary, Dr. Dulal Kalita, Dr. Ankumoni Saikia and Anjanamoyee contributed to analyze the data and designing the manuscript.

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