



## EVALUTION OF INTRAVENOUS BLOOD GLUCOSE LEVEL AND ITS METABOLISM IN NON DIABETIC PATIENTS

**Dr. Nandkishore K. Agrawal**

Professor, Dept. Of Anesthesiology JNM C Wardha

**Dr. Kiran Bhatia\***

Associate Professor in SHKM Govt. Medical College, Nalhar, Mewat, Haryana  
\*Corresponding Author

### ABSTRACT

**Aims and objective:** The aim the study was to monitor how blood glucose level changes during the period of infusion of dextrose containing fluid. To study the change in BGL and time required for blood glucose level to come to the baseline level when dextrose containing fluid is given. This study will be helpful in the patients who are diabetic and the BGL monitoring is required intra operatively.

**METHOD:** 25 gm glucose was given to 30 patient posted for surgery under regional anaesthesia, in pre operative room blood glucose level was done, than the patients were given dextrose containing 25 gm glucose over fifteen minute and blood glucose level were checked at 15, 30, 60, and 90 minutes.

**RESULT:** It was observed that at 15 minute the blood sugar level rose by 56% average of  $153.37 \pm 19.77$  from  $96.00 \pm 10.82$  with P value < 0.0001 extremely statistically significant, at 30 minute it was  $139.66 \pm 15.89$  high by 43 %, at 60 minute it was  $111.09 \pm 7.77$  higher by 15 % which came down to  $97.50 \pm 7.84$  at 90 minutes P value = 0.6968 statistically not significant.

**CONCLUSION:** It was concluded that the intravenous dextrose infusion raises the blood glucose level significantly which may give false BGL if we are monitoring BGL, we should avoid measuring blood glucose level up to 90 minutes when a glucose containing fluid is being given.

**KEYWORDS:** Blood glucose level, dextrose, intravenous, intra operative

### INTRODUCTION

Glucose is an important energy source for body, cells like brain cells, red blood cells, liver cells and adrenal medulla, depend on glucose for energy. In routine surgeries it is required to give intra venous fluid containing glucose to prevent hypoglycaemia intra operatively. Use of glucose containing fluid has been a matter of debate. Many researchers like to infuse glucose while few are against it. [1,2,3]

When the fluid containing glucose is being given, how it influences the blood glucose level, how much time is required to metabolise the glucose have been studied less often. When glucose containing fluid is given in diabetic patients, it requires more consideration. Blood glucose level are to be maintained between 100 mg/dl to 140 mg/dl to have best possible result post operatively. Decrease in blood glucose level below 60 mg/dl or increase in blood glucose level above 200 mg/dl affects the metabolism significantly. [2,3]

We need to do blood glucose level intra operatively when a glucose containing fluid is being given. This may increase the blood glucose level, which may also give hyper inflated BGL which may misguide the management. [4]

Glucose is an important source of energy, when it is given intravenously, it raises blood glucose level which takes some time to come to baseline level, if a dextrose containing fluid is running or given few minutes before, it may affect the blood glucose monitoring. In this proposed study we tried to study the change in blood glucose level and time required to return to the baseline blood glucose level when given intravenously.

### Methodology:

Randomly 30 patients posted for surgery, ASA Grade I or II were selected. The patients were explained regarding the study and consent was obtained accordingly. Patients who were diabetic or not willing were excluded. Ethical committee approval was obtained duly before the study started.

All the patients having diabetes or whose blood glucose was > 200 mg/dl before surgery were excluded. Only the patients posted for surgery under regional anaesthesia were included. Baseline Blood glucose level was done before the fluid started, it was taken as zero

hour. An intra venous line secured with 20 G intra cath and 5% dextrose containing 25 gm Glucose given intravenously over a period of 15 minutes. There-after, blood glucose level were done at 15, 30, 60 and 90 minute.

### Observations

**TABLE 1 DEMOGRAPHIC AND BASELINE CLINICAL PROFILE**

Variables	Group n=30 Mean±SD	P value	significance
Age (years)	46.02± 8.07	P>0.05	Not significant
Height (cm)	162.50 ± 6.34	P>0.05	Not significant
Weight (kg)	54.77 ± 8.05	P>0.05	Not significant
Duration of surgery (min)	202.90 ± 57.41	P>0.05	Not significant
FBS (mg/dL)	75.65 ± 6.39	P>0.05	Not significant
PPBS (mg/dL)	90.53±7.93	P>0.05	Not significant
CBG 0 (mg/dL)	84.02±12.27	P>0.05	Not significant

**TABLE 2 ACHIEVED AVERAGE BLOOD GLUCOSE LEVEL AND SIGNIFICANCE**

Average weight Kg	Average blood glucose level Mg/dl	Target blood glucose level Mg/dl	Average insulin required	Achieved average blood glucose level mg/dl	P value
70.70 ± 13.88	300.38 ± 45.55	160	2.8±1.1	164.97 ± 13.46	= 0.0528 not significant

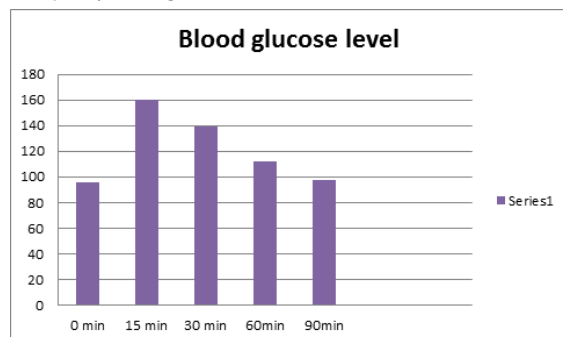
CBG values at 15 min, 30 min, 60 min and 90 min are significantly higher in group B than group A. But there is no significant difference between 120 min and 150 min. CBG values between the groups.

**TABLE 3 CONFIDENCE INTERVAL OF THE DIFFERENCE**

CBG	Sig		95% confidence interval of the difference	Upper	Lower
	Mean± SD	Median			
CBG 0 min	109.87±18.28	115.00	0.002	-13.137	-2.883
CBG 15 min	123.17±24.46	127.50	<0.001	-20.521	-7.739

<b>CBG 30 min</b>	132.16±22.81	132.00	<0.001	-19.998	-6.451
<b>CBG 60 min</b>	145.18±21.27	142.50	0.103		
<b>CBG 90 min</b>	138.0±118.0	139.00	0.075	-19.973	1.064

Group statistics for capillary blood glucose\_1, capillary blood glucose\_2, capillary blood glucose\_3, capillary blood glucose\_4 and capillary blood glucose\_5



We have observed that after the dextrose containing fluid is infused at 15 minutes the capillary blood glucose level rises up to  $153.37 \pm 19.77$  mg/dl from level of  $97.43 \pm 10.82$  raise by 56%, P value < 0.0001 extra statistically significant with 95 % confidence, which was  $139.66 \pm 15.89$  at 30 minutes which was higher by 43 % P value < 0.0002 statistically significant. The average blood glucose level at 60 minutes was  $111.09 \pm 7.77$  higher by 15%, P value < 0.6968 statically not significant, it came to  $97.28 \pm 7.84$  mg/dl after 90 minutes, P value < 0.2167 statically not significant.

## RESULT

We observed that at 15 minute the blood sugar level rose by 56% average of  $153.37 \pm 19.77$  from  $96.00 \pm 10.82$  with P value < 0.0001 extremely statistically significant which was higher up to 60 minute, came down to 97.50 at 90 minutes P value = 0.6968 statistically not significant.

## STATISTICAL ANALYSIS

Data was analyzed using SPSS 20 statistical package. A descriptive analysis was done on all variables to obtain a frequency distribution. The mean  $\pm$  SD and ranges were calculated for quantitative variables. Continuous variables were compared by the Student t test. Proportions were analyzed with the chi-square test. A P value of 0.05 or less was considered statistically significant

## DISCUSSION

The etiology of lower glucose tolerance was entirely different in lean and obese subjects. Lean, lower tolerance was related to pancreatic insufficiency (phi 2 77% lower than in good tolerance controls [P less than 0.03]), but insulin sensitivity was normal (P greater than 0.5). In contrast, obese lower tolerance was entirely due to insulin resistance (SI diminished 60% [P less than 0.01]); pancreatic responsiveness was not different from lean, good tolerance controls (phi 1: P greater than 0.06; phi 2: P greater than 0.40). Subjects (regardless of weight) could be segregated into good and lower tolerance by the product of second-phase beta-cell responsivity and insulin sensitivity (phi 2 . SI). Thus, these two factors were primarily responsible for overall determination of glucose tolerance. This study demonstrates the feasibility of the minimal model technique to determine the etiology of impaired glucose tolerance.[1]

Seeder F et al studied relation of glucose and brain. In this study they focused on the neurologic issues concerning the treatment of hypo or hyperglycemia in the critically ill patient. Moderate hypoglycemia may evoke a significant stress response, behavioral changes, and alterations in cerebral blood flow and metabolism. It is unclear what effect prolonged or repeated episodes of moderate hypoglycemia may have on patient outcome. Depending on its severity, hypoglycemia has varying influences on neurologic

damage after ischemia. Hyperglycemia may impair neuronal recovery following cerebral ischemia. However, the detrimental effects of hyperglycemia vary depending on the types of brain ischemia sustained (focal or global). Hyperglycemia during global and incomplete global ischemia events is detrimental to neurologic outcome. However, the relationship between hyperglycemia and outcome after focal ischemia is controversial. So they concluded that because both hypo- and hyperglycemia may produce neurologic changes, aggressive management of abnormal glucose values is warranted.[2]

Stangnaso et al studied perioperative glucose control. Perioperative management of the patient with diabetes presents unique challenges. The benefit of glycemic control must be balanced against the danger of hypoglycemia. They concluded that, through a clear understanding of the hormonal and metabolic alterations that accompany surgery, as well as careful glucose monitoring, the diabetic can be safely and effectively managed during the perioperative period.[3]

Bergman RN et al did physiologic evaluation of factors controlling glucose tolerance in man. They did measurement of insulin sensitivity and beta-cell glucose sensitivity from the response to intravenous glucose. The quantitative contributions of pancreatic responsiveness and insulin sensitivity to glucose tolerance were measured using the "minimal modeling technique" in 18 lean and obese subjects (88-206% ideal body wt). The individual contributions of insulin secretion and action were measured by interpreting the dynamics of plasma glucose and insulin during the intravenous glucose tolerance test in terms of two mathematical models.[4]

BRUNZELL JD et al studied relationships between fasting plasma glucose levels and insulin secretion during intravenous glucose tolerance tests. In their study, Insulin secretion and glucose disappearance rate were measured in 66 subjects with a wide range of fasting plasma glucose levels. The acute insulin response was present in subjects with fasting glucose levels below 115 mg/dl but was absent above this level. The glucose disappearance rate related to the relative acute insulin response in subjects with fasting glucose below 115 mg/dl and to total insulin response when fasting glucose levels were above 115 mg/dl. A calculated glucose disappearance rate of 1.06 per cent per minute was found when the acute insulin response was zero. All subjects with fasting glucose levels > 115 mg/dl had glucose disappearance rates < 1.06. These studies support 1) epidemiological data indicating 115 mg/dl as an upper limit of normal for fasting plasma glucose levels and 1.0 per cent per minute as a lower limit of normal for the glucose disappearance rate, and 2) evidence for an important role for the acute insulin response the determination of glucose disappearance rates during intravenous glucose tolerance tests.[5]

## Menu

Balentine JR (1998) in his study of Effect of 50 ml of 50 % dextrose in water administration on the blood sugar of euglycaemic volunteer observed that the BGL rose from  $82.3 \pm 13.5$  mg/dl to  $244.4 \pm 44.6$  which came to  $88.1 \pm 28.8$  mg/dl at 60 minute, similarly in our study we observed that the BGL rose by 53 % to  $153.37 \pm 19.77$  from a baseline level of  $96.00 \pm 10.82$ , which was higher up to 60 minute, and fell down to 97.50 at 90 minute, the baseline level. This type of increase in BGL may be highly significant in the patients who are diabetic, undergoing surgical procedure. If during the procedure BGL monitoring is needed, and the patient is being given dextrose containing fluid, one must keep this observation while calculating the dose of anti diabetic intravenous dextrose when given intravenously it raises the BGL by 56% from pre operative level.[6]

The rate of uptake of glucose only increased during the 4 mg/kg-min infusion, even though there were significant elevations in the plasma glucose and insulin concentrations during the 2 mg/kg-min infusion as well. The glucose clearance rate increased only when

sufficient insulin was infused with the 4 mg/kg-min glucose infusion to control the hyperglycemia that developed if no insulin was administered. Approximately 43% of the infused glucose was directly oxidized when the infusion rate was 1 or 2 mg/kg-min. That value fell to 32% when the infusion rate was increased to 4 mg/kg-min, regardless of whether insulin was infused or not.[7]

Similar study was done by Wolfe RR et al who studied glucose metabolism in man and responses to intravenous glucose infusion. The level of insulin after an overnight fast (basal) in 37 obese and nonobese male subjects with normal and abnormal carbohydrate tolerance was directly related to the increase in insulin concentration during a 3 hr 100 g oral glucose tolerance test. Obesity, but not diabetes, was associated with an elevation of this basal insulin level. Thus obesity predicted with the magnitude of the insulin response to glucose ingestion. When the individual insulin values were expressed as per cent change from the basal level, this effect of obesity was excluded. The insulin levels of all subjects with normal carbohydrate tolerance promptly rose 5-7-fold, and reached peak values 1 hr after oral glucose. In contrast, the diabetic response (as per cent increase) was markedly reduced during the 1st hr, and maximal (but still subnormal) insulin levels were not attained until 2 hr. In all subjects the insulin response (quantitated by calculation of the area circumscribed by a plot of the per cent change in insulin with time) showed a significant inverse correlation with the glucose response. Thus increasing degrees of carbohydrate intolerance were associated with decreasing insulin responses. Elevated levels of insulin, in both the basal state and in response to glucose, were related to obesity.[8]

Raskin P et al did a study on hyperglucagonemia and its suppression: importance in the metabolic control of diabetes. Replacement of glucagon raised glucagon to  $272 \pm 30$  pg per milliliter, glucose to  $202 \pm 20$  mg per deciliter, glucose excretion to  $14 \pm 7$  g per 24 hours, ketone excretion to 0.8 mmol per 24 hours and urea nitrogen excretion to  $11 \pm 2$  g per 24 hours. In a subsequent study, similar improvement occurred on a diet of 30 g of carbohydrate daily, when absorption of dietary glucose was negligible. Therefore the authors concluded that hyperglucagonemia has an important role in diabetes; its correction reduces diabetic abnormalities to or toward normal.[9]

Berg AH et al studied adiponectin: an adipokine regulating glucose and lipid metabolism. Adipocytes are not merely inert storage depots for triglycerides but rather highly active cells with potent autocrine, paracrine and endocrine functions. Adipose tissue secretes a large number of physiologically active polypeptides. A strong correlation between plasma Acrp30 levels and systemic insulin sensitivity is well established and the protein has putative anti-atherogenic properties that are relevant for the prevention of formation of atherosclerotic plaques. Their challenge was to understand the molecular mechanisms through which the protein exerts its multiple functions.[10]

## CONCLUSION

Here we conclude that the intravenous dextrose infusion raises the blood glucose level significantly which may give false BGL, we should avoid measuring blood glucose level up to 90 minutes when a glucose containing fluid is being given.

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