Blood Glucose in High Risk Newborn Infants

Thesis

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By

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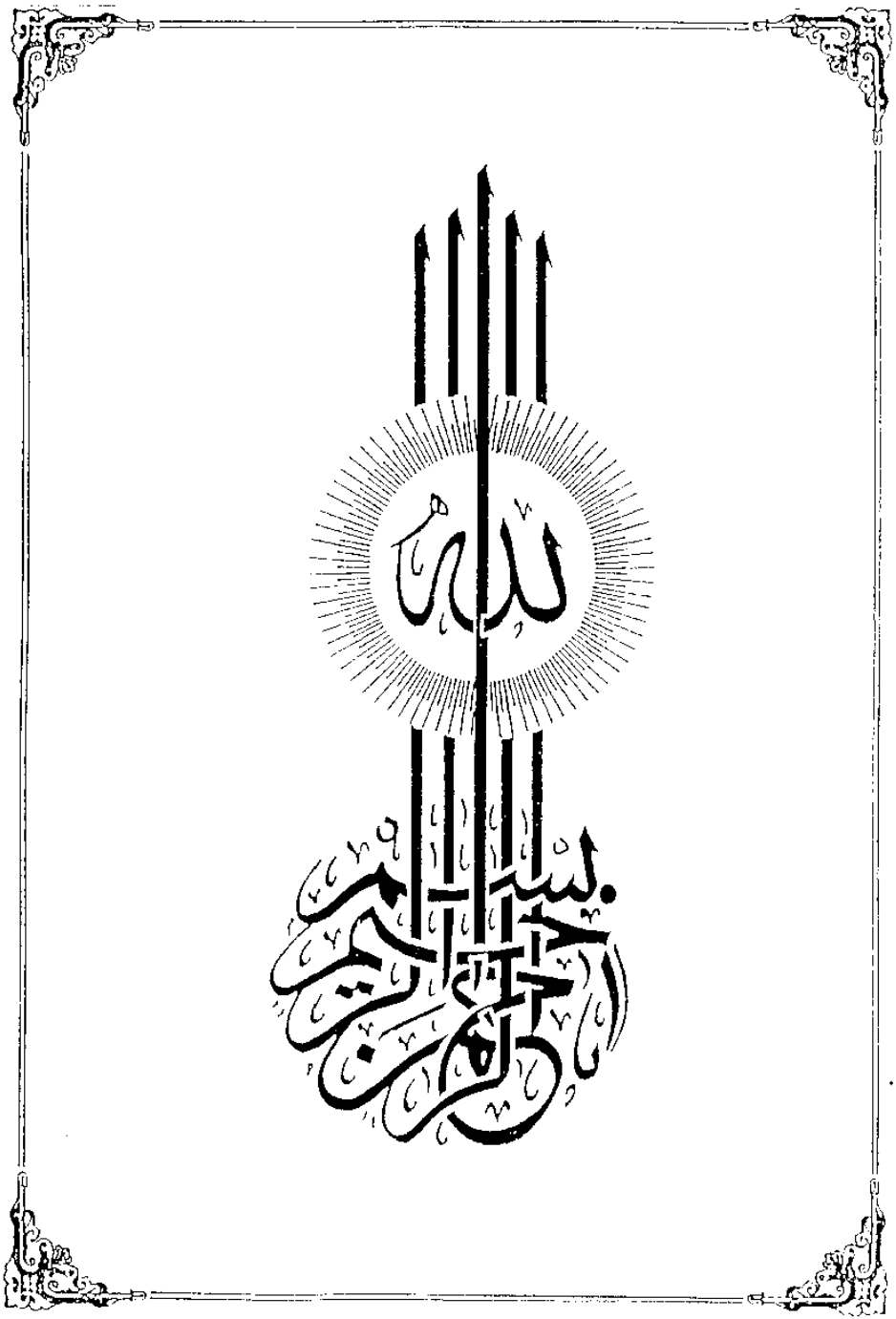
To ...

My Nephews ,

Diana, Moh. A, Heba, Ahmed, Moh. S.,

Jasmin and Lotfy Jr.



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Abbreviations Used

FFA	: Free fatty acids
CNS	: central nervous system
AGA	: Appropriate for gestational age
SGA	: Small " " "
LGA	: Large " " "
LBW	: Low birth weight
I.M.	: Intramuscular
I.V.	: Intravenous
S.C.	: Subcutaneous
Hr.	: hour
Min.	: minute
IDM	: Infant of diabetic mother
F.T.	: full term
C.S.	: caesarean section
RDS	: Respiratory Dystress Syndrome
NICU	: Neonatal Intensive Care Unit

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C O N T E N T S

CHAPTER 1

INTRODUCTION AND AIM OF THE WORK

Introduction:

During fetal life the major source of glucose is derived from the mother. After delivery the baby is suddenly deprived from that major source and must rely on endogenous sources of glucose untill feeding starts. Maintainance of glucose homeostaisis during that period requires some major physiologic alterations. This might be easy for normal neonates but it isn't for the high risk ones hence there is an added potential risk of hypoglyceamia.

Because glucose is the major metabolic feuel for the brain, so faliure to recognise and treat neonatal hypoglyceamia as early as possible may result in serious neurological and psychological sequelae and might lead to death.

Infants in the high risk category include those:

(1) Born before 37 or after 42 weeks gestation.

(2) Weighing less than 2500 or more than 4000 gm.

(3) Deviating from expected size or development e.g. low or very high for gestational age.

(4) With a history of serious neonatal illness or death of a silbing or of more than 2 fetal deaths of silbings.

(5) In poor condition at delivery, (Apgar 0-4 at 1 min.) or requiring resuscitation in the delivery room.

(6) Born to mothers who have infections or a history of any illness during pregnancy or premature rupture of the membranes; a history of severe social problem such as teen age pregnancy, drug addiction, or absence of mate, of absent or long-delayed prenatal care, of minimal or no weight gain during pregnancy, of prolonged infertility, or of 4 or more previous pregnancies; who are 35 yr or more of age (especially if primiparous); or who have a history of taking any of the medications known to affect the fetus adversely.

(7) Of multiple pregnancy or of a gestation commencing within 3 mo. of a previous pregnancy.

(8) Delivered operatively or with any unusual obstetric complication, including hydramnios, abruptio placentea, placenta previa or abnormal presentation.

(9) Having single umblical artery or any important malformation or suspicion of one.

(10) Being observed for anemia or blood group incompatibility.

(11) Born to mothers who have had stressful events during gestation such as severe emotional problems, hyperemesis gravidarum, serious accidents or general aneathesia.
(Behrman and Vaughan, 1983).

Aim of the Work :

To obtain further informations about blood glucose homeostasis in high risk newborn infants born in Egypt.

CHAPTER 2
REVIEW OF LITERATURE

GLUCOSE HOMEOSTASIS IN THE NEWBORN

During fetal life, the major source of glucose is derived from the mother by a facilitated passive diffusion across the placenta so that the fetal plasma glucose level closely approximates the maternal concentration (King et al., 1972).

Although little is known about the mechanisms of gluconeogenesis in the human fetus, information from animal studies indicates that certain key rate-limiting enzyme activities are low near the time of parturition and do not reach full activity until several hours to days after delivery (Pagliara et al., 1973).

Liver glycogen in the human fetus, as in many other species of animals, steadily increases until term at which time the level is about twice the adult concentration (Shelley, 1961).

The enzymes responsible for glycogenolysis, phosphorylase and glurose-6-phosphatase show low activity until very close to birth in rat and guinea pig livers, but increase rapidly at term and in the immediate postnatal period (Kornfeld et al., 1963).

After delivery, liver glycogen concentration decreases rapidly to 10% of it's original value within 2-3 hours and then rises gradually to reach the adult level by 2-3 weeks (Shelley, 1961).

The adult human being is capable of maintaining a normal blood glucose level even when totally deprived of calories for weeks. In contrast, the normal neonate and child exhibit a progressive fall in blood glucose to hypoglycemic levels when fasted for even short periods (Pagliara et al., 1973).

The close relationship between maternal and fetal plasma glucose, the frequent occurrence of hypo or hyperglycemia in the newborn and the relatively slow disappearance of a glucose load give the impression that control of carbohydrate metabolism is poorly developed in the perinatal period (Shelley et al., 1975).

The concentration of glucose in the umbilical venous blood approximates 70 to 80% of that in the mother. During the first four to six hours of postnatal life, glucose values fall, stabilizing between 50 and 60 mg/100 ml blood. By the third day, glucose values equilibrate at 60 to 70 mg/100 ml blood in

the full-term neonates and at lower levels in the low birth weight babies, it then increases to reach adult values by about the 10th day (Cornblath and Schwartz, 1976).

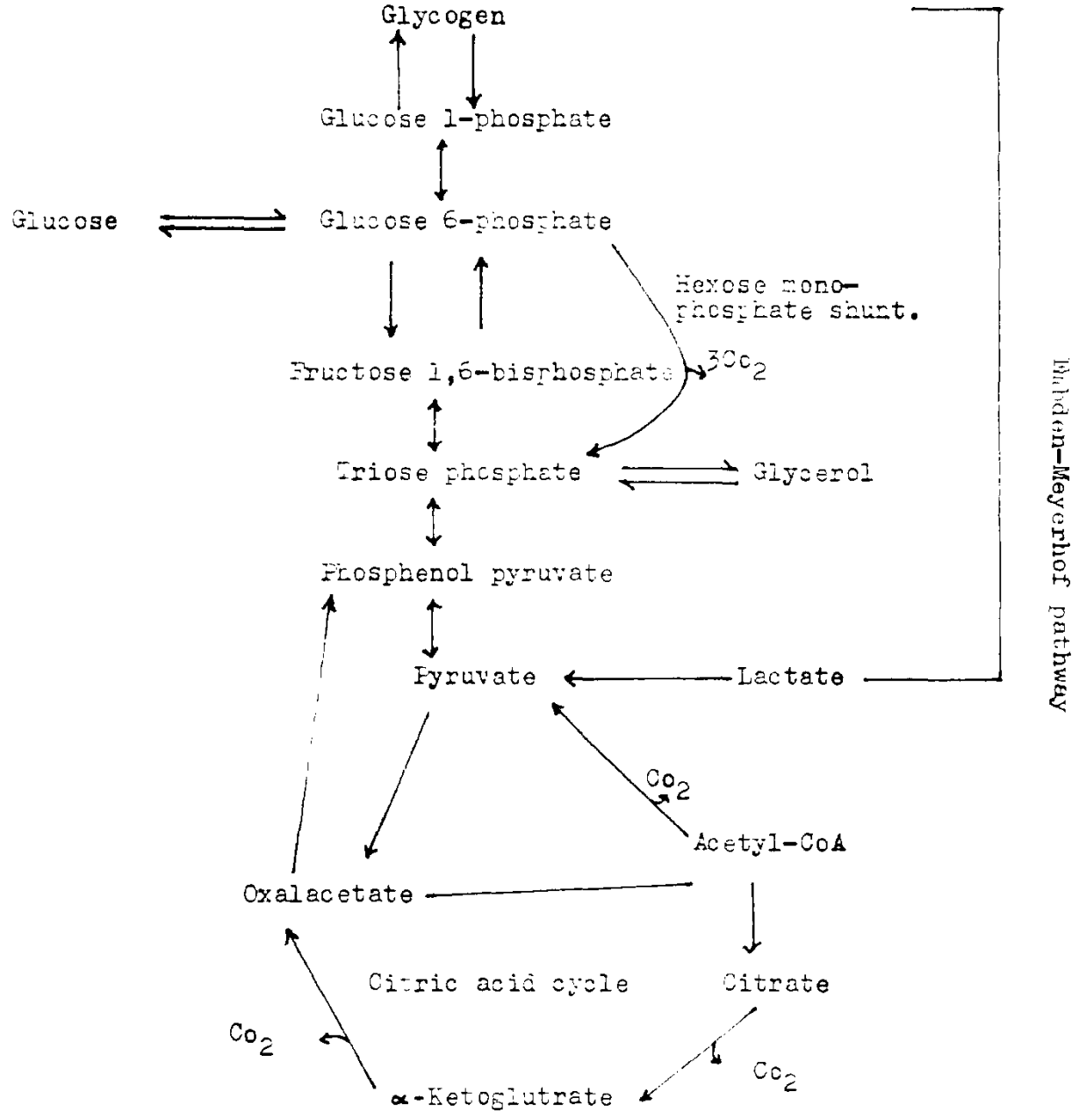
Blood glucose concentration is normally maintained at a relatively constant level by a fine balance between hepatic glucose output and peripheral glucose uptake. The latter is influenced by factors such as body temperature, muscular activity and insulin concentration (Fanaroff et al., 1983).

In the immediate postnatal period (i.e. 4 to 8 hours), glucose supply is derived primarily from hepatic glycogen stores, whereas with prolonged fasting the infant must depend upon the denovo synthesis of glucose (i.e., gluconeogenesis).

Associated with the great postnatal loss of hepatic glycogen which has been demonstrated in many species of animals, is great increase in the activity of phosphorylase enzyme while that of glycogen synthetase decreases, thus glucose production is enhanced (Kawai and Arinze, 1981). (Fig. 1, 2).

Glucagon and epinephrine increase the concentration of cyclic AMP and thereby stimulate conversion

Fig. (1): Major pathways of carbohydrate metabolism.



Quoted from Martin et al., (1962).