

# RED BLOOD CELL INDICES IN FULLTERMS VERSUS PRETERMS

Thesis

Submitted For Partial Fulfilment  
Of Master Degree In Pediatrics.

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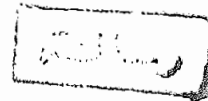
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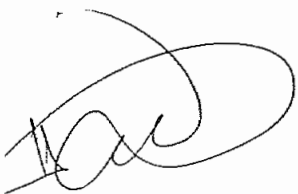
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## List of Abbreviations

AGA	: Appropriate For Gestational Age.
BFU-E	: Burst forming Unit Erythroid.
CD	: Clusters of differentiation.
CFU-E	: Colony forming Unit Erythroid.
CVS	: Cardiovascular system.
D.M	: Diabetes Mellitus.
EPO	: Erythropoietin.
ESF	: Erythropoietin Stimulating Factor.
FL	: Femto Litter.
Ges.Age	: Gestational age.
Hb	: Hemoglobin.
Hct	: Hematocrit.
IDM	: Infant of Diabetic Mother.
LBW	: Low Birth Weight.
m-RNA	: Messenger Ribonucleic acid.
MCH	: Mean Corpuscular Hemoglobin.
MCHC	: Mean Corpuscular Hemoglobin Concentration.
MCV	: Mean Corpuscular Volume.
PCV	: Packed Cell Volume.
PG	: Pico Gram.
REC	: Reticulo Endothelial Cells.
RES	: Reticulo Endothelial System.
SGA	: Small For Gestational Age.
SGA	: Small for Gestational Age.
TIBC	: Total iron binding capacity.

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# *Introduction*



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## **Introduction**

A preterm infant is a neonate who is born before 37 completed weeks of gestation regardless of birth weight while a full term neonate is a neonate who is born between 38 weeks and 42 completed weeks of gestation [W.H.O.1950]. The infant is considered small for gestation if its weight is less than 10th centile in weight expected for gestation, while it is appropriate for age if its weight lies between 10th and 90th centiles of weight expected for gestation. A low birth weight infant is that one born with birth weight below 2.5 kg [McIntosh, 1992].

Knowledge of the normal blood values during the dynamic period of growth is a prerequisite to the interpretation of a particular blood response in infancy and childhood. The hematologic values are subjected to wide variations, both in each individual and among members of an age group. [Miller, 1989].

Several variables influence the interpretation of what may be considered normal values for hemoglobin, hematocrit, red cell indices at the time of birth and during the early weeks of life [Nathan and Oski, 1992].

Iron stores at birth are influenced by gestation, birth weight and the extent of placental transfusion at delivery [Lucas A., 1992].

## *Aim of the Work*



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## ***Aim of the work***

The aim of this work is to study red blood cell indices and iron status at birth in preterms in comparison to full terms .



# *Review of Literature*

## ***Erythropoiesis***

The production of erythrocyte is modulated by a circuit of a stimulators including the circulating red cell mass and its precursors in bone marrow, the quantity and functional characteristics of hemoglobin, the intraerythrocyte environment [which influences the affinity of hemoglobin for O<sub>2</sub>], the functional capacity of the C.V.S and, pulmonary system and, hormonal regulator of erythropoietin [Erythropoietin EPO] [Miller,1989]. That is to maintain a circulating red cell volume of 30 ml/kg by the production approximately  $3 \times 10^6$  erythrocyte /second. This is maintained by erythroid marrow through its continued replenishment by the entry of committed red progenitors from the self renewing stem cell pool [Sawada et al., 1988].

### **Factors maintaining erythropoiesis:-**

- 1- Adequate stem cells establishment in favorable environment and sensitive to various influences ordering their maturation until they eventually emerge into the circulation as erythrocyte.
- 2- Adequate supply of erythropoietin, erythroid burst promoting activity necessary for the normal growth and development of these erythroid cells at all stages as well as the existence of normal regulatory mechanisms .
- 3- The circulating erythrocytes must not suffer such a degree of premature destruction that the blood forming tissues are unable to maintain normal numbers of cells in the circulation [Wintrobe,1981].

### **Control of erythropoiesis :-**

The main regulator of R.B.Cs production is the tissue O<sub>2</sub> tension. A fall in tissue O<sub>2</sub> resulting from anemia or pulmonary insufficiency is followed by increase in R.B.Cs production . These is done through the action of EPO produced by the kidney [*Bondurant and Kaury, 1986*] .

The rate of erythropoiesis is controlled by intracellular aerobic metabolism and delivery of O<sub>2</sub> to the tissue.

Excessive O<sub>2</sub> suppress erythropoiesis while O<sub>2</sub> starvation stimulate it. The fetal response to hypoxia or anemia is poor compared with that of the mature infant or adult . This decrease sensitivity to the stimulus of EPO probably prevents accelerated erythropoiesis and hyperviscosity of the blood in healthy fetus [*Dallman, 1986*].

Although term infants tend to respond appropriately to fall in hemoglobin concentration after birth by increase EPO production, the preterm infants retain its in-utero hyporesponsivness to hypoxic stimuli with an inappropriately impaired erythropoietic response [*Stockman, 1988*] .

Other factors, such as insulin like growth factor appear to have direct effects on both fetal and early postnatal erythropoiesis [*Kurtz et al., 1988*] moreover, EPO production may be dependent on the rate of body growth [*Widness et al., 1990*].

## **Production and regulation of EPO:-**

EPO is a glycoprotein produced mainly by the kidney in the adult [Schuster *et al.*, 1987]. During fetal, and neonatal periods, the liver is the primary EPO producing organ [Zanjani *et al.*, 1977]. The switch from liver to kidney production of EPO occurs soon after birth [Zanjani *et al.*, 1981]. EPO is controlled by a single gene which is present on the long arm of chromosome (q11-q22) [Law *et al.*, 1986].

In situ hybridization studies indicate variously that peritubular [Kaury *et al.*, 1988] or renal tubular cells [Maxwell *et al.*, 1990] form EPO. It has been suggested that macrophages are a cell type which express EPO and that some formation may occur in bone marrow [Vogt *et al.*, 1980].

In the fetus, EPO can be detected as early as at 19 weeks of gestation [Thomas *et al.*, 1983].

During the last trimester of pregnancy; EPO levels increase markedly along with hemoglobin concentration, and at term, cord blood EPO levels are two folds to fourfolds higher than in normal adult [Eckardt K. U. *et al.*, 1990]. Serum level normally 10-20mu/ml is sufficient to maintain a stable R.B.C mass [Egrie *et al.*, 1987].

Materno-fetal transfer of EPO does not seem to occur although this is a controversial area [Sawyer *et al.*, 1987]. The regulation of EPO level in serum is self regulating system acting through feedback via the oxygen sensor system, where increased oxygenation lead to decrease in EPO formation [Cotes *et al.*, 1989].