CALCIUM AND PHOSPHORUS HOMEOSTASIS IN NEWBORNS AND THEIR MOTHERS

THESIS

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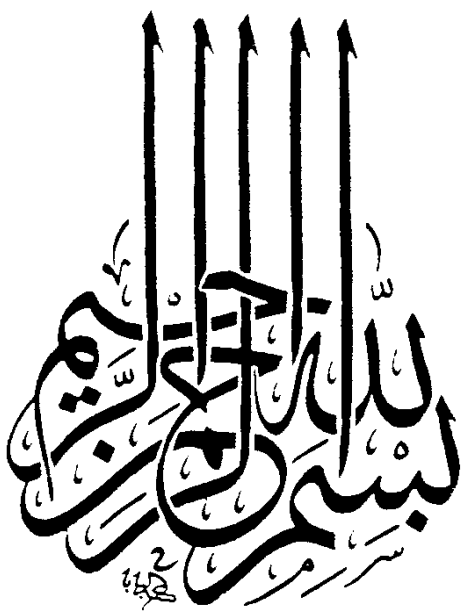

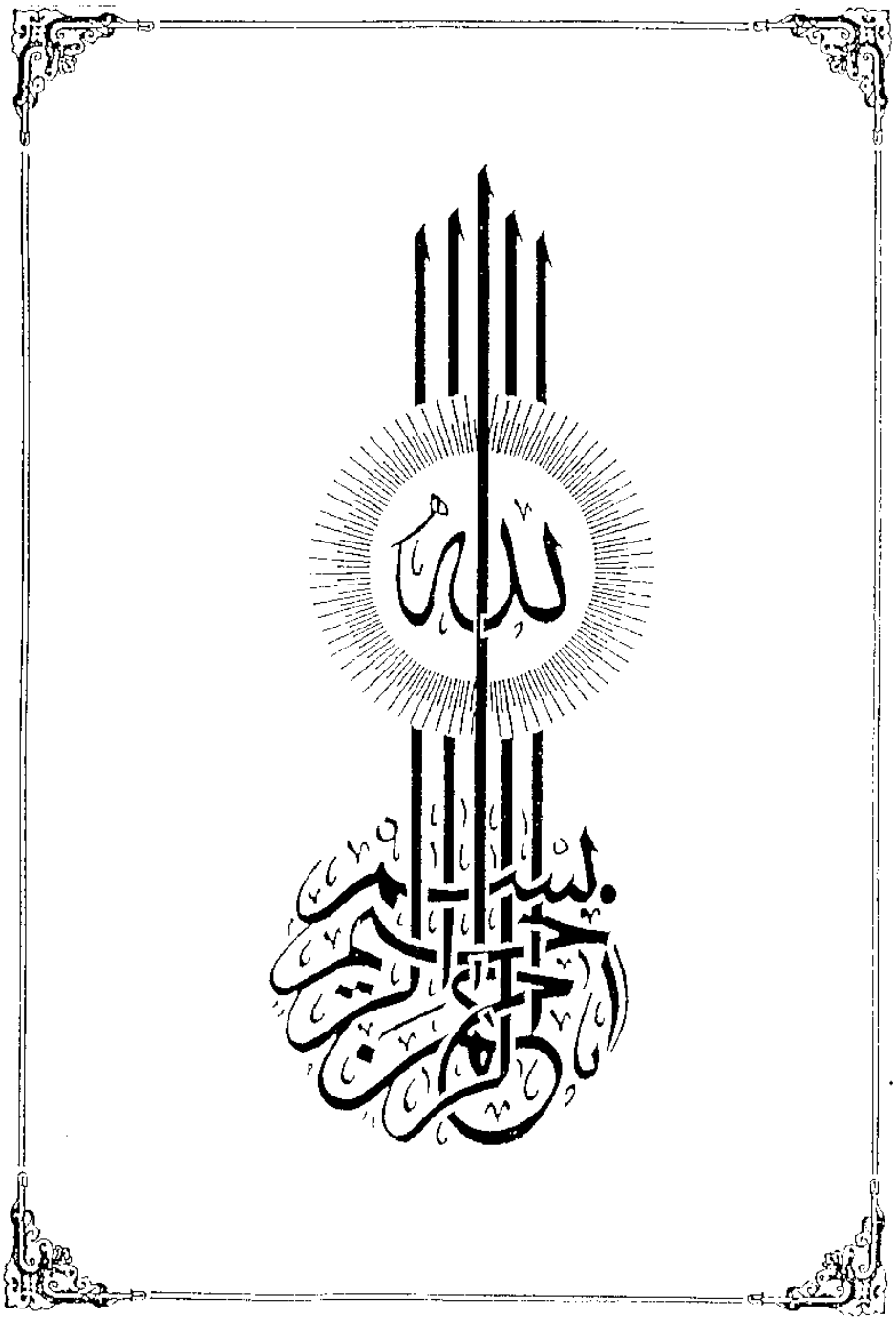

TO MY
PARENTS

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INTRODUCTION AND AIM OF WORK

Introduction

Perinatal mineral homeostasis is regulated by a complex system of endocrine and non endocrine control mechanisms. Non endocrine factors include the ratio between dietary calcium and phosphorus intake, the amount of fecal calcium loss secondary to malabsorption or increased intestinal calcium secretion, and factors related to acid base control and kidney maturation. Hormones involved with the regulation of mineral homeostasis include steroid hormones, growth hormone, thyroid hormones, prolactin and probably prostaglandins.

However, the predominant endocrine control of calcium metabolism is mediated by parathyroid hormone (PTH), calcitonin (CT) and active vitamin D metabolites (Schedewie and Fisher, 1980).

Early postnatal mineral homeostasis is influenced significantly by metabolic conditions prevailing in the fetal-maternal unit prior to delivery. Under normal circumstances, fetal metabolism during the last trimester of pregnancy is geared to mineral deposition into the rapidly growing skeleton. Bone mineral content in the fetus increases exponentially throughout gestation and in linear correlation to body weight. About two thirds of the total amount of fetal bone mineral are deposited during the final

trimester. Calcium, phosphorus and magnesium required for the formation of hydroxyapatite are actively transferred across the placenta by way of the so-called "placental calcium pump" (Scothorne, 1964).

While the nature of this unique transport system remains to be established, it does not appear to involve direct hormone action. The placental pump maintains a positive concentration gradient of the above minerals toward the fetus. Thus, fetal calcium concentrations at term average 1 to 2 mg/dl. above corresponding maternal levels (Pitkin et al., 1980).

Fetal phosphorus concentrations are relatively greater, since the hyperphosphatemic effect of active transplacental transport is enhanced by fetal hypoparathyroidism and pseudohypoparathyroidism. Maintenance of serum calcium, magnesium and phosphorus concentrations in utero does not, under physiologic circumstances, require the activation of fetal endocrine systems. However, supraphysiologic calcium and magnesium concentrations provide a chronic stimulus to the fetus C cell apparatus. The resulting hypercalcitonemia helps to enhance bone mineralization. Neither parathyroid hormone nor calcitonin has been demonstrated to cross the placenta in either direction (Northrop et al., 1977 and Erenberg et al., 1978).

Profound adjustments of hormonal control mechanisms are necessary to ensure the uncomplicated transition of the fetus from intrauterine to extrauterine life. The need for bone mineral deposition continues after birth. However, maintenance of adequate calcium concentrations in the blood becomes the immediate and overriding task for the neonate who, with the clamping of the umbilical cord, is disconnected abruptly from previously abundant mineral supplies (Schedewie and Fisher, 1980.

Aim of the Work:

To find out the effect of:

a. Gestational age.

b. Birth weight.

c. Maternal levels of Calcium, Phosphorus, Parathyroid hormone and Calcitonin on their levels in plasma cord blood.