Introduction & Aim of Work

Pregnancy places additional requirements for iron on the mother.

Anaemia is present in up to 5% of all pregnancies and it involves risks of still birth and hypoxia at birth (Mary et al., 1978), (MacGrogor 1962), has shown that prematurity is 3 times as frequent in iron deficiency females. (Browne, 1978) found that patients with severe anaemia (under 60% Haemoglobin) had a perinatal mortality double that of patient with a haemoglobin 70% or more. (Bulter, 1975) has claimed that incidence of respiratory chest infections and hyaline membrane in infants of severely anaemic mothers is increased.

The rapidly growing human fetus requires a large supply of iron. The fetus is entirely dependent on transfer of iron across the placenta to satisfy prenatal needs.

The fetus effectively parasitize the iron stores of the mothers despite the status of her iron hemostasis (Roger and Stevenoson, 1973).

The placenta plays a key role in regulating the supply of iron to fetus. Placental mechanisms trap maternal transferrin remove iron and actively transport it across the fetus against a high concentration gradient (Okuyama et al., 1985).

In both anaemic and non-anaemic pregnancies, serum ferritin levels decreased and total iron binding capacity increased as gastation progressed. (Okuyama et al., 1985).

Some investigators have found a difference in cord serum ferritin levels in infants born of iron deficient as versus iron supplemented females (Van Mik et al., 1978).

Others however have found signifincatly lowered cord ferritin concentrations among infants born of females whose levels were below normal when compared with infants born of females with normal levels (Kelly et al., 1978).

The aim of this work is to through light on to what extent the faetal iron haemostasis is affected by the severity of the maternal anaemia. For this purpose iron, ferritin and treansferrin is determind in both maternal and cord blood of both normal and anaemic pregnant women.