INTRODUCTION
Infection is the significant cause of mortality and long term morbidity in fetus and newborn. Intrauterine growth retardation is one of the common manifestations of intrauterine infection. Due to constraints in conducting specific tests for the diagnosis of perinatal infection, one could explore the possibility of finding out the value of cord serum Ig M levels as a screening procedure for intrauterine infections in full term intrauterine growth retarded (IUGR) babies. The human fetus is thought to develop immune competence to some antigens by 20th week of gestation or earlier. Gittin and Biasucci in 1968 detected immunoglobulin M (Ig M) synthesis in in-vitro cultures of human embryonic spleens at 10.5 weeks of gestation. Most embryonic and fetal sera didn't contain detectable amounts of Ig M until 35 weeks of gestation because Ig M didn't cross the placenta in substantial amounts.

The immune response in fetus results in antibody production which is quantitatively and qualitatively different from that of adult. Infection of the human fetus in utero elicits an immunologic response in which specific antibodies, Ig M in character, are elaborated. Depending
upon the duration of the process in utero and magnitude of antigenic stimulus, congenital infection may result in elevation of cord Ig M beyond the expected value. Both the symptomatic and sub-clinical varieties of intrauterine infection provoke an immune response in the fetus in the form of increased Ig M synthesis.

Since maternal Ig M is excluded from the fetal compartment, an elevated Ig M is indicative of immunologic stimulation of the fetus in utero. For this purpose, a quantitative gel diffusion technique with antibody incorporated into the agar was referred by Feinberg in 1957. This approach was developed by Mancini et al (1965) and has been utilized in commercially supplied immunochemical equipment. An agar plate is prepared incorporating antibody throughout the agar. The test sample is put into a small antigen well and, on diffusion into the agar, forms a ring of antigen-antibody precipitate around the well. The diameter of the precipitate ring reflects the concentration of antigen. This in turn will indicate the level of Ig M.

There has been an increasing concern over the discrepancy between gestational age and expected weight of fetus at birth. IUGR is conventionally and statistically defined as infant falling below 2 standard deviation of the mean or between the third percentile in weight for gestation.
Babies of IUGR are not a homogenous population and atleast 2 morphological groups are distinguished. The severity of retarded foetal growth depends on the etiology, duration and degree of intrauterine insult to the foetus. If severe insult begins in the first trimester of pregnancy, the foetus is proportionately small in all parameters and the case is therefore, referred to as symmetric IUGR. The typical causes of this form of IUGR include -

(a) intrauterine infection
(b) severe maternal malnutrition
(c) chromosomal abnormalities
(d) severe congenital anaemia

A low genetic growth potential also gives rise to symmetric form of IUGR. If IUGR process begins in the late 2nd or early 3rd trimester, there is a relative sparing of the foetal head and body length in comparison to foetal soft tissue mass and weight. This form of IUGR is referred to as asymmetric IUGR and is usually the result of placental insufficiency.

Several large studies have established that IUGR babies can have a 3-10 fold increase in perinatal mortality when compared to infants whose birth weight is appropriate for gestational age (Ghosh, 1970). In India, incidence of birth weight less than 2500 g is as high as 30%.
IUGR babies are subject to numerous problems during the immediate postpartum period such as intrapartum asphyxia, neonatal hypoglycemia, acidosis, aspiration in utero, congenital malformation and pulmonary haemorrhage (Singh, 1985).

Organisms known to cause intrauterine infections are to toxoplasma and rubella group, Treponema pallidum, Mycobacterium tuberculosis, plasmodia etc (Seth et al, 1985).

The diagnosis of perinatal infection is difficult since a number of babies are asymptomatic at birth and very specific tests are required for the diagnosis. The effects of intrauterine infection on the foetus are manifold but IUGR is one of the silent manifestations. Both symptomatic and subclinical varieties of intrauterine infection can provoke an immune response in the foetus in the form of increased Ig M synthesis. This augmented Ig M synthesis probably does not help to limit established infection but it can be used as a screening investigation for perinatal infection in IUGR babies who had no obvious clinical cause for growth retardation.