f Introduction and aim of the work ♣

Diabetes mellitus is the most common endocrine disorder in the world .Women with diabetes has an increased risk of adverse pregnancy outcome, such as preeclampsia, macrosomia, and stillbirth, contributing to both maternal and fetal morbidity and mortality ***(Garner et al., 1990).***

Because newborn of pregnancies in which just the father diabetes appear to develop normally, it is believed that morbidities in the offspring are due to abnormalities in the maternal metabolic environment, rather than genetic influence ***(Chung & Myrianthopoulos., 1975)***.

Pederson «***1954***», was the first to propose a mechanism whereby maternal fuels may exert a direct effect on the fetus .In attempting to explain the (large babies) that may be seen in pregnancies complicated by diabetes, he advanced the «hyperglycemia-hyperinsulinism» hypothesis. He postulated that more maternal glucose gains access to the fetus whenever maternal insulin is inadequate and this «extra » glucose stimulates insulin release in the fetus and thereby produces an increase of fetal mass.

During the first 24weeks of gestation, the fetus receives its required nutrients from the mother, the transport of which can lead to maternal hyperglycemia. The mother generally requires less insulin during this part of pregnancy .However, in the late second and third trimesters, the demand for insulin increase .This is due, in part, to an increase in placental hormones that are diabetogenic ***(Hare &White., 1980).***

There is increasing evidence that these malformations, which today account for 30 to 50% of all deaths in infants of diabetic mothers, may be attributed to hyperglycemia during early weeks of pregnancy. Hemoglobin A_{1c} levels reflect glycemic control in previous weeks and months .The incidence of major anomalies is significantly higher in pregnancies of diabetic mothers who have elevated hemoglobin A_{1c} concentrations during the 1^{st} trimester ***(Miller et al., 1981).***

Baker et al., (1981), have recently demonstrated that the increased incidence of lumbosacral skeletal defects observed in fetuses of diabetic rat mothers can be reduced to control levels by aggressive insulin treatment.

Experimental, clinical, and epidemiological studies indicate that the increased risks of congenital malformations and spontaneous abortions in pregnancies complicated by diabetes are linked to disturbances in maternal metabolism around the time of conception ***(Metzger et al., 1997)***.

Several reports suggest that the frequency of spontaneous abortion increases in direct proportion to glycohemoglobin concentrations measured around the time of conception ***(Green et al., 1989)***.

A number of other factors that might mediate metabolic teratogenesis have been described[myoinositol depletion ***(Strieleman et al., 1992)*** ,polyol accumulation ***(Hashimoto et al., 1990)*** , aracadonic acid deficiency ***(Goldman et al., 1985)*** ,free oxygen radical generation ***(Eriksson & Borg ., 1993)***] .

This study was aimed to observe the possible changes in the myometrial spontaneous contractions occurring in diabetes mellitus ,and the responses of uterine smooth muscles to some uterotonic agents such as oxytocin and prostaglandin $F_{2\alpha}$which are stimulants of both frequency and force of uterine contraction .The study was extended to investigate the effect of both oxytocin and prostaglandin $F_{2\alpha}$ (in vitro) on pregnant and non pregnant rat uterine strips.