Background and aims We studied the feto-placental interface, in Pregnancy Induced Hypertension (PIH), to present his specific structural modifications.

Method We have studied the histocinogical modifications of 68 placentas obtained after delivery for two equal groups representing mothers with PIH and normotensive. The samples obtained by sections were specifically prepared for the study using three types of histological stains. We used optical microscopy for observing mainly the lumen of spiral arteriole and changes in its intima and medial tunica.

Results We registered the following specific structural modifications in the pregnancies with PIH: fibrosis in the middle of the villous, fibrinoid necrosis, condensation of stromal connective tissue, syncytiolayer agglutinations of the villos or intervillus spaces (nodes, buds, or bridges), thrombosis and/or infarction of the spiral vessels and villos capillary endothelial atheromatosis. Conclusions Our study was done to find a better understanding of the histo-logical changes of the preelcaptic feto-maternal interface concerning his role in PIH. The morphological modifications of the feto-placental interface in the PIH represent a marker of the fetal and postnatal hypoxia/ischemia with an immediate and late impact upon their cerebral development.

Background and aims We studied the feto-placental interface, in the Gestational diabetes mellitus (GDM), to present his specific structural modifications and his cellular injuries.

Method An optical microscopic analysis was performed on 30 placentas, obtained after delivery for two equal groups representing mothers with GDM and normal pregnancies. The samples obtained by sections were specifically prepared for the study using three types of histological stains. The histological observation centred upon the: trophoblast, villous stroma and fetal capillary. The statistical study of the data was performed using SPSS 17.0.

Results Through optical microscopy were identified varying degrees of lesions consisting of: villous oedema, proliferation and villous fibrosis of the capillaries, large number of syncytial knots, important fibrinoid necrosis, moderate fibrin thrombi, hyperplasia of the syncytiotrophoblast, chorangiosis, slightly thickened of the basement membrane of the feto-maternal interface. Conclusion Histological changes in the placentas of women with GDM are significant factors contributing to fetal anoxia with impact on placental vascular permeability. A diabetic milieu causes vascular dysfunction, increasing angiogenesis in GDM is considered to be the cause of the placental abnormalities and complications (miscarriage, stillbirth, macrosomia, and congenital anomalies).