THE PLACENTA IN MATERNAL DIABETES MELLITUS

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The findings in the placentae of the gestational diabetic were identical to those seen in the placenta of the patient with long standing diabetes mellitus. It is true that in general terms the changes seen in the placentae of the gestational diabetic were less extensive and less severe than were those in the placenta of the well established diabetic, but nevertheless there was some degree of overlap and it is not possible by looking at a placenta, either by the light microscope or electron microscope, to tell whether it comes from a patient with mild, transitory, easily controlled, gestational diabetes mellitus or from a woman with long standing, moderately controlled, insulin dependent diabetes mellitus.

These findings indicate that the degree of control of the diabetic state achieved during pregnancy, the severity of the diabetic condition and the duration of the diabetes mellitus are largely unrelated to the presence and severity of pathological lessions within the placenta of the diabetic woman. In a recent morphometric study, Teasdale (1981) came to a similar conclusion. It is of interest to note also that Bjork and Person (1982) detected pathological findings in the placentae of insulin dependent diabetics, and could demonstrate no relation between the extent of the placental abnormalities and the degree of control achieved in the diabetic condition in late pregnancy; these workers did, however, show a significant correlation with the blood glucose values obtained in the earlier stages of pregnancy and their study did tend to indicate that the degree of metabolic control obtained in early gestation is reflected in the placenta during late pregnancy. It is difficult to reconcile this finding, however, with the fact that abnormalities of significant degree are also observed in the placenta of gestational diabetic women despite the fact that in our series these women did not have glycosuria during the early stages of gestation.

What then is the cause of the placental damage in maternal diabetes mellitus? It must be concluded that the damage is due to some component of the diabetic state and to some factor in the altered and abnormal environment. What this factor is must, however, remain a subject for speculation in our present state of knowledge though it does appear highly probable that it is a factor which is not adequately controlled either by diet or by insulin.

The significance of placental damage in maternal diabetes mellitus

It is clear that the placenta is damaged in maternal diabetes but it does not appear that placental function is adversely affected by such damage. The extent of residual syncytial damage is extremely small and it is clear that any damage to the syncytiotrophoblast is readily and rapidly repaired by an uninhibited cytotrophoblastic hyperplasia/repair system. Furthermore, the morphological parameters of syncytiotrophoblast function suggest that this tissue is, if anything functioning at optimal capacity; thus the excessive massive number of secretory droplets indicates increased secretory activity and this would be in

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accord with the elevated level of hPL which has been noted in maternal diabetes. Furthermore, the transfer activity of the syncytiotrophoblast, as evidenced by the presence of pinocytotic vesicles, appears to be increased as does evidence of protein synthesis by the endoplasmic reticulum.

It must be appreciated that the human placenta is a vigorous, versatile organ with a very considerable functional reserve capacity and remarkable ability to repair any damage upon it. Any consideration of the placenta in maternal diabetes suggests that although the placenta is damaged to a minimal extent, it is nevertheless readily repaired and is generally functioning at above optimal level, presumably to compensate for the unfavourable maternal environment. It is extremely unlikely therefore that any of the ills that may beset pregnancy in the diabetic woman or any of the problems that may occur in the fetus of the diabetic woman are in any way attributable to placental malfunction.