STRUCTURAL BASIS OF COMPENSATORY AND ADAPTIVE REACTIONS OF HUMAN PLACENTA

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The maintenance of homeostasis is associated with compensatory and adaptive reactions [10]. However, quite a number of unsolved problems remain in relation to certain organs and, in particular, the placenta. The placenta performs many different functions, of which the most important are transport, barrier, and hormonal. Analysis of the published research [1, 4, 5, 7, 11, 12, 14] leads to the conclusion that the morphological equivalent of the functions of the placenta is formed by the same structures of the villi: the chorionic epithelium (ChE), its basement membrane, the interstitial tissue, the basement membrane of the vessels, and the endothelium. It can be asserted on a priori grounds that this universal character of the structural organization of the placenta, if modified in order to enhance the effect of one function, must inevitably adversely affect another function. The aim of this investigation was to identify the morphological substrate of compensatory and adaptive processes in the human placenta.

EXPERIMENTAL METHOD

A morphological study was undertaken of 25 placentas from women with nephropathy of pregnancy, who had given birth to living infants at term without any signs of intrauterine malnutrition (experiment). The comparison group (control) consisted of 18 placentas from women with uncomplicated pregnancy, with delivery at term. All 43 infants were born in a head presentation and scored 8-10 points on the Apgar scale. The placentas were studied by standard methods of organometry and morphometry [8]. Paraffin sections were stained with hematoxylin and eosin and by Van Gieson's, Mallory's, Shueninov's, and Einarson's methods.

EXPERIMENTAL RESULTS

The microscopic study of the placentas revealed various morphological changes. For instance, besides villi with the normal structure (which were much smaller in nephropathy than in the control) there were also congested, edematous, sclerotic villi, with fibrinoid changes. Disturbances were observed in ChE. In some villi it covered the whole surface, but in others, either focal desquamation was present or, on the other hand, it proliferated.

Narrowing of the intervillous space (IVS) was observed in some areas and widening in others; layers of desquamated syncytium and fibrin clots accumulated in it, and led to "adhesion" of the villi together. In the basal lamina extensive areas of fibrinoid could be seen, around which lymphocytes collected. However, it is difficult to obtain a reliable estimate of the intensity of compensatory and adaptive processes in the placenta purely on the basis of its morphological study, and for that reason a stereohistometric investigation also was undertaken (Table 1).

Analysis of the results of the morphological and morphometric investigations revealed that structural changes develop in the placenta in patients with nephropathy of pregnancy, some of which inactivate certain areas from the functional point of view, whereas others, on the other hand, enhance placental activity. The necessity for this enhancement of placental function is due not only (possibly not so much) to morphological changes in the placenta itself, but also to the development of anoxemia and hypertension in the mother with nephropathy, accompanied by a sharp decrease in the volume of blood entering the placenta. In nephropathy of pregnancy metabolic processes also are disturbed with the accumulation of toxic products and autoantigens in the maternal blood [3, 6, 9, 13].

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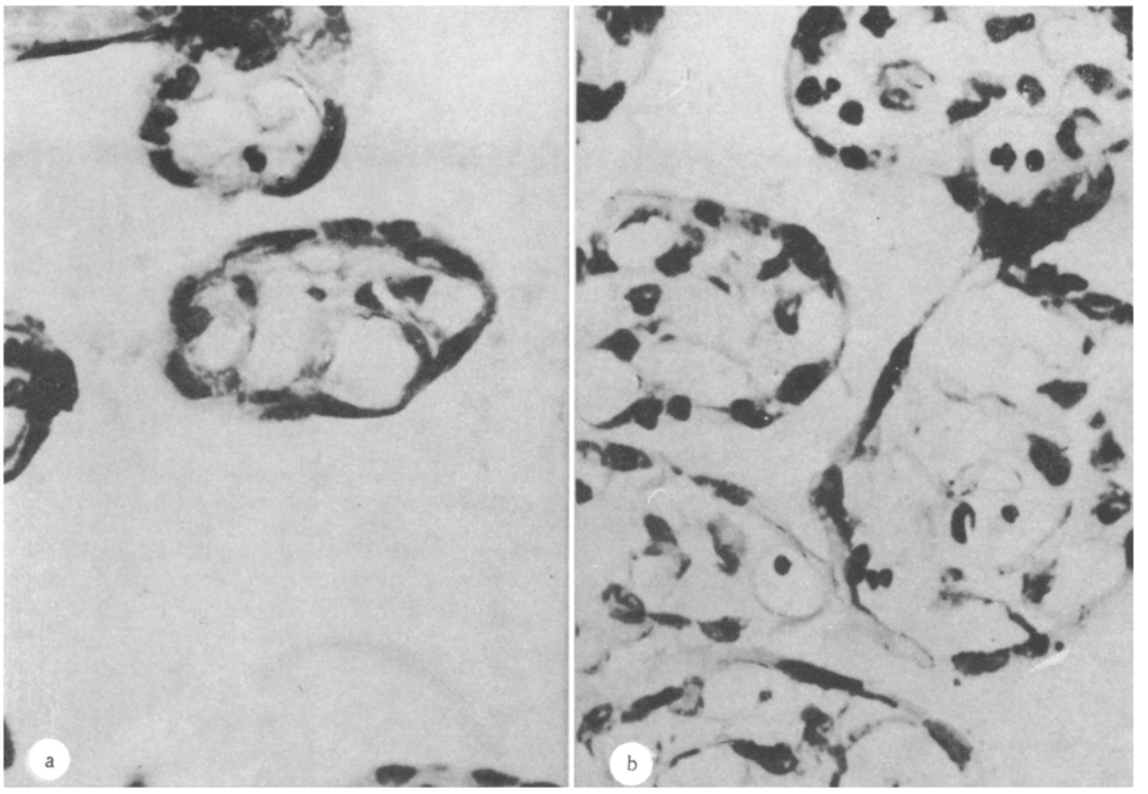

Fig 1. Structure of terminal villi of placenta. a) Normal pregnancy: blood vessels displaced toward basement membrane of ChE with the formation of two or three syncytiovascular membranes. Hematoxylin and eosin: b) nephropathy of pregnancy of the III degree: displacement of blood vessels toward basement membrane of ChE with the formation of four or five syncytiovascular membranes and thinning of ChE. Einarson's stain. 650 ×.

TABLE 1. Stereohistometric Parameters (in %) of Placenta of Infants Born to Women with Nephropathy of Pregnancy

Parameter	Control	Experiment
XP	9,23±0,86	7,51±0,96
VS	5,45±0,82	7,12±0,90
IVS	39,87±2,11	37,61±3,49
MF	1,02±0,15	1,20±0,11*
Interstitial tissues	21,62±1,06	16,86±0,73*
FF	0,45±0,10	1,99±0,30*
Vascular bed	8,95±0,58	14,17±1,90
ChE	6,89±0,66	4,10±0,88*
SB	1,78±0,17	1,42±0,60
PT	2,79±0,12	3,33±0,57*
"Adherent" villi	1,85±0,23	4,60±1,79*
Infarcts	—	—
Foci of calcification	0,10±0,05	—
Foci of inflammatory infiltration	—	—

Legend. Asterisk indicates $p < 0.05$-0.01; in all other cases $p > 0.05$.

There is no doubt that the changes developing in the placenta and in the mother affected by nephropathy of pregnancy ought to be compensated by enhanced activity of the entire mother–placenta–fetus biological system. Nevertheless, it can legitimately be asked what function or functions of the placenta require enhancement. Analysis of the results of this investigation indicates that structural changes take place during nephropathy, reflecting the increased transport function. Under these circumstances the number of congested villi is increased and the blood vessels are displaced toward the basement membrane of ChE. Syncytio-capillary membranes are frequently formed, ChE is reduced in thickness, and the rela-

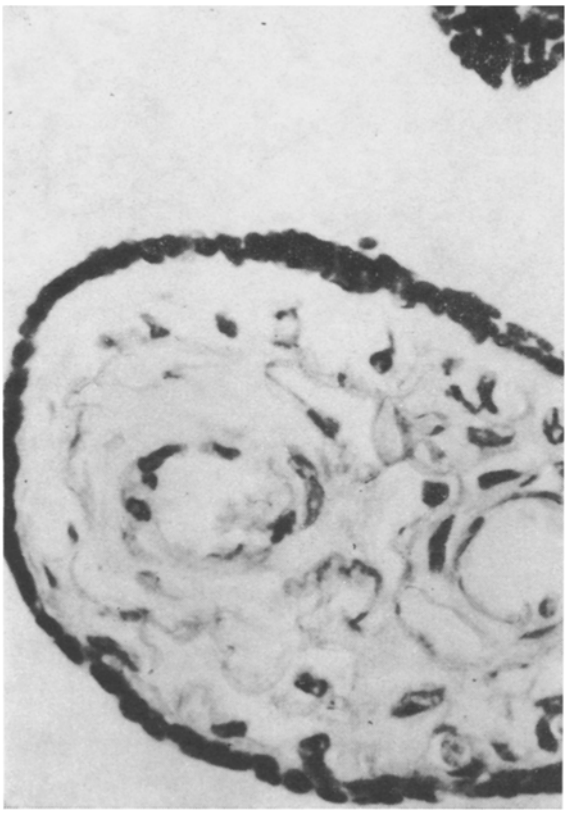

Fig. 2. Placenta (nephropathy of pregnancy of the II degree). Wide layer of ChE. Blood vessels located in center of villus. Hematoxylin and eosin. 650 ×.

tive volume of the vascular bed is increased by 58.5% compared with the control ($p < 0.05$), reflecting a relative change in the value of the parameter (in %) compared with the control, taken as 100%. The relative volume of the interstitial tissues (by 22%, $p < 0.01$) and of ChE (by 40.5%, $p < 0.05$) is reduced, i.e., conditions leading to the closest possible approximation of the maternal and fetal blood are created (Fig. 1).

The barrier function in general and the function of the immunologic barrier in particular rest on the same material basis as the transport function, but with the opposite sign. Under these circumstances maternal and fetal blood are separated as far as possible. The structural components which carry out the barrier function most effectively are the wide layer of ChE, the central position of the blood vessels in the villi, and the interstitial tissues, especially when affected by fibrinoid changes and sclerosis (Fig. 2).

The view was held initially that in nephropathy of pregnancy structural changes take place in the chorion frondosum, capable of enhancing the barrier function. However, it can be concluded from the results of the stereohistometric investigation that only the relative volume of fetal fibrinoid (fibrinoid in the interstitial tissues of the villi) is significantly higher than in the control. The other parameters of ChE, the interstitial tissues, and the vascular bed are evidence not of improvement, but of worsening of the state of the barrier function of the placenta.

The results of the stereohistometric investigation may lead to a logically incorrect conclusion: in nephropathy the mother–placenta–fetus biological system does not need enhancement of the barrier function. The characteristics of the course of nephropathy indicated above, however, do not allow this unambiguous conclusion to be drawn. Besides the placenta, both the mother and the fetus itself will also be involved in neutralization and isolation of the "noxious principles" of late toxemia of pregnancy. In this connection it can be postulated that enhancement of the barrier function of the placenta, including the function of the immunologic barrier, takes place not only on account of the structures of the villi (for this leads to worsening of the transport function), or indeed, not so much due to those structures as an account of other components of the placenta: maternal fibrinoid (MF) and the peripheral trophoblast (PT) (incidentally, the relative volume of MF and PT is greater in nephropathy that in the normal control). This hypothesis is not without foundation. Some workers [2, 15] consider that the fibrinoid and trophoblast are responsible for eliminating and binding antigens in the maternal blood in IVS.

The trophoblast, both that part which covers the villi and the peripheral part, located in isolated areas of the septae and in the form of compact islets among the villi immured in fibrin, serves as the material substrate for the hormonal function of the placenta.

We considered that in nephropathy of pregnancy all three parameters — ChE, syncytial buds (SB), and PT, would be higher than in the control. However, as Table 1 shows, only the relative volumes of PT was higher than that in the control, and the relative volume of ChE and SB were lower.

We attribute this phenomenon to be particular features of the placenta, in which many functions are performed mainly on the same material basis. Marked proliferation of the chorionic epithelium (parameters ChE and SB) must therefore unavoidably impair performance of the transport function. It can be tentatively suggested that in the interests of the latter, structural changes in the chorion frondosum will take place very economically, and if possible on account of other components of the placenta without impairing performance of the basic functions.

The placenta is thus a polyfunctional organ whose morphological organization is designed to perform different functions (transport, barrier, hormonal) by the same structures. During the development of compensatory and adaptive reactions these morphological features are such that, while they enhance the activity of one function, they reduce the efficiency of another. When morphological changes in the placenta are analyzed, therefore, compensatory and adaptive changes in it must not be evaluated always on the same basis, without allowing for its concrete functions.

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