# GENERAL PATHOLOGY AND PATHOLOGICAL PHYSIOLOGY

# Structure of Vascular Plexuses of Brain Ventricles in Ontogeny under Normal Conditions and in Hypoxia

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The structure of vascular plexuses of brain ventricles in newborns developed under hypoxic conditions does not correspond to gestational age. Chronic hypoxia decreases activity of succinate dehydrogenase and iron content in vascular plexuses of brain ventricles.

**Key Words:** *choroid plexus; hypoxia; newborn*

Hypoxia, a universal damaging factor, occupies a special place among factors affecting newborn brain [2]. In many cases, asphyxia in newborns is a consequence of fetal hypoxia. Hypoxia and asphyxia are responsible for 72.4% fetal deaths at delivery and in the early neonatal period [4]. Asphyxia at delivery or in the neonatal period is detrimental for developing brain [1,5]. Normal function of the brain depends on the blood—brain barrier [3]. Vascular plexuses of brain ventricles (VPBV) are the basic structures of the blood-brain barrier separating blood and liquor. VPBV participate in optimization of compensatory, repair, and adaptive processes, maintenance of optimal metabolism in damaged brain [6].

Our aim was to study VPBV structure in newborn children experienced hypoxia (asphyxia).

## MATERIALS AND METHODS

Postmortem specimens of 30 newborns obtained in Central Astrakhan Pathologoanatomic Bureau were examined.

The specimens of VPBV were taken from acinar region of the lateral ventricles. VPBV structure was studied in paraffin and frozen media. Routine histological staining with hematoxylin and eosin was made according to Van Gieson. Glycosaminoglycans, succinate dehydrogenase (SDH), and iron were revealed by the methods of Moury, Nachlas, and Perls, respectively.

## RESULTS

The newborns were delivered at term (40%) or at gestational weeks 36-37 (60%). In most women pregnancy was accompanied by early and late gestoses

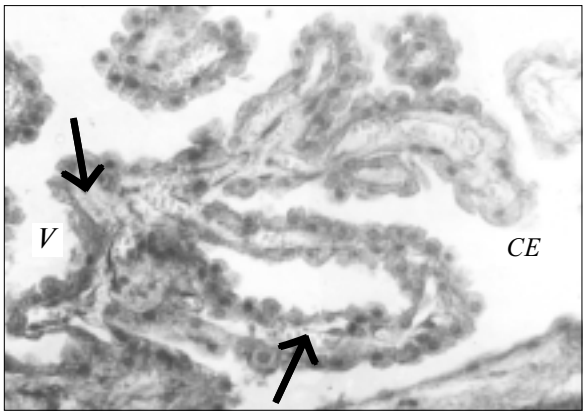


**Fig. 1.** Vascular plexuses of brain ventricles in newborn (gestational age 37 week) developed under hypoxic conditions. Hematoxylin and eosin staining, ×600. CE: choroid epithelium, V: villi. Arrows show blood cells in vessels.

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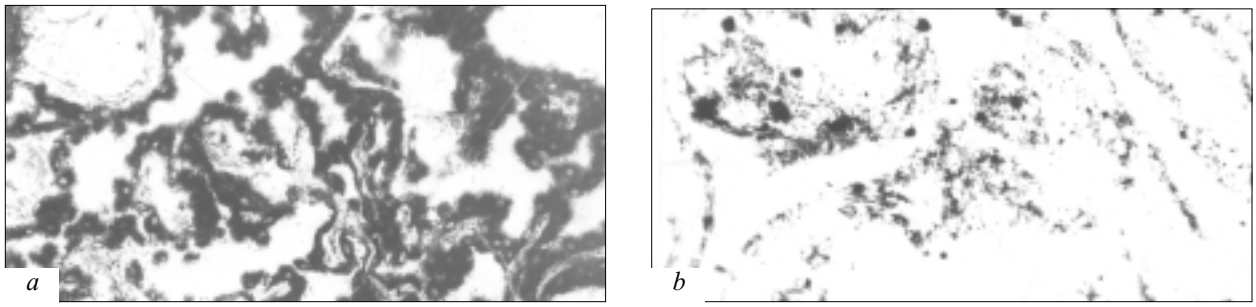


**Fig. 2**. Activity of succinate dehydrogenase in vascular plexuses of brain ventricles in newborns (gestational age 36-37 and 37 weeks) developed under normal (*a*) and hypoxic (*b*) conditions. Nachlas method, ×300.

(33.3%), threat of spontaneous abortion (16.6%), anemia (20%), and acute respiratory viral infection (16.6%); 13.5% women were not examined.

In 16.6% newborns VPBV had no villi, in 43.3% newborns columnal arborization of villi was disturbed, and in 40% newborns the structure of VPBV villi corresponded to gestational age. In 66.6% newborns the microvascular bed was poorly developed and occupied small areas. The vessels were plethoric (Fig. 1).

Although in 16.6% newborns the death was caused by infectious disease (viral and bacterial infection), none specimens had sings of perivascular infiltration.

In 13.3% newborns VPBV contained elongated giant cells. In 3.3% preparations vessel walls in VPBV were thickened, psammoma bodies were sometimes seen. The connective tissue was poorly developed, epithelial cells demonstrated signs of apocrine secretion. In 46.6% newborns fragments of stratified epithelium were found in VPBV. In $^1/_3$ cases VPBV contained multinucleated cells with 2-6 nuclei. The sites with damaged epithelium were also observed, but in most cases the vascular walls consisted of cubic and cylindrical epithelium characteristic of normal VPBV. The highest SDH activity was observed in epithelial cells, and it dropped during chronic hypoxia (Fig. 2, *a*, *b*). Iron content in cells of the choroid epithelium followed the same regularity as SDH activity.

Thus we revealed structural immaturity of VPBV and a decreased level of oxidation-reduction processes in newborns with cerebral hypoxia.

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