

MORPHOLOGY AND PATHOMORPHOLOGY

Persistence of Physiological Hydrocephalus in the Newborn

S. A. Kakabadze and V. P. Tumanov

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The *in utero* development of the brain is characterized by a significant expansion of the ventricular system during the first four months, which has been termed physiological hydrocephalus [1]. Later on, the ventricular system shrinks, providing the necessary conditions for the normal development of the brain. This process can be disturbed by diverse factors promoting the development of the known forms of congenital hydrocephalus [1-4], or a delay of the normal outcome of physiological hydrocephalus. The latter state, which we call persistence of physiological hydrocephalus (PPH), has been reliably detected in the absence of morphological signs of hydrocephalus development, which are sometimes insignificant and clinically negative at the moment of birth [4]. In this study we aimed to determine the frequency and reliable criteria of PPH in the newborn (NB) during the early neonatal period.

MATERIALS AND METHODS

The NB contingent consisted of the following groups: I) NB of 28-32 weeks gestation, II) NB of 33-37 weeks gestation, III) NB delivered at term. The presence or absence of interthalamic commissure (ITC) was determined (Table 1, Figs. 1, 2). The investiga-

tions comprised the standard set of methods, including a detailed study of the state of the ventricular system and of the subarachnoidal space of the cerebrum. The ITC with the adjacent parts of the optic chiasma was cut horizontally and subjected to histological study. The quantitative data were analyzed using Student's *t* test.

RESULTS

None of the groups observed had a history of hereditary disorders and all had a similar range of the mothers' age. In the anamnesis of all groups abortions, miscarriages, stillbirths, and perinatal mortality were registered with approximately equal frequency. A pathological course of pregnancy was less frequently observed in the groups with an absence of ITC (25.0-40.0%). In the same groups the frequency of infectious diseases in the mother was highest, dropping from 50% in group I to 30.7% in group III, while the percentage of caesarean deliveries and the frequency of abnormal positions of the fetus were significantly lower. A certain regularity was found for the frequency of the main groups of diseases with a lethal outcome (Table 2). Among infectious diseases, sepsis was more often encountered in observations in the presence of ITC and was mainly noted in group III. Meningitis was involved in sepsis in approximately half of the observations; ependymitis and encephalitis were observed rarely. The marked preva-

Department of Pathological Anatomy, North Ossetian State Medical Institute, Vladikavkas; Department of Pathological Anatomy, A. V. Vishnevskii Institute of Surgery, Moscow. (Presented by D. S. Sarkisov, Member of the Russian Academy of Medical Sciences)

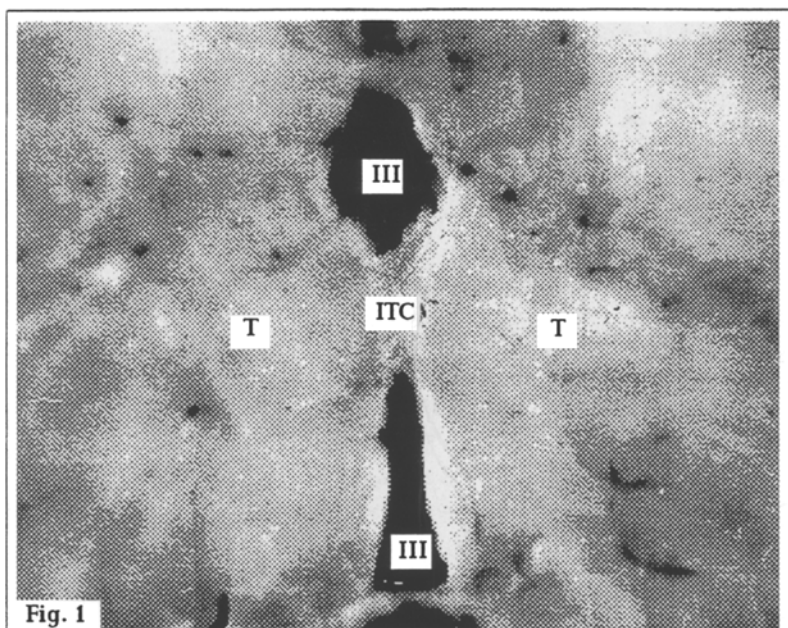


Fig. 1

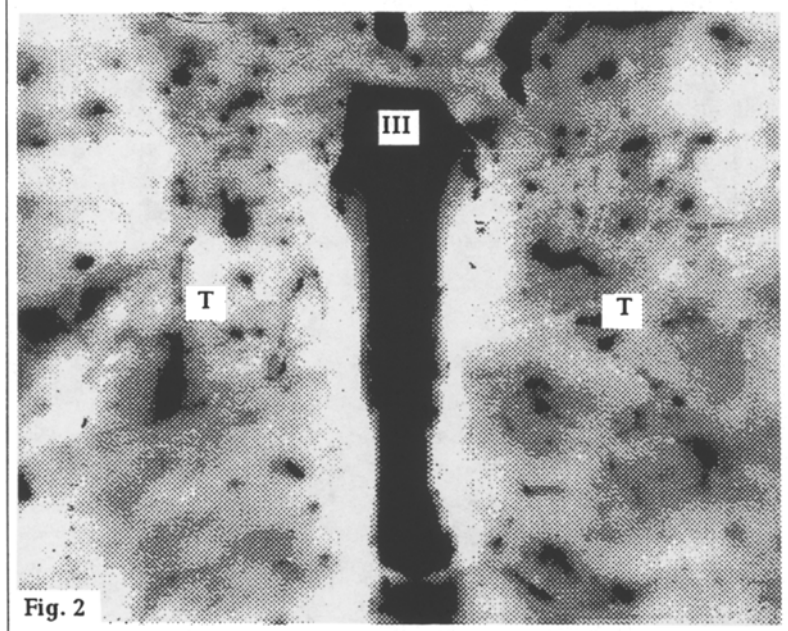


Fig. 2

Fig. 1. Interthalamic commissure well developed in a newborn delivered at term. T: thalamus; ITC: interthalamic commissure, III: the third ventricle. Macropreparation $\times 4.4$.

Fig. 2. Absence of interthalamic commissure in a newborn delivered at term. Designations are the same as in Fig. 1. Macropreparation $\times 5.2$.

lence of developmental defects in groups I and III in the absence of ITC is typical. Identical anthropometric indexes were found among all the groups, the weight and anatomical formation of the brain corresponding to the age standard (Table 1). The permeability of the cerebrospinal fluid communications was not disturbed, and signs of old or fresh pathological processes were absent in their region.

A moderate or significant widening of the posterior horns and, partially, of the inferior horns of the lateral ventricles, this often being accompanied by a slight dilatation of the third ventricle, was observed in 41.6, 42.1, and 7.7% of cases in groups I, II, and III (in the absence of ITC), respectively. Edema of the brain tissue and constriction of the ventricular system were noted in the other observations, where an ITC was absent. The majority of observations (68.4-83.3%) were characterized by a significant subarachnoidal liquorstasis, this occurring independently of the state of the ventricular system. Histological investigations showed the usual spectrum of pathology due to injurious factors of a pathological pregnancy and delivery, corresponding to the main disease. A general cerebral symptomatics was determined clinically in more than half the observations, usually corresponding to the degree of hemostatic plethora, edema, and subarachnoidal liquorstasis in the brain.

The groups of NB were characterized by a similar frequency and degree of cerebral symptomatics in the presence of ITC. In most cases the ITC was well marked, had a length of 3-6 mm, a thickness of 2-3 mm, and an oval profile in the sagittal plane. In a few observations it was weakly expressed (point). In rare cases of a combined widening of the posterior horns of the lateral and third ventricles, the ITC

TABLE 1. Number of Observations, Anthropometric Data, and Weight of Brain in Groups in the Presence or Absence of ITC

Group	Number of observations		Body weight, kg		Body length, cm		Weight of brain, g	
	with ITC	without ITC	with ITC	without ITC	with ITC	without ITC	with ITC	without ITC
I	38	14 (26.9%)	1.72 ± 0.04	1.59 ± 0.05	42.3 ± 0.45	42.3 ± 0.65	231.4 ± 5.8	230.7 ± 9.7
II	46	20 (30.3%)	2.36 ± 0.10	2.30 ± 0.09	46.0 ± 0.43	45.2 ± 0.68	297.8 ± 6.6	296.2 ± 11.2
III	64	18 (21.9%)	3.25 ± 0.07	3.22 ± 0.18	50.9 ± 0.27	51.1 ± 0.46	368.9 ± 5.5	362.5 ± 12.0

TABLE 2. Diseases in Groups with Presence or Absence of ITC (%)

Disease	Groups of newborns					
	I		II		III	
	with ITC	without ITC	with ITC	without ITC	with ITC	without ITC
Pneumopathies	65.8	55.5	38.5	47.8	8.8	8.35
Infectious diseases*	24.3	22.2	36.8	33.3	49.4	45.8
Defects of development**	7.3	11.15	19.2	19.1	26.8	37.5
Other	2.6	11.15	5.5	—	15.0	8.35

Note: *) only observations with an identified bacterial flora; **) observations without defects of CNS development.

was a narrow strip with a length of 3-4 mm. In the case of a well-formed ITC, the zone of juncture was scattered with small neurocytes. No passage of bundles of myelinated nerve fibers through the ITC was found.

Thus, the absence of an ITC can be considered a reliable morphological indicator of PPH independently of the state of the brain tissue or of the degree of dilatation the lateral and third ventricles in NB.

Dyschronia of the formation of the cerebrospinal fluid communications together with the mechanisms of familial transitory hydrocephalus have been shown to be able to induce PPH [4]. The results of our investigation confirmed this. The contingent of NB with PPH included several cases with familial transitory hydrocephalus, encountered more often in boys and remaining clinically undetected in the early postnatal period. A significant predominance of boys (80.7% on average) was observed among the NB in the groups without ITC, while in the groups where ITC was present, the proportion of boys accounted for 54.0% on average. In PPH pathogenesis a complex of factors interfering with the morphogenetic processes in the earlier prenatal stages has the greatest significance, as is confirmed by the higher frequency of developmental defects in this contingent of observations. From this point of view, PPH may be the initial stage of hydrocephalus development, considering that it frequently goes along with dysplasia in other locations [2]. On the other hand, some of the observations of PPH could have demonstrated a spontaneous compensation of congenital hydrocephalus, this being possible for its transmitted forms [4]. After such an outcome, however, signs of incomplete development of the cortex, besides the dilatation of the ventricles and the absence of an ITC, are preserved during the neonatal period, this not being typical for PPH. Unlike colpocephalus, which is a rare defect [6], PPH is a rather common phenomenon and is characterized only by reduced signs of the fetal ventricular system. Differentiating PPH from some autosomal syndromes (Edwards', partial trisomy 10p), characterized, in particular, by a dilatation of

the posterior horns of the lateral ventricles [3], is not complicated.

The results of our investigation and the differential diagnostics show, therefore, that PPH is in most cases an independent process distinctly documented morphologically, having, however, no definite clinical manifestations and hard to diagnose *intra vitam*. Specific etiological factors are absent in PPH, but the nature of its pathogenesis and morphological peculiarities and the fact that it is frequently accompanied by noncerebral dysplasias, justify its being referred to, by analogy with minimal brain dysfunction, as minimal brain dysplasia. In accordance with the contemporary classification of developmental tissue defects [5], PPH can be referred, according to its morphological features and ontogeny, to the group of brain dysplasias characterized by the persistence of embryonic structures having no (or an undetermined) functional significance and often being incidental.

The outcome of PPH can be at least of two kinds. In some cases, PPH can provide a favorable background for the development of a particular form of *in utero* or postnatal hydrocephalus, when the corresponding pathogenic factors are present. In most cases, however, PPH is eliminated by itself during the postnatal period, but at the same time ITC formation may not occur.

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