

CORRESPONDENCE

The Development of Extremely Premature Infants

Results From the Lower Saxony Longitudinal Study of Prematurity (Niedersächsisches Frühgeborenen-Nachuntersuchungsprojekt)

by Dr. med. Wolfgang Voss, Dr. med. Elke Hobbiebrunken, Dr. Uta Ungermann Ph.D., Dr. med. Michael Wagner, and Gabriele Damm in issue 51–52/2016

The Causes and Prevention of Symptoms in Premature Babies

Follow-up examinations of premature infants show the substantial deficits in the subsequent development of these babies. The cause of such developmental disorders is the lack of oxygen in the first phase of life. This is caused by the fact that the oxygen transport system in premature infants differs from that in mature neonates. This is caused by high concentrations of fetal erythrocytes with fetal hemoglobin (HbF) in the first months of life. The oxygen affinity of these fetal erythrocytes is very much greater than that of adult platelets, and the result is chronic tissue hypoxia. This chronic undersupply of oxygen causes retinopathy of prematurity and the neurological deficits that Voss et al. observed in later follow-up investigations (1).

Avoiding this damage to premature infants is possible only by eliminating the cause and by avoiding oxygen deficiency (2). Measuring HbF in this risk group shows high concentrations. Exchanging fetal platelets with adult platelets in high-risk groups can avoid hypoxia in this phase of life. The exchange has to take place rapidly—that is, postpartum—and to a sufficient degree, in order for the prevention to be successful. The effect of this measure should be monitored by measuring HbF concentrations.

Neonatology will have to face this challenge in order to prevent cerebral deficits and retinopathy of prematurity. This necessity stems in particular from the fact that 0.6% of babies are born before the 28th week of gestation. As the authors stated, 25.1% in this at-risk group die. Any option for prevention should therefore be sought out.

DOI: 10.3238/arztebl.2017.0298a

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In Reply:

Hammerstein appropriately mentioned the raised concentration of HbF in the blood of very small premature neonates compared with children born at term, and the different oxygen binding curves. Global tissue hypoxia of prematurity can, however, not be deduced from this. In particular, the problem of retinopathy of prematurity mentioned by Hammerstein, which used to be substantial in the past, has been notably alleviated by improved ventilation regimens after applying exogenous surfactants (1). In our population, only 0.2% of children have been affected by bilateral blindness subsequent to retinopathy (2).

In our article we explained the relevant factors for a poor outcome (prematurity <26th week of gestation, intraventricular cerebral hemorrhage, low maternal educational status). The physiologically raised HbF concentrations of premature babies, however, affect all children, including those with a good outcome. Effectively lowering of HbF concentration would be possible only by exchange transfusion, a maneuver that is associated with substantial risks, especially in this patient population. Relevant studies of such treatment are therefore missing in the literatures.

Further progress in terms of survival and the long-term development of extremely premature neonates can certainly be achieved in the specialisms of perinatology and neonatology. Our group of authors has contributed to analyzing the current healthcare situation. We do not, however, presume to question therapeutic regimens in neonatology.

DOI: 10.3238/arztebl.2017.0298b

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Conflict of interest statement

The authors of both contributions declare that no conflict of interest exists.