Concerning the paper of Blennow et al. Monoamine Neurotransmitters and Metabolites in the Cerebrospinal Fluid following Perinatal Asphyxia

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We have read with interest the article by Blennow et al. [1] on the levels of monoamines and the main cerebral monoamine metabolites in cerebrospinal fluid in full-term newborn infants with and without perinatal asphyxia. They found lower noradrenaline (NA) levels and more rapid NA turnover in asphyxiated infants with moderate or severe hypoxic-ischemic encephalopathy (HIE) than in those infants with no or mild HIE and concluded that the brain NA store may be exhausted in some cases and the response pattern of brain NA to asphyxia may be responsible for the clinical symptoms.

In a previous study on the cerebrospinal fluid NA content in new-born infants recovering from perinatal asphyxia we found an increase in NA level from 2.61 ± 1.45 to 10.86 ± 4.02 $\mu\text{g/l}$ ($p < 0.001$) in full-term and a decrease from 2.39 ± 0.72 to 1.25 ± 0.70 $\mu\text{g/l}$ ($p < 0.01$) in preterm neo-nates, respectively. These findings were regarded to indicate the importance of central noradrenergic structures in the organization of perinatal asphyxia-induced stress response. Furthermore, this pioneer study provided indirect evidence for the less developed brain NA reserves in preterm compared to full-term newborn infants who can be depleted more rapidly when subjected to hypoxemia [2].

With respect to the similarities in the study objectives, the clinical implications of the results obtained and in the conclusions drawn it would have been relevant to refer to our study.

References

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Bódis J, Sulyok E, Hartmann G, Ertl T, Csaba IF: Noradrenaline content of cerebrospinal fluid in preterm and term infants recovering from perinatal asphyxia. Acta Paediatr Hung 1984;25:343-346.

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Reply

J. Bódis and E. Sulyok are right. We regret this negligence.

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