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Abstract

Rebecca Nyengaard (Aarhus University)

Quantification of histopathological changes in the inner ear and electrophysiological assessment of hearing function in newborn piglets with hypoxic-ischemic-encephalopathy following perinatal asphyxia

Joint with Tommi Anttonen, Jens Randel Nyengaard, Kasper Jacobsen Kyng, Tine Brink Henriksen and Christer Zøylner Swan

Perinatal asphyxia results in hypoxic-ischemic-encephalopathy (HIE) in 1–6 per 1000 live human births, of which 10%–60% of affected infants die. 30%–100% of the survivors suffer mild to severe systemic and neurologic deficits, including sensorineural hearing loss (SNHL). Previous descriptive histological studies have suggested that perinatal asphyxia-induced SNHL is due to drastic cellular damage occurring in the central and/or peripheral auditory system. However, quantified histopathological evidence of HIE-induced SNHL related to electrophysiological assessment of auditory pathways is limited.

In order to study cellular damage in the inner ear following perinatal asphyxia, we applied a piglet survival model of perinatal HIE. HIE was induced in 15 Danish Landrace <24 h old piglets.

Auditory brainstem response (ABR) was performed pre- and post-asphyxia to evaluate auditory pathway function and will be correlated to cellular changes in the inner ear and central pathways. The severity and tonotopical distribution of cellular damage will be determined based on morphological and immunohistochemically related signs of cell death on virtual slides from the inner ears of the piglets by using the physical fractionator. Total hair cell number will be quantified using the optical fractionator. Our results seek to determine the extent to which inner ear damage in perinatal asphyxia contributes to early risk assessment of patients in risk of developing SNHL following perinatal asphyxia.