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Electroencephalography and brain damage in preterm infants

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Abstract Electroencephalography (EEG) is a sensitive method for detection of brain injury in preterm infants. Although the acute and chronic EEG changes are mainly non-specific regarding type of damage, they correlate with later neurological and cognitive function. In infants developing brain white matter damage, acute EEG findings include depression of background activity and presence of epileptic seizure activity. The chronic EEG changes associated with white matter injury and abnormal neurological development include delayed maturation, and presence of abundant Rolandic sharp waves. Cognitive limitations in preterm infants have been associated with changes in various sleep measures in EEG's recorded at full term. Continuous EEG-monitoring during neonatal intensive care shows that cerebral electrical activity during this vulnerable period can be affected by several extracerebral factors, e.g. cerebral blood flow, acidosis and some commonly used medications. For diagnosis of brain damage in preterm infants with neurophysiological methods, a combination of early continuous EEG monitoring during the initial intensive care period and full EEG, performed at later stages, is probably optimal.
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1. The normal EEG of the very preterm infant

Our knowledge about early postnatal EEG in healthy preterm infants has increased considerably during the last few years. Studies in extremely preterm infants are methodologically difficult due to the vulnerability of these infants during their first days of extrauterine life. Study populations are thus rather small. Nevertheless, a few studies have managed to include very immature infants without cerebral pathology on cranial ultrasound or magnetic resonance imaging and with normal long-term follow up.

Extrauterine life is not a normal condition in preterm infants, and therefore data on "normal" postnatal development of EEG in extremely preterm infants is lacking. Serial EEG's of preterm infants without brain damage who developed normally give

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the closest approximation of normal EEG development. However, some features of the EEG of healthy preterm infants at full-term, e.g., sleep-state variables, may differ from the EEG of term newborns [1,2].

Postconceptional age (PCA) is a commonly used term in EEG studies to describe the infant's degree of maturity. It is often defined as gestational age (GA) in weeks at delivery plus postnatal age in weeks. However, some authors use other terms, e.g. conceptional age and postmenstrual age. In this review we have chosen to use the authors' own terminology regarding the infant's maturity.

Magnetoencephalography (MEG) is an advanced non-invasive method that can be performed on normal fetuses. It is likely that MEG-data in the future can add knowledge on the normal neurophysiological development in fetuses during the last trimester. Furthermore, MEG also opens new perspectives regarding evaluation of fetal cerebral function in pregnancies with complications affecting the fetus, e.g. intrauterine growth restriction [3].

One dominant feature of the extremely preterm infants EEG is discontinuity, i.e. the EEG contains periods with bursts of activity mixed with periods with attenuated low-voltage activity (interburst interval, IBI) [4]. The duration of EEG bursts increases with gestational age and in preterm infants IBI decreases with increasing gestational age [5]. One study of 15 infants with EEG's recorded at 21—26 weeks postconceptional age suggests that it might be relevant also for the most immature infants [6].

In 10 infants born at 24—26 gestational weeks, EEG's were recorded during the first five days. Nine infants had normal outcome at 3 years of age. The EEG was discontinuous in all infants, and dominated by bursts of high voltage delta activity. The bursts were mainly synchronous, bursts with amplitude >50 μV could last for periods up to 83 s. No infant had maximum burst intervals (amplitude <15 μV) exceeding 1 min. Crude sleep-state organization was present at 25 weeks gestation [7]. In another study, including 17 slightly more mature infants recorded at a 26—28 weeks conceptional age, EEG background was also mainly discontinuous. Synchronous bursts of activity (amplitudes ≥ 30 μV) appeared with up to 3 min duration, and there was an occipital predominance of activity. No infant had inter-burst intervals exceeding 46 s. The dominating EEG frequency was delta activity with superimposed alpha, beta and theta. Sleep state differentiation could be seen at 26 weeks conceptional age [8].

Some maturational EEG features can be considered "normal" at some gestational ages, but abnormal if present at later stages. Rhythmic theta activity appearing in the temporal regions "temporal sawtooth" is associated with normal outcome when present at 27—30 weeks postmenstrual age, but an abnormal EEG feature if present a few weeks later [9]. Temporal sharp waves may also appear in the EEG during the first weeks of life in infants born at 31—32 weeks gestation. However, if abundant, or persisting, they are associated with brain damage [10] (Fig. 1).

2. Perinatal morbidity and preterm EEG

Perinatal infection and inflammation is a common antecedent of preterm delivery. Moreover, fetal inflammation has been shown in several studies to correlate with development of white matter damage in preterm infants. We are aware of only one small study investigating a possible relationship between endotoxin exposure and early EEG in preterm infants, and the results were inconclusive [11].

Changes in cerebral blood flow are associated with development of brain damage in preterm infants. There seems to be a relation between early EEG activity in preterm infants and global

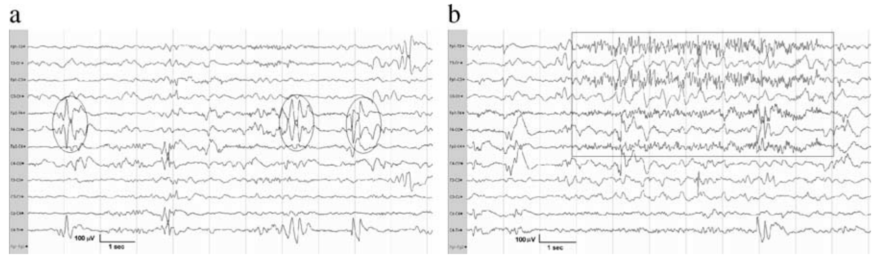

Figure 1 a (left) and b (right). This infant had a gestational age of 24 weeks and developed a right-sided intraventricular and intraparenchymal hemorrhage (IVH 4), and later frontal PVL on the right side. The EEG was recorded at 28 weeks gestational age showing abundant positive temporal sharp waves over the right hemisphere (left), and a short bifrontal seizure (right).

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