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# Benign familial neonatal convulsions: abnormal intrauterine movements, provocation by feeding and *ICTAL* EEG

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An infant with benign familial neonatal convulsions had abnormal movements during the last 2 months of pregnancy suggestive of intrauterine seizures. His postnatal seizures, one of which was captured by electroencephalography, had both partial and generalized features. Most seizures appeared to be provoked by feeding.

*Key words:* intrauterine; seizures; benign; familial; neonatal; provocation.

Abnormal intrauterine movements reported by mothers of neonates with seizure disorders are usually assumed to represent intrauterine seizures. They seem relatively rare, but have been described with cortical malformations (including Ohtahara syndrome), pyridoxine dependency, maternal opiate withdrawal and following prenatal insults. This paper describes similar movements in a child with benign familial neonatal convulsions. Seizures in our patient had partial and generalized features and appeared to be provoked by feeding.

Following an apparently uneventful pregnancy, the infant was delivered by ventouse at term, having had a low heart rate on the cardiograph. The Apgar rating was 9 at 1 and 5 minutes and birth weight was 3.61 kg. The infant fed well from birth. On day 3 of life he developed hemiclonic and multifocal clonic seizures lasting 1–2 minutes. Some were preceded by version of the eyes and head and asymmetric tonic posturing. The majority occurred immediately after breast feeds. Between seizures the neurological examination was normal. Two electroencephalogram (EEG) studies showed normal background activity. During one, a seizure was recorded where he screamed, developed eyelid flickering, followed by clonic facial twitching and then generalized twitching, and finishing with clonic twitching of the right side of the face and right arm. At the start of the seizure, the EEG showed a build-up of high-voltage slow-wave activity in the right anterior temporal region, which then spread to the left and became generalized. As the seizure progressed to right-sided clonic twitching, spike and wave activity was seen lateralized over the left anterior temporal region. In the post-ictal phase, the EEG was relatively flattened with occasional bursts of sharp/slow-wave activity seen generalized, and also independently, over both hemispheres. In view of the presumed diagnosis, no treatment was given and the seizures ceased spontaneously on day 10. He has since developed normally, up to the age of 14 months.

According to his maternal grandmother, his mother had identical episodes from days 1–10, which were ascribed to colic, without any problems since. There had been no abnormal movements during her pregnancy. She herself had never had any seizures, but her own mother had a history of convulsions in infancy.

On questioning, his mother described unusual repetitive prenatal movements with a rhythm similar to those of the fits seen postnatally, with an onset at 7 months, that lasted for 1–2 minutes and recurred in single episodes several times weekly, usually at night, for the rest of the pregnancy.

The assumption that abnormal intrauterine movements represent seizures is unproven in the absence of concomitant EEG recordings, and seems likely to remain so. However, the recognition by his mother of their similarity to the postnatal seizures is strong

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circumstantial evidence. One other case of benign familial neonatal convulsions has been mentioned in which the mother had possible intrauterine seizures[1]. This is the first detailed description of these movements.

In other respects the clinical features are typical of benign neonatal familial convulsions. Although classified as a generalized seizure syndrome, recent reports and the clinical and EEG description of seizures in this child, also suggest a focal component[1–3]. The EEG features, highlighted by Ronen and colleagues[1], of slow waves followed by polyspike discharges which become intermittent with suppression periods in between, occurred in our patient, although there was no initial suppression of amplitude. The presence of both partial and generalized seizures may be influenced by the immaturity of the neonatal brain, where inter- and intrahemispheric transmission is reduced by the lack of myelination of the corpus callosum and cortical white matter. In families where later seizures have occurred, the majority have been generalized, but it is unclear whether these are primary or secondary and one case with late partial seizures has been described[4].

In our patient, it is interesting that seizures seemed provoked by feeding. This has not been described before. Feeding is mediated by subcortical pathways, as demonstrated by babies with hydranencephaly. As the EEG pattern suggests a subcortical or brain-stem origin[1], feeding could be provocative through its involvement of nearby pathways. In some patients, seizures in later like can also be provoked by auditory stimuli or significant emotional stress, which again may involve subcortical pathways[1].

Since anticonvulsant treatment acts purely to suppress seizures, and can have major side effects on respiration and feeding, it was appropriate to withhold treatment in this child.

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