NORMAL AND ABNORMAL NEONATAL AND PEDIATRIC EEG

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Pediatric and Neonatal EEG has a wealth of normal variants and abnormal findings. Recognizing that slow activity is normal for particular ages and states will avoid misdiagnosing a normal child or neonate as having pathologic brain activity. In addition, accurate characterization of abnormal background patterns can be helpful for diagnosing certain developmental malformations or inborn errors of metabolism. Finally, identification of normal and abnormal patterns after hypoxia ischemia is important for prognostication.

Background activity is slowest in the neonatal period and gradually increases in frequency with increasing age. Development of faster frequency activity occurs first in the posterior regions and later anteriorly, mirroring the pattern of brain organization and myelination. The posterior basic rhythm is not seen well until 4 months of age, when it is 3.5-4.5 Hz. By 12 months, it is 5-6 Hz and it keeps increasing until it is 8Hz at 3 years (fast enough to describe as an occipital alpha rhythm). There is a slower increase to 9Hz at 9 years and 10 Hz by 15 years of age. This rhythm may be seen in the central and temporal regions in children.

Other normally slow patterns include frontal and frontocentral theta activity and posterior slowing of youth. The frontal and frontocentral theta activity is usually 6-7 Hz, less than 15μV and can be rhythmic. It is enhanced by drowsiness, strong emotions and mental tasks. Posterior slowing of youth consists of normal 2.5-4.5 Hz slowing intermixed with the posterior basic rhythm that rarely lasts more than 3 seconds and occurs in up to 2% of the eeg. Characteristics that suggest the activity is abnormal include polymorphic, asynchronous, unilateral slow activity. In addition if the amplitude is more than 1.5 times the child's occipital alpha rhythm/posterior basic rhythm or occurs with eyes open, the slow activity is more likely to be abnormal.

Children also have different background features in drowsiness and sleep that can be mistaken for abnormal activity. It is especially important to recognize that infants and children can appear to be awake clinically when they have drowsy features on eeg. In addition, they can have prolonged periods with drowsy eeg patterns. Hypnagogic hypersynchrony (as the child starts to fall asleep) starts with generalized, high amplitude, monomorphic, rhythmic slow activity that is 3-4 Hz in early infancy and 4-5 Hz in children.

Closer to sleep onset, the posterior basic rhythm is replaced by 4-6 Hz frontocentral and central monomorphic, rhythmic activity that can be up to 200 μV. Some children can have slowing of the posterior basic rhythm and an increase in random occipital high amplitude 3-4 Hz activity. There may also be bursts of paroxysmal 100-350 μV anterior activity. This should not be mistaken for epileptiform activity, particularly when it is notched or has a spike that is very low amplitude relative to the slow waves.

Awakening can result in the rhythmic, slow activity of hypnapompic hypersynchrony. A three month infant will have a high amplitude slow wave followed by rhythmic hypersynchronous slowing. By seven months, the initial slow wave is followed by diffuse slowing and high amplitude frontocentral 4-4.5 hz monomorphic slow waves. With increasing age, the frontocentral activity becomes rhythmic and the frequency increases until it reaches the adult 8-14 Hz frequency. After the rhythmic fast activity, some children can have generalized monomorphic semi-rhythmic slowing that is initially more prominent frontally but then shifts to the posterior regions prior to appearance of the posterior basic rhythm.

There is also maturation of eeg activity in sleep. Vertex waves appear at 5-6 months and while maximal at the vertex, can extend more anteriorly, most prominent frontocentral. Preschool age children may have vertex waves that are high amplitude, sharp contoured and occur in runs. Sleep spindles appear at 1-2 months of age and do not have the adult fusiform appearance. They can be asynchronous up to two years of age. Children may have spindles that are 10-12 Hz and located frontally. If the spindle frequency is persistently slower on one side, there may be an ipsilateral lesion. Unilateral spindles suggest a lesion on the contralateral side. Morphine and halothane anesthesia can cause 10 Hz frontal spindles that are continuous and unreactive. Occipital slow transients are seen in children in the transition between light and deep sleep. These high amplitude slow waves can have a cone or diphasic morphology.

There are also normal rhythmic or sharp features of the eeg that may be mistaken for abnormal activity in children. Occipital intermittent rhythmic delta activity (OIRDA) must be interpreted in the clinical context. It can be seen in normal children or children with absence epilepsy. Lambda waves are surface negative polyphasic occipital sharp waves or spikes. They occur during scanning of a complex picture. They can be distinguished from epileptiform activity by their disappearance with eye closure or staring at a blank page. Fourteen and six Hz positive bursts are a more monomorphic pattern with sharp features that occurs in drowsiness and light sleep. They are arch shaped with a rounded negative wave alternating with a spiky positive wave and can occur in trains for up to 1 second. They are maximal in the posterior temporal region and can be asynchronous or unilateral. They may be mistaken for epileptiform activity when a single waveform is present.

There are patterns of abnormal activity that are seen more commonly in certain neurologic disorders. Lissencephaly can have high amplitude alpha and 14-16 Hz beta activity. Even faster 16-24 Hz high amplitude fast activity that is unreactive to eye closure is present in infantile neuroaxonal dystrophy. Angelman syndrome can have high amplitude 1-3 Hz notched slow waves with a frontal predominance. Pyridoxine dependency can have high voltage delta slowing/spikes with periods of asynchronous attenuation, burst suppression, or hypsarhythmia. Krabbe's disease can have a disorganized background with high amplitude slow waves and multifocal spikes. There are neonatal disorders that can present with a burst suppression pattern on eeg: nonketotic hyperglycinemia (glycine encephalopathy), acute citrullinemia, propionic acidemia, neonatal adrenoleukodystrophy, molybdenum cofactor deficiency, sulfite oxidase deficiency, multiple carboxylase deficiency and patients with early infantile epileptic encephalopathy (Ohtahara syndrome) or early myoclonic encephalopathy. Some conditions can have a hypsarhythmia pattern: nonketotic hyperglycinemia (glycine encephalopathy), some peroxisomal disorders, pyruvate carboxylase deficiency. Neonates with maple syrup urine disease or propionic academia can have a comb-like rhythm consisting of monomorphic 5-7 Hz rhythmic central and parasagittal activity. There are infantile disorders with burst suppression: biotinidase deficiency. Infant disorders with hypsarhythmia: phenylketonuria. Patients with organic acidurias in infancy can have a normal EEG in between episodes of metabolic crises. Infantile neuronal ceroid lipfuscinosis patients demonstrate progressively less activity on eeg early in the disease.

As with any eeg, normalcy in a neonatal eeg is age dependent. Tracé discontinu is a normal discontinuous pattern in preterm infants, consisting of high voltage (50-300 µV pp) activity alternating with low voltage interburst periods (< 25 µV pp). The interburst interval duration decreases with age. This pattern is replaced by continuous activity in active sleep around 31-34 weeks post menstrual age (PMA). Continuous activity occurs next in wakefulness around 34 weeks PMA and last in quiet sleep around 37-40 weeks PMA. Tracé alternant appears discontinuous but is a pattern in quiet sleep in term infants with bursts of 50-150 µV delta activity alternating with 25-50 µV theta activity. An excessively discontinuous eeg has interburst interval activity < 25 µV pp with a duration that is too long for PMA. Burst suppression is defined by an invariant pattern with no normal activity in the bursts and an interburst interval amplitude <5 µV pp. Another pattern besides burst suppression that is associated with increased morbidity and severe neurologic deficits is low voltage suppressed. This has no normal features and the voltage is < 10 µV pp and is invariant. Normal features in the eeg include delta brushes (maximal 32-34 weeks PMA), rhythmic temporal theta (maximal 29-32 weeks PMA), anterior dysrhythmia (term infant), and encoches frontales (term infant).

There are differences in definitions for epileptiform activity and seizures in neonates. Spikes have a duration < 100 msecs. Sharp waves are 100-200 msecs. These transients should be quantified during wakefulness and active sleep since there can be sharply contoured activity in the discontinuous quiet sleep periods. A recent guideline suggests that abnormal negative sharp waves be defined by: focality (region or hemisphere), atypical location (frontal, vertex, occipital), more than 13/hour (term) or 11/hour (preterm), and occurring in runs or trains. Positive sharp waves occurring > 1.5/hour (term) or >3/hour (preterm) can be abnormal. A seizure is defined as an evolving pattern with a minimum 2µV pp amplitude and lasting at least ten seconds. There must be 10 seconds between seizures to consider them separate seizures. Status epilepticus is defined most commonly as seizures occurring for more than 50% of a particular time period. Brief rhythmic discharges evolve like a seizure but are under 10 seconds in duration.

There is a recent update on brain death criteria for children. It is suggested that the first brain death evaluation be performed 24 hours or later after insult and they must be more than 35°C. For children >30 days old, two examinations must be performed. Ancillary studies such as EEG can not substitute for an exam but can decrease the time between exams to less than the standard 12 hours. For a term neonate, two examinations, with the

initial exam at 24 hours of life or older and the second 24 hours later, are more reliable than EEG to demonstrate brain death. If an EEG demonstrates electrocerebral inactivity and the infant meets clinical criteria for brain death, further studies are not needed.

Prognosis after neonatal hypoxia ischemia is based upon the eeg pattern at a particular time after injury. An inactive EEG is more predictive of severe disability or death 12 hours or more after insult. Lack of sleep wake cycles at 48 hours of life is also associated with poor outcomes. A normal EEG is most predictive of good outcome if this pattern is seen between 24-36 hours of life. Serial EEGs can refine prognostication since improving EEG background activity in the first week of life is associated with better outcomes.

There are some differences in eeg patterns and prognosis for children and neonates with hypoxia-ischemia after hypothermia treatment. Children with unreactive, discontinuous or suppressed eeg patterns have a PPV of 88% for severe disability or death. Neonates with a burst suppression or inactive pattern after 24 hours of life either die or have severe disability. The presence of sleep wake cycling by 120 hours of life is associated with a normal outcome.

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