Practice guide to neonatal seizures

Maria Chalia

Topun Austin

Prevalence

Neonatal seizures occur in 1.5–3.5 per 1000 term live births, with a higher incidence (10–130 per 1000) in preterm live births. They often remain undiagnosed, accurate diagnosis is highly dependent upon clinical expertise and the availability of monitoring equipment. There is a strong association between neonatal seizures and adverse neurodevelopmental outcome. Up to 50% of infants with seizures will go on to have some form of long term neurological sequelae, e.g. epilepsy, cerebral palsy, and/or intellectual disability. Effective monitoring leads to earlier identification, and supports effective management.

Definition and classification

Seizures are secondary to an excessive synchronous depolarisation of neurons within the central nervous system. The neurophysiological definition is of a sudden, stereotypical and repetitive episode of abnormal electro-cortical activity with a peak-to-peak amplitude of at least 2μV, a minimum duration of 10 seconds, and dynamic evolution over time with clear beginning, middle, and end.

Neonatal seizures are a separate clinical entity compared to those of older children and adults and also vary when comparing preterm with term infants. This is primarily due to a series of neuroanatomical and neurophysiological maturing processes of the developing brain. Neonatal seizures can be clinically difficult to differentiate from spontaneous normal movements and infantile mannerisms. Although the International League Against Epilepsy has updated its classification incorporating both adult and neonatal seizures, the Volpe classification of the different types of seizures remains the one widely accepted (Table 1).

Aetiology and differential diagnosis

There are a variety of potential causes and underlying pathologies which are summarised in Table 2. Identifying the underlying cause of neonatal seizures is essential towards their effective management.

Maria Chalia *MD MRCPCH, Specialty Registrar in Neonatology at neoLab, The Evelyn Perinatal Imaging Centre, Rosie Hospital, Cambridge University Hospitals NHS Foundation Trust, Cambridge Biomedical Campus, Cambridge, UK. Conflicts of interest: none.*

Topun Austin *BSc MBBS MRCP MRCPCH PhD, Consultant Neonatologist at The Evelyn Perinatal Imaging Centre, Rosie Hospital, Cambridge University Hospitals NHS Foundation Trust, Cambridge Biomedical Campus, Cambridge, UK. Conflicts of interest: none.*

How to diagnose neonatal seizures

Clinical signs may be subtle or lacking entirely. Neonatal seizures are separated into those with electroencephalographical (EEG) evidence with or without clinical manifestation. The phenomenon of electroclinical dissociation (ECD) where there is electrical seizure activity with no clinical correlate is seen in term and preterm infants.

From a clinical perspective, rhythmic clonic movements, generalised myoclonic jerking as opposed to focal or multi-focal tonic posturing, eye deviation or rhythmic eye movements are more likely to be of epileptic nature. Similarly, it is important to be able to recognise a series of paroxysmal non-epileptic phenomena seen in the newborn and often mistaken as seizures; for example jitteriness of newborn infants, which can be associated with underlying hypoglycaemia, hypocalcaemia/hypomagnesaemia or neonatal abstinence syndrome, but can also be idiopathic and resolve gradually in the first 2 months of life.

The ability of clinicians to diagnose seizures based on clinical signs alone is poor. Studies show that on average only 50% of clinicians correctly identified seizures on clinical grounds alone. There is poor inter-observer agreement and no difference in diagnostic performance between medical and non-medical staff.

The gold standard in accurate diagnosis of neonatal seizures is the multi-channel video-EEG. Video-EEG provides the identification of clinical manifestations associated with detected abnormal electrical activity and also an insight on medical interventions, overall infant behaviour, and artefact (secondary to patting, suctioning, bag-valve mask inflation breaths, etc.), thus facilitating the EEG interpretation. However, if there is limited availability or lack of neurophysiology support, an amplitude integrated EEG (aEEG) can be applied as a screening and monitoring tool.

In a study of just over 800 neonatal seizures it was shown that up to 80% occur in the cross-cerebral region (C3 to C4), which is usual location for single-channel aEEG application. Despite the principal limitations of low number of electrodes (2–4) and attenuation of high (>15 Hz) and low (<2 Hz) frequencies, aEEG should be applied in at risk infants with the view to complete the investigation with multi-channel video-EEG.

Investigation of neonatal seizures

Any infant presenting with a possible seizure should have their blood glucose and electrolytes measured immediately and cardiorespiratory supportive measures initiated as a matter of urgency. The second step is to confirm their electrographic presence and identify the underlying cause. At this point one should consider which are the appropriate initial and more advanced investigations required. Our approach to investigation, divided in first and second line investigations are summarised in Table 3.

Treatment options

Following diagnosis it is necessary to consider the use of anti-epileptic drugs (AEDs). There is limited evidence to guide clinicians about which AED is best in any given situation. Most of the evidence originates from research based on clinically observed seizures rather than EEG-based studies.

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Types of neonatal seizures

Type	Clinical Manifestation	Frequency and EEG correlate
Subtle	Eye flickering, eye deviation, stare, blinking. Apnoea. Cycling, boxing, stepping, swimming movements of the limbs. Mouthing, chewing, lip smacking, smiling.	~50% of neonatal seizures; EEG correlation is variable; EEG changes occur most likely with ocular movements.
Tonic	Stiffening, decerebrate posturing	~5% of neonatal seizures; EEG correlation is variable.
Clonic	Repetitive jerking, distinct from jitteriness. May be uni- or multifocal.	25–30% of neonatal seizures; EEG correlation highly likely especially if focal.
Myoclonic	Myoclonic jerks; sleep myoclonus can be benign.	15–20% of neonatal seizures. EEG normal if focal or if sleep myoclonus.

Table 1

However, treatment is warranted. Evidence from animal and large clinical studies show an association between ongoing seizure activity and brain tissue injury and poor neuro-developmental outcomes. Early and effective treatment of even clinically occult seizures i.e. pure electrical seizures even those with brief electrical discharges is likely to optimise longer term outcomes. In Figure 1, we offer a comprehensive management approach for neonatal seizures.

Causes of neonatal seizures

	Frequency
Hypoxic-ischaemic encephalopathy	37–57%
Ischaemic infarction or sinovenous thrombosis	7–18%
Intracranial haemorrhage	5–17%
– subdural	
– subarachnoid	
Infections	3–20%
– encephalitis, meningitis, brain abscess	
– intrauterine (rubella, toxoplasmosis, syphilis, viral – such as cytomegalovirus, herpes simplex virus, human immunodeficiency virus, coxsackie virus B)	
Metabolic	3–19%
– hypoglycaemia	
– hypocalcaemia	
– hypomagnesaemia (alongside hypocalcaemia or isolated)	
– hyponatraemia	
– hypernatraemia	
Inborn Errors of Metabolism	1–11%
Congenital malformations of the central nervous system	4–11%
Early onset epileptic encephalopathies	1%
– benign neonatal seizures (familial, non-familial)	
– early myoclonic encephalopathy, early infantile epileptic encephalopathy (Ohtahara syndrome)/neurogenetic encephalopathies (e.g. CDKL5, KCNQ2, SCN2A)	
Intoxication	0.5%
– withdrawal	
– anaesthesia	

Table 2

Investigations of suspected neonatal seizures

First Line

1. Initial Lab Investigations

Blood gas and Glucose
Ionised Calcium, Sodium, Phosphate, Magnesium
Liver and renal function
Serum Bilirubin
Full blood count, C-reactive protein
Blood and urine culture
Cerebrospinal fluid microscopy and culture
Consider saving cerebrospinal fluid for metabolic tests, i.e. glycine
Consider herpes simplex virus

2. Initial neuro-monitoring and neuro-investigations

Cranial ultrasound aEEG
multi-channel video EEG

Second Line

1. Lab Investigations

Thyroid function test
Ammonia (urea cycle defects)
Plasma amino acids (amino acid disorders)
Urine amino acid and organic acids (amino/organic acid disorders)
Urate (molybdenum cofactor deficiency)
Acylcarnitine (fatty acid oxidation defects)
Very long chain fatty acids (peroxisomal defects)
Biotinidase (biotinidase deficiency)
Maternal and neonatal urine sample
Consider congenital infections
Consider genetic testing

2. Further Imaging/Investigations

Magnetic Resonance Imaging
Ophthalmological assessment
Woods light examination

Table 3

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