GUIDELINES 2013

The following guidelines[1] have been produced by the South East Thames Paediatric Epilepsy Group (SETPEG) to help paediatricians, paediatricians in training, paediatric neurologists and paediatric neurologists in training when investigating, diagnosing and managing children and young people with epilepsy and other paroxysmal disorders. They do not replace other teaching resources and those professionals using these guidelines are encouraged to attend the Paediatric Epilepsy Training (PET) courses run through the British Paediatric Neurology Association (BPNA).

1. The use of EEG in children
2. Use of brain imaging in children with epileptic seizures
3. Investigations in children with epileptic seizures,
    a) infants age 1-12 months
    b) children and adolescents
4. Further neuro-metabolic studies in children with epilepsy
5. Further assessments genetic and autoimmune in children with epilepsy
6. General guidelines for management of seizures in children > 1 year
7. Status Epilepticus: Management protocol
8. References
9. SE Epilepsy Interest Group (contributing group May 2005, 2007, 2012)

[1] Drs Elaine Hughes and Ruth Williams have produced these guidelines with the SETPEG. First Edition November 2005 (revised June 2007, November 2007 and January 2013).

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USE OF EEG IN CHILDREN

A. GENERAL POINTS

1. The value of the EEG in paediatrics is predominantly to classify rather than diagnose epilepsy. Accurate classification may assist treatment decisions.

2. The EEG is not a screening tool.

3. Detailed clinical information provided in the referral/request may influence the way the EEG is carried out and its interpretation. Discussion between clinical and neurophysiology staff is recommended to:

   - optimise the usefulness of the EEG
   - to aid interpretation and the production of a helpful report.

4. It is important to be aware that people with definite epilepsy may have repeatedly normal EEGs and conversely that interictal EEG abnormalities may be seen in individuals who have never had a seizure.

5. The yield from sleep EEGs in children with focal epilepsy, some of the symptomatic generalised epilepsies and on occasions, idiopathic generalised epilepsy is significantly higher than from awake EEGs alone. If in doubt, discuss your request with the neurophysiology department.

6. Drugs may modify the EEG appearance.

B. WHEN IS AN EEG INDICATED?

1. After the second **afebrile** seizure

   - to provide a syndromic classification if possible, e.g. benign epilepsy with centro-temporal spikes
   - to guide future treatment choices, e.g.. in juvenile myoclonic epilepsy (JME)
   - * it may be considered after the first seizure, as per specialist advice (see NICE guidelines)

2. After a complicated febrile convulsion (more than 15 minutes duration, recurrent within 24 hours ,or asymmetrical).

3. In monitoring efficacy of treatment

   - Infantile spasms

- Landau Kleffner (LKS) and Epilepsy with Electrical Status Epilepticus in Slow Sleep (ESES) – will need sleep EEG
- In persistent status epilepticus

4. In the presence of an acute encephalopathy of unexplained aetiology.

5. In the presence of unexplained global deterioration in cognitive performance or dementia or specific cognitive decline, e.g. auditory agnosia, as in Landau Kleffner Syndrome (LKS)

   - to exclude non convulsive status
   - to exclude electrical status in sleep (ESES) – sleep EEG required

6. When a new seizure type or pattern of seizures becomes apparent, or to review the epilepsy syndrome diagnosis.

Where EEG is indicated in the initial investigation of children with epilepsy, NICE recommends it should be carried out within 4 weeks of request being made

C. WHEN IS A SLEEP EEG INDICATED?

1. In the presence of recurrent seizures and a normal awake EEG

   - where seizures arise focally, a sleep EEG increases the yield of epileptiform abnormalities from 50% to 85%
   - if generalized seizure disorder, a sleep EEG may be diagnostic if awake EEG is normal.

2. Where LKS or ESES is suspected (see above).

3. To follow up response to treatment in babies with infantile spasms.

There is a reasonable body of evidence supporting the request for a sleep EEG for every child with a history of seizures, except those with typical absences. Sleep recordings are now commonly obtained using Melatonin induced sleep rather than sleep deprivation so that a waking state EEG can also be obtained during the same recording period. In idiopathic generalised epilepsies such as juvenile myoclonic epilepsy or juvenile absence epilepsy, sleep deprived recordings may increase the yield of abnormalities.

D. WHEN MAY CONTINUOUS VIDEO EEG MONITORING (TELEMETRY) BE REQUESTED?

1 When there is persistent uncertainty about the nature of clinical events despite appropriate investigations, and these events occur frequently enough to be captured in this way (in practice this means events occurring 2-3 times per week at least).

2 When a child is being referred for evaluation for possible epilepsy surgery.

3 To detect or determine the frequency of subtle ictal events.

Where telemetry is being considered, it is recommended that it should be discussed with neurology or neurophysiology first, to get the most information from the study

E. AMBULATORY EEG

Consider in certain circumstances, e.g. monitoring ESES.

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USE OF BRAIN IMAGING IN CHILDREN WITH SEIZURES

A. GENERAL POINTS:

1. MRI using an epilepsy protocol is the preferred imaging modality but CT (and cerebral ultrasound) may have a role in the acute situation
2. MRI in young children will require a GA or sedation and this has implications for anaesthetic resources
3. The yield of abnormal findings in MRI is related to the definition of the MRI scanner, use of appropriate scanning protocols and the expertise of the reporting radiologist
4. Interpretation and reporting of Paediatric Neuroimaging requires special expertise and appropriate training in paediatric neuroradiology and review of scans is desirable in the context of complex epilepsy.

B. INDICATIONS (Non urgent MRI Brain Imaging):

1. All children with seizures, presenting under 2 years of age.
2. All children with focal seizures should have brain imaging except in some cases of benign partial epilepsy if clinical features and EEG are typical, e.g. BECTS.
3. All children who have had more than one afebrile generalized seizure with the exception of typical childhood absence epilepsy, juvenile myoclonic or juvenile absence epilepsy. Where these types of epilepsy fail to respond to first line treatment, imaging is also indicated.
4. Children with epilepsy and learning difficulties should **not** be excluded from this guideline but where the aetiology of the epilepsy appears clear, risk/benefit of GA for MRI needs to be carefully discussed.

MRI cannot be routinely used in children with implantable electrical devices, including VNS and cochlear implants. With VNS, head imaging is possible in certain situations in certain centres – must be discussed with a neuroradiologist.

Where brain imaging with MRI is indicated, NICE recommends that this is carried out within 4 weeks of request

INVESTIGATIONS IN CHILDREN WITH AFEBRILE EPILEPTIC SEIZURES OF UNKNOWN AETIOLOGY

A. INFANTS : AGE FROM 1 MONTH TO 24 MONTHS

- EEG and brain imaging as appropriate (see previous section)
- 12-lead ECG (and consider Echocardiography in infants with infantile spasms where tuberous sclerosis is a possible underlying diagnosis)
- Screening for infection (if appropriate)
- Routine biochemistry, including glucose, Mg, Ca, venous pH, LFTs
- Ammonia
- Urate (Both low and high levels are significant)
- Plasma lactate
- Plasma biotinidase
- Plasma acylcarnitines
- Plasma amino acids
- Urinary organic acids, aminoacids, dipstick fresh urine for sulphites and ketones
- Consider Fasting CSF lactate (not post-ictal sample), protein, glucose, glycine (paired with blood samples) in discussion with the Regional Service
- CGH arrays, and ask lab to save DNA
- Woods light examination and ophthalmology examination may be helpful

Pyridoxine and pyridoxal-P dependency

It is now recommended that an adequate trial of oral pyridoxine therapy should be given for any persisting seizure disorder starting within the first year of life regardless of EEG pattern, response to AEDs, CNS malformations or extra CNS features.

Beware : a single dose of IV pyridoxine is insufficient trial for some patients and breakthrough seizures may still occur with febrile precipitants.

Be aware that a small subset of children will only respond to pyridoxal phosphate.

Discussion with a paediatric neurologist is recommended outside the neonatal period.

B. CHILDREN AND ADOLESCENTS

- EEG and brain imaging as appropriate (see previous section)
- ECG should be carried out in all children presenting with convulsive seizure or collapse with calculation of corrected QT interval (QTc = QT/ RR) and a referral made to a cardiologist if the history is worrying and/or there is a long QTc.
- Routine haematology including a platelet count
- Routine biochemistry (glucose, electrolytes, calcium, magnesium and LFTs) at presentation if possible.

FURTHER NEURO-METABOLIC STUDIES IN CHILDREN WITH EPILEPTIC SEIZURES

The investigations listed previously in Section 3 should have been done. It is likely that these additional investigations will be planned **in conjunction with the Regional Paediatric Epilepsy or Neurology Service.**

- The extent of investigations will depend on the child's presentation **but**
- It is assumed that metabolic investigations will be in addition to brain imaging, EEG studies, ophthalmology review and genetics.
- No single seizure type can reliably predict metabolic disease but myoclonic seizures, especially fragmentary or multi-focal myoclonus, are more common.
- 'Progressive myoclonic epilepsies' constitute less than 1% of the epilepsy syndromes.

Neonatal period – first 6 months

Nb: it is likely that an infection screen will also be indicated in the acute situation

Differential diagnosis	Appropriate Investigation
Acidosis, haemorrhage Hypoglycaemia Hypocalcaemia Hypomagnesaemia	+ PH, clotting, transaminases, blood glucose, calcium, magnesium,
Hypo / hypernatraemia	sodium, acylcarnitines
Biotinidase deficiency	Biotinidase level
* Pyridoxine dependency	Trial of pyridoxine
* Maple Syrup Urine Disease Urea cycle disorders	Ammonia, plasma amino acids, organic acids
Pyridoxal phosphate dependent seizures	Trial of pyridoxal phosphate
* Non ketotic hyperglycinaemia	CSF:plasma glycine ratio
Glucose transporter deficiency	CSF:plasma fasting glucose ratio < 0.46
Serine biosynthesis deficiency	CSF:plasma serine ratio
Folinic acid responsive seizures Creatine deficiency syndromes (GAMT)	
Molybdenum co factor deficiency / sulphite oxidase deficiency	Urinary sulphite, plasma urate (low)

* EEG may be a clue

Nb: abnormalities on structural imaging eg. callosal agenesis, cerebellar hypoplasia are common in metabolic disorders

	Mitochondrial disorders	Non-acute CSF, plasma lactate, urine organic acids muscle +/or liver biopsy
	Peroxisomal disorders chain fatty acids	Very long
	Menkes	Hair light microscopy, copper studies, alkaline phosphatase (low)
infancy and early childhood	Fatty acid oxidation problems (MCADD - medium chain acyl-CoA dehydrogenase deficiency, VLCADD - very long chain acyl-CoA dehydrogenase deficiency)	Acylcarnitines, urine organic acids
	Lysosomal Storage disorders: Tay Sachs, Krabbes, Nieman Pick A, Infantile Gauchers, * Late infantile onset neuronal ceroid lipofuscinoses (Batten's) – see genetics section	White cell enzymes, including PPT and TPPI if Batten suspected; Buffy coat preparation (electron microscopy), DNA save, Consider Bone Marrow aspirate (NPA), urine oligosaccaharides
	* Alpers (PNDC – progressive Neuronal degeneration of childhood) –see genetics section	LFT's, ammonia and clotting; genetics may now mean invasive studies not necessary
	Glucose transporter deficiency	CSF:plasma fasting glucose ratio, DNA save
Childhood – adolescence	CDG syndrome	Transferrin isoforms
	NCLs – see genetics section	Vacuolated lymphocytes, Buffy coat (for EM), DNA save
	Mitochondrial disorders - see also genetics section	Plasma and CSF lactates, mt DNA mutations, muscle biopsy
	Gauchers	White cell enzymes, DNA save. Bone Marrow Aspiration
	Lafora body	Axillary skin biopsy (sweat glands)
	GM2 gangliosidosis/	White cell enzymes, DNA save
		Urine mucopolysaccharides white cell enzymes, DNA save, fibroblast

Oligosaccharidoses/Sialidosis

San Filippo

Recognition of the abnormal metabolic results is only the first step!

FURTHER STUDIES IN THE INVESTIGATION OF EPILEPTIC SEIZURES IN CHILDREN – GENETICS AND AUTOIMMUNE DISORDERS

A) GENETIC STUDIES

More than half of all epilepsies have a genetic basis, and clinically useful tests (ie where predictive value and clinical utility have been determined) are available for a small but growing proportion of these. Genetic evaluation begins with a comprehensive medical history, examination, EEG and MRI brain scan. Documentation of a three generation family history (not just for seizures) is a prerequisite for genetic testing.

Genetic studies may encompass metabolic as well as cytogenetic and DNA analyses and may involve sampling of blood, urine, CSF, muscle or skin. When the genome is analysed, this might be either genomewide eg comparative genomic hybridisation (CGH) array for recurrent structural genomic variants, or may be focused on specific gene mutations known to be associated with an epilepsy syndrome.

The decision to order a genetic test depends on a number of factors including the need to make a definitive and up-to-date aetiological diagnosis; the possibility that management might be changed by the result; and the implications for sibling recurrence or prenatal counselling.

The results of genetic tests can be positive (discovery of a pathogenic mutation or variant), negative (failure to find a pathogenic mutation), or ambiguous (discovery of a variant of unknown significance). Negative test results only indicate that the method used did not identify a mutation, and do not guarantee that a genetic cause is not present.

In the South-east region, the Genetics Department policy is to first screen all referral samples using genomewide CGH array, regardless of indication or suspected epilepsy syndrome diagnosis. A number of epilepsy syndromes are associated with recurrent CNVs eg 1p36 deletion, Wolf-Hirschorn etc. It is worthwhile requesting some DNA to be stored for future testing. Further testing should be guided by the phenotypic presentation, as below.

Dravet and Related Syndromes

Mutation and deletion (serial) testing is available for SCN1A, SCN1B, GABRG2 and PCDH19. Testing is not recommended for non-SMEI forms of GEFS+. Click here for the Request Form.

Infantile Spasms

No specific panel is available yet in the UK but several gene mutations are known

Epilepsy with Brain Malformations

Tests are guided by MRI appearance and clinical features. Not all epilepsy with brain malformation has a genetic cause

Epilepsy with neurodevelopmental comorbidity or dysmorphic features

CGH yield is high eg for autism spectrum, language or intellectual impairment. Note that if ring chromosome 20 is suspected, a conventional karyotype must be requested.

Angelman and Prader-Willi syndrome also need special testing for uniparental disomy (not revealed by CGH).

Epilepsy that is treatment resistant to appropriate first-line drug

CGH yield is high and may correlate with neurodevelopmental comorbidity.
Idic 15 can readily be detected by CGH array.

Common idiopathic epilepsies

Tests for Autosomal Dominant Nocturnal Frontal Lobe Epilepsy are available.
There are no useful clinical tests for common generalised or focal epilepsies.

Glut-1 (Glucose Transporter) Deficiency

Mutational analysis of the coding regions is available; this should currently be accompanied by the recommended paired CSF-blood glucose measurement.

Progressive and Mitochondrial Epilepsies

Gene testing is now available for a variety of disorders.

B) AUTOIMMUNE STUDIES

These may be indicated especially in the context of encephalopathic or neuropsychiatric presentation with seizures. This should be discussed with the Regional Paediatric Neurology Service.

MANAGEMENT OF EPILEPTIC SEIZURES IN CHILDREN

GENERAL PRINCIPLES

Children presenting with afebrile seizures should be referred to a paediatric epilepsy clinic. **Each Paediatric Department should therefore have a general paediatrician who has a special interest and responsibility for epilepsy as recommended in the NICE guidelines.**

Why?

- Because epilepsy is a common conditionwith a prevalence of 0.5% in the population.
- Because there is evidence of widespread diagnostic difficulties. . 20 - 40% of children referred with a diagnosis of epilepsy to a specialist service are found to have been misdiagnosed and there is no definitive diagnostic test.
- Because accurate diagnosis of the epilepsy and where possible of the epilepsy syndrome may assist management and influence prognosis.

NICE also recommends that children who have had a first afebrile seizure should be seen within 2 weeks by a specialist in the management of the epilepsies. NICE defined a 'specialist' in this context as a general paediatrician rather than a GP

TREATMENT PRINCIPLES:

- Begin with a single first line drug.
- Start low, go slow.
- Titrate to the lowest dose necessary to control seizures.
- If seizures continue on the maximum tolerated dose of first-line drug, consider substituting a second line drug. Best practice would be to either discontinue the first drug before starting the second choice or adding a second drug and then discontinuing the first.
- Management should be guided by the clinical response to treatment rather than by drug levels

- Always calculate the daily dose per kg (up to approximately 40kg) and then refer to adult guidance for treatment doses.
- Remember that the dose may need to be increased in line with the child's growth but may remain unchanged if the child is seizure-free.

PROVIDE INFORMATION FOR PARENTS AND CARERS IN CONJUNCTION WITH AN EPILEPSY NURSE SPECIALIST WHERE POSSIBLE

about the:

- use of rescue medication and care plans.
- risk eg. with bathing, road safety and sports.
- risk of SUDEP where appropriate.
- schooling related issues and liaison with school.
- gender specific issues eg in relation to fertility, contraception, pregnancy, driving regulations and careers
- appropriate support organisations.

DISCONTINUATION OF AEDs

- Discuss the risks and benefits of discontinuation of AEDs after 2 years seizure freedom.
- Overall risk of relapse is 1/3 but depends on syndrome & aetiology.
- Withdraw AED over 2-3 months except benzodiazeines & phenobarbitone (minimum 6 months).
- Withdraw one AED at a time.
- Update safety advice and a provide written plan.

MANAGEMENT OF EPILEPSY IN CHILDHOOD

RESCUE MEDICATION

- Only prescribe buccal midazolam or rectal diazepam for use in the community if the child has had a previous episode of prolonged or serial (3 episodes in 1 hour) convulsive seizures.

- Buccal midazolam should be used as first–line treatment in the community for a previous episode of prolonged or frequent seizures.
- Rectal diazepam can be used if preferred, or midazolam is not available.
- If IV access is established, use intravenous lorazepam.

TREATMENT PATHWAYS FOR SEIZURES IN CHILDHOOD

See NICE Guidance for treatment options according to seizure type or epilepsy syndrome

- When introducing treatment, refer to the general principles outlined above.
- In all cases, you must refer to the current licensing status and Children's BNF as age guidance varies with the type of epilepsy or epilepsy syndrome
- A basic metabolic screen (LFTs, Ammonia, plasma amino acids, acylcarnitines, lactate and urine organic acids) is indicated in children less than 2 years of age, before starting valproate. or if there is concern regarding a possible metabolic condition in children over this age.
- If using carbamazepine, where possible, use controlled release preparations.
- Carbamazepine is contraindicated in people of Han Chinese or Thai origin.

DRUG LEVELS

- Check if concerned about toxicity or compliance
- For Phenytoin, pharmacokinetics mean that small changes in dose can result in large changes in the blood level at critical doses.
- a Full Blood Count (FBC) and renal and liver function tests should be checked after starting treatment and then specific monitoring as guided by the paediatric formulary.

REFERRAL AND/OR DISCUSSION WITH THE REGIONAL PAEDIATRIC EPILEPSY OR PAEDIATRIC NEUROLOGY SERVICE:

- Where there is continuing diagnostic uncertainty.

- Where seizures fail to respond to treatment with 2 different anti-epileptic drugs given in adequate dosage - **see also below**
- In the presence of possible epilepsy related language, behavioural or cognitive difficulties
- With an 'epileptic encephalopathy'
- Where the child is aged less than 2 years.

REFERRAL FOR EPILEPSY SURGERY ASSESSMENT

- If there is focal structural pathology on an MRI brain scan refer **immediately** to the on-call paediatric neurosurgeon particularly if there are concerns about the nature of lesion, eg. a possible or definite tumour.
- With structural pathology on MRI, referral should be made to the Regional Paediatric Epilepsy Service at the time of identification of the abnormality and always after the failure of 2 appropriate drugs given in adequate dose.
- Without evidence of structural abnormality, referral should be discussed after the failure of 2 appropriate drugs given in adequate dose. Referral should be made earlier if there is evidence of a specific cognitive or neurological deficit, eg. acquired epileptic aphasia. Referral should be made after discussion with the Regional Paediatric Epilepsy Service.
- Further investigations are likely to include an epilepsy protocol MRI brain scan, neuropsychometry and video EEG telemetry. All children referred will be discussed in the Paediatric Epilepsy Surgery Meeting at Kings College Hospital regarding the options of resective surgery or VNS.

FURTHER INVESTIGATIONS SHOULD BE DISCUSSED WITH THE REGIONAL PAEDIATRIC EPILEPSY OR NEUROLOGY SERVICE

Discussion should take place when:

- 2 AEDs given in appropriate doses have failed to control the seizures.
- an EEG is not consistent with the diagnosis.
- there is a possible progressive disorder or there are other concerns.

- further neuroimaging (MRI) and EEG studies, including video EEG telemetry may be indicated together with neurometabolic and genetic studies

KETOGENIC DIET

- Refer if seizures have not responded to appropriate AEDs.
- Use as adjunctive therapy with AEDs, in those not suitable for surgery.

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TREATMENT GUIDELINE FOR AN ACUTE TONIC-CLONIC SEIZURE INCLUDING ESTABLISHED CONVULSIVE STATUS EPILEPTICUS

- **AIRWAY BREATHING CIRCULATION**

  GIVE HIGH FLOW OXYGEN
  MEASURE BLOOD GLUCOSE
  CONFIRM EPILEPTIC SEIZURE

- Prior treatment with rectal diazepam or buccal midazolam outside hospital must be counted. No more than 2 doses of benzodiazepines to be given in total.

IMMEDIATE IV ACCESS

1. LORAZEPAM 0.1mgs/KG IV
   (give over 30-60 seconds) (max. 4mgs)

↓

seizure continuing at 10 mins

↓

2. LORAZEPAM 0.1Mgs/KG IV
   (give over 30-60 seconds) (max. 4mgs)

↓

seizure continuing at 10 mins

↓

NO IV ACCESS

1. DIAZEPAM 0.5Mgs/KG PR
   or
   BUCCAL MIDAZOLAM 0.5mg/kg
   Maximum dose 10mg

↓

seizure continuing at 10 mins

↓

2. REPEAT ABOVE

↓

seizure continuing at 10 mins

↓

IV ACCESS

1. PHENYTOIN 18Mgs/KG IV OVER 20 MINUTES
   OR
   IF ALREADY ON PHENYTOIN GIVE PHENOBARBITONE 20Mgs/KG IV OVER 10 MINS
   (USE INTRAOSSEOUS ROUTE IF STILL NO IV ACCESS)

(Consider PARALDEHYDE 0.4ml/KG + SAME VOLUME OLIVE OIL IF NOT ALREADY GIVEN PR)

CALL ON-CALL ANAETHETIST OR INTENSIVE CARE UNIT

↓

Seizure continues after 20 minutes since commencing step 3

↓

4. RAPID SEQUENCE INDUCTION OF ANAESTHETIA WITH THIOPENTONE 4mgs/KG IV
   TRANSFER TO INTENSIVE CARE UNIT

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* 2005 Guidelines