Queensland Clinical Guidelines

Translating evidence into best clinical practice

Maternity and Neonatal **Clinical Guideline**

Neonatal seizures

Queensland Government

Document title:	Neonatal seizures
Publication date:	December 2022
Document number:	MN22.23-V4-R27
Document supplement:	The document supplement details development processes and implementation activities, and is integral to and should be read in conjunction with this guideline.
Amendments:	Full version history is supplied in the document supplement.
Amendment date:	December 2022
Replaces document:	MN17.23-V3-R22
Author:	Queensland Clinical Guidelines
Audience:	Health professionals in Queensland public and private maternity and neonatal services
Review date:	December 2027
Endorsed by:	Queensland Clinical Guidelines Steering Committee Queensland Maternity and Neonatal Clinical Network
Contact:	Email: Guidelines@health.qld.gov.au URL: www.health.qld.gov.au/qcg

Cultural acknowledgement

The Department of Health acknowledges the Traditional Custodians of the lands, waters and seas across the State of Queensland on which we work and live. We also acknowledge First Nations peoples in Queensland are both Aboriginal Peoples and Torres Strait Islander Peoples and pay respect to the Aboriginal and Torres Strait Islander Elders past, present and emerging.

Disclaimer

This guideline is intended as a guide and provided for information purposes only. The information has been prepared using a multidisciplinary approach with reference to the best information and evidence available at the time of preparation. No assurance is given that the information is entirely complete, current, or accurate in every respect.

The guideline is not a substitute for clinical judgement, knowledge and expertise, or medical advice. Variation from the guideline, taking into account individual circumstances, may be appropriate.

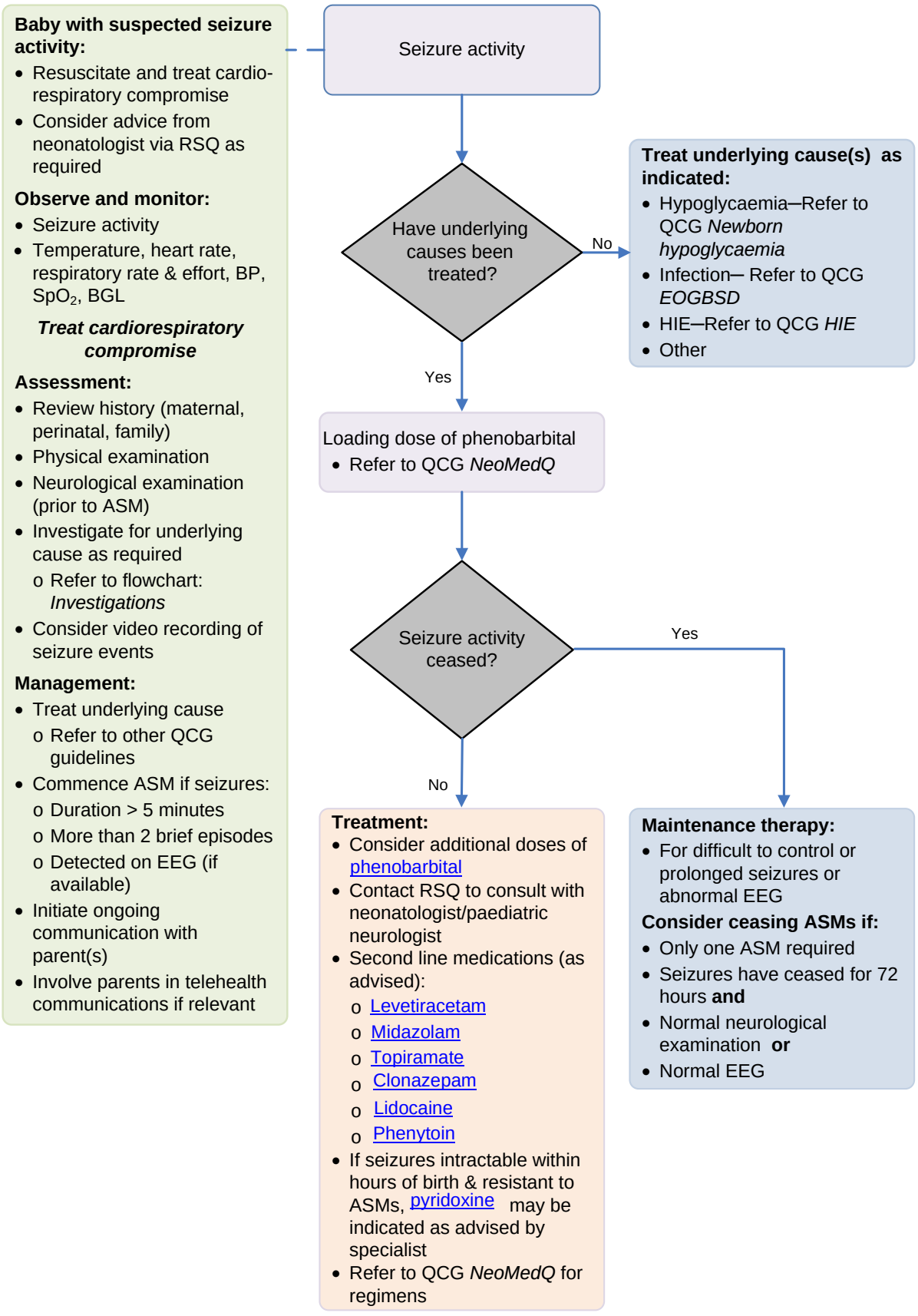
This guideline does not address all elements of standard practice and accepts that individual clinicians are responsible for:

- Providing care within the context of locally available resources, expertise, and scope of practice
- Supporting consumer rights and informed decision making, including the right to decline intervention or ongoing management
- Advising consumers of their choices in an environment that is culturally appropriate and which enables comfortable and confidential discussion. This includes the use of interpreter services where necessary
- Ensuring informed consent is obtained prior to delivering care
- Meeting all legislative requirements and professional standards
- Applying standard precautions, and additional precautions as necessary, when delivering care
- Documenting all care in accordance with mandatory and local requirements

Queensland Health disclaims, to the maximum extent permitted by law, all responsibility and all liability (including without limitation, liability in negligence) for all expenses, losses, damages and costs incurred for any reason associated with the use of this guideline, including the materials within or referred to throughout this document being in any way inaccurate, out of context, incomplete or unavailable.

Recommended citation: Queensland Clinical Guidelines. Neonatal seizures Guideline No. MN22.23-V4-R27. Queensland Health.2022. Available from: http://www.health.qld.gov.au/qcg

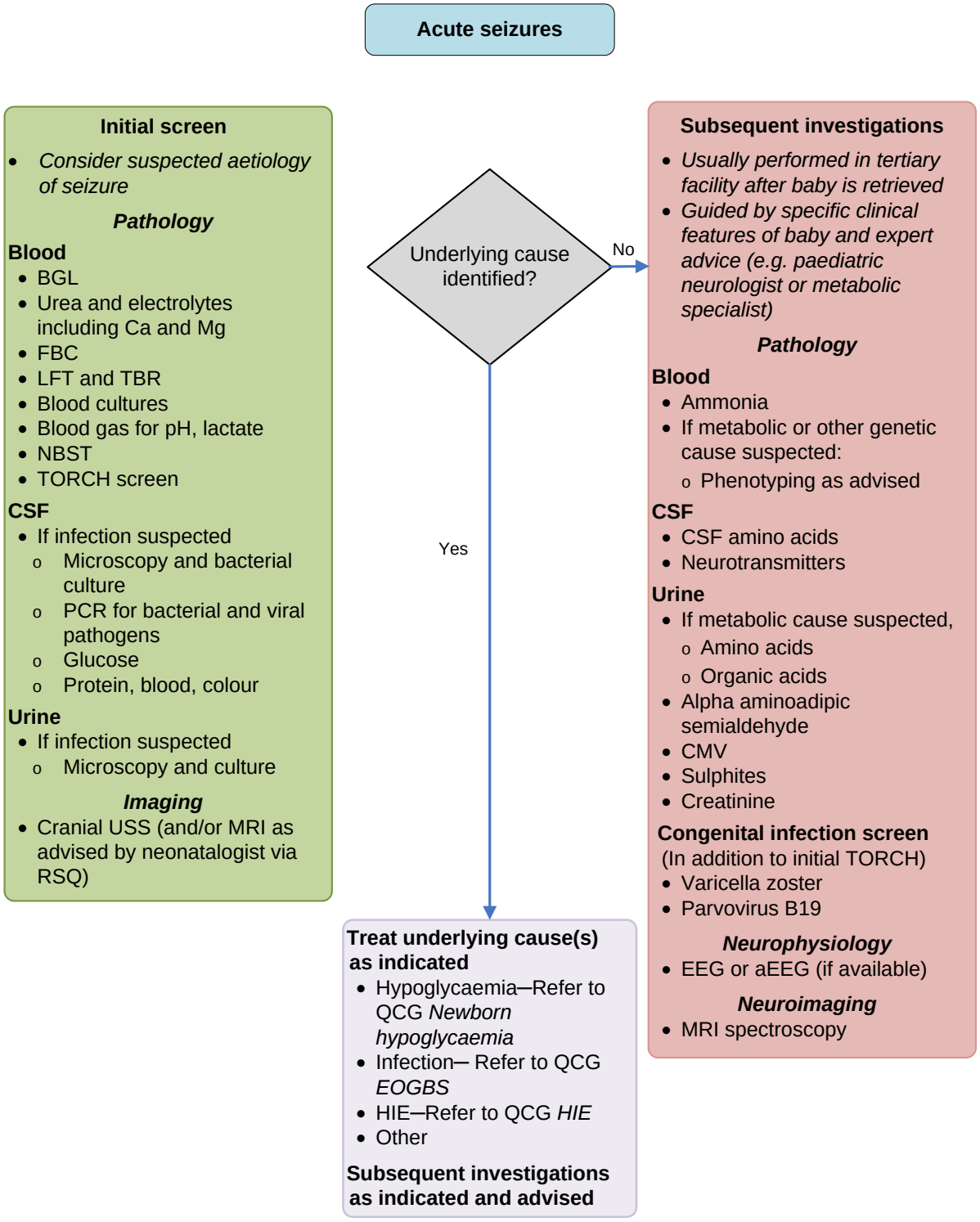
Flow Chart: Assessment and management of neonatal seizures



ASM: anti-seizure medication(s); **BGL:** blood glucose level; **BP:** blood pressure; **EEG:** electroencephalogram; **EOGBSD:** early onset Group B Streptococcal disease; **HIE:** hypoxic ischaemic encephalopathy; **IV:** intravenous; **QCG:** Queensland Clinical Guidelines, **RSQ:** Retrieval Services Queensland, **SpO_2** peripheral oxygen saturation; **>:** greater than

Flowchart: F22.23-1-V2-R27

Flow Chart: Investigations for neonatal seizures



aEEG electroencephalogram, **BGL:** blood glucose level, **CMV:** cytomegalovirus, **CSF:** cerebrospinal fluid, **EEG:** electroencephalogram, **EOGBSD:** early onset Group B Streptococcal disease, **FBC:** full blood count, **HIE:** hypoxic ischaemic encephalopathy, **HSV:** herpes simplex virus, **LFT:** liver function tests, **MRI:** magnetic resonance imaging, **NBST:** newborn bloodspot screening test, **QCG:** Queensland Clinical Guidelines, **SpO2:** peripheral capillary oxygen saturation, **TBR:** Total bilirubin, **TORCH:** toxoplasmosis, other (e.g. syphilis), rubella, cytomegalovirus, herpes simplex, **USS:** ultrasound scan

Flowchart: F22.23-2-V2-R27

Table of Contents

List of tables

Abbreviations

aEEG	Amplitude integrated electro-encephalogram
ASM	Antiseizure medication
BGL	Blood glucose level
EEG	Electro-encephalogram
CNS	Central nervous system
CSF	Cerebro-spinal fluid
EEG	Electroencephalogram
GMA	General movements assessment
HIE	Hypoxic ischaemic encephalopathy
HSV	Herpes simplex virus
MRI	Magnetic resonance imaging
IM	Intramuscular
IV	Intravenous
IVH	Intraventricular haemorrhage
PCR	Polymerase chain reaction
TH	Therapeutic hypothermia
TORCH	Toxoplasmosis, other (syphilis), rubella, cytomegalovirus, Herpes simplex
USS	Ultrasound scan

Definitions

Apoptosis	Process of programmed cell death.[1]
Acute provoked neonatal seizure	Seizure activity in the neonatal period occurring in the context of an acute illness/underlying condition.[2]
Electroclinical dissociation	Electrographical seizures with no clinical correlate.
Hydrocephalus ex-vacuo	Increased cerebrospinal fluid (CSF) volume but normal pressure when there is shrinkage of brain substance following damage to the brain caused by stroke or injury.[3]
Holoprosencephaly	Brain malformation resulting in varying degrees of lack of separation of the cerebral hemispheres.[4]
Hydranencephaly	Life-limiting brain anomaly where there are no cerebral hemispheres and the cranium is filled with cerebrospinal fluid CSF).[5]
Hypsarrhythmia	Abnormal inter-ictal pattern with electroencephalogram (EEG) high amplitude and irregular waves and spikes with background of chaotic and disorganised activity.[6]
Ictal	Relating to seizures.[7]
Lissencephaly	Rare, gene-linked brain malformation where there is absence of normal convolutions (folds) in the cerebral cortex and an abnormal, small head (although normal size at birth).[8]
Pyridoxamine 5'-phosphate oxidase (PNPO) deficiency	A very rare disorder of the PNPO gene resulting in ineffective metabolism of pyridoxine and pyridoxamine causing seizures soon after birth.[9]
Schizencephaly	A rare congenital anomaly where unilateral or bilateral clefts in the cerebral hemispheres develop that may be filled with cerebrospinal fluid.[10]
Seizure burden	Ictal (seizure) EEG activity expressed as summed electrographic seizure seconds within a given period of EEG recording.[2]
Spasticity	Muscular hypertonicity with increased resistance to stretch.[11]
Sudomotor	Sudomotor function refers to the autonomic nervous system control of sweat gland activity in response to various environmental and individual factors.

1 Introduction

Seizures are sudden paroxysmal and abnormal alterations in electrographic activity, and are a sign of neurological dysfunction.[12,13] They are a neurological emergency but can be difficult to diagnose and treat.[14]

Neonates are at especially high risk of seizures compared to other age groups.[14] The clinical presentation of neonatal seizures is variable.[13] This has led to under-diagnosis and occasional over-diagnosis.[15,16]

The majority of seizures demonstrated on video electroencephalogram (EEG) monitoring do not have overt clinical signs.[14] Conversely, newborn babies can have movements that can be mistaken for seizures, where the EEG is normal.[16]

1.1 Context

Table 1. Context

Aspect	Consideration
Background	• Seizures occur more frequently in neonatal period than any other time • Higher incidence is reported in preterm babies, especially lower gestational age and birth weight, but are more difficult to diagnose o Due to associated morbidity of cerebral insults (e.g. intraventricular haemorrhage)
Incidence	• Generally: o Term babies: 1–5/1000 live births[13,15,17] o Preterm babies: 10–15/1000[15] • Birth weight[18]: o 1500–2500: 4.4/1000 live births o Less than 1500 grams: 55–130/1000 live births o Less than 1000 grams: up to 64/1000 live births
Clinical standards	• Use dedicated seizure form to document seizure o Refer to Table 10.History and examination • Refer to Queensland Clinical Guideline *Standard care*[19] for care considered 'usual' or 'standard' o Includes for example: privacy, consent, decision making, sensitive communication, medication administration, staff education and support, culturally appropriate care
Referral	• Consider: o Early discussion with neonatologist by contacting Retrieval Services Queensland (RSQ) regarding assessment, diagnosis and potential for transfer to a higher level nursery o Video recording of suspected seizure activity for subsequent telehealth consultation • Refer to Queensland Clinical Guideline *Neonatal stabilisation for retrieval*[20]

2 Aetiology

Seizures occur when excessive and synchronised depolarisation occurs in a large group of neurons.[21] Most neonatal seizures are acute provoked and occur in the context of a diagnosable underlying condition.[22] The underlying condition reflects different pre-, peri-, or postnatal disorders of the central nervous system (CNS). Infants are vulnerable due to the relative excitability of the developing neonatal brain[14] and high risk for brain injury during the neonatal period.
Neonatal seizures may evolve in frequency and type over time. The peak incidence occurs between 12 and 24 hours of age, but the time of onset is dependent on aetiology and treatment. Often the seizures (if acute provoked rather than epileptic) cease by 72 hours of age.[23]

2.1 CNS causes

Table 2. CNS causes

Cause	Consideration
Hypoxic-ischaemic[12,24]	• Most common cause of seizures in term babies[25-27] • Clinical features may differ with gestational age[28] • Usually present before 24 hours of age and with second peak during and after rewarming period[26] (if therapeutic hypothermia (TH) is used) • May be focal motor seizures[2] • Results from excessive depolarisation caused by a disruption to the adenosine triphosphate (ATP)-dependent pump[26] • Refer to Queensland Clinical Guideline *Hypoxic ischaemic encephalopathy*[29]
Intracranial haemorrhage[12,22,30]	• Common cause of neonatal seizures[26] • Intraventricular haemorrhage (IVH) (more common in preterm babies especially born before 34 weeks gestation[31,32]) • Includes birth related head trauma (less common)[2] • Subarachnoid and subdural haemorrhages (more commonly recognised in term babies)[26]
Infection of CNS[22]	• Acute infection requires urgent investigation and consideration of treatment pending results[26,33,34] • Congenital infections may also require urgent investigation and treatment if suspected active infection • Consider virus (HSV), cytomegalovirus (CMV), toxoplasmosis, syphilis (*Treponema pallidum*), varicella zoster, [35] • Urgently investigate for meningitis and encephalitis: o Bacterial pathogens:[12,14,36] consider *Streptococcus agalactiae* (Group B Streptococcus) and *Listeria monocytogenes,* and *Escherichia coli* and other gram negative bacilli o Viruses[12,14] (e.g. HSV, enterovirus, CMV) o Parasites such as *Toxoplasma gondii* o Septicaemia without meningitis may also result in seizures[36]
Other cerebrovascular[22]	• Congenital vascular anomalies[26] • Thrombosis–associated with venous infarct[26] • Stroke[12]

2.2 Other causes

Table 3. Other causes

Cause	Consideration
Biochemical/ metabolic[22]	• Hypoglycaemia[12,24,33] • Hypocalcaemia[12,33] • Hypomagnesaemia[12,33] • Hyponatraemia[12] • Hypernatraemia[12]
Inborn errors of metabolism[22]	• Rare inborn errors of metabolism include: o Pyridoxine responsive seizures o Other vitamin dependent epileptic encephalopathies[12,37] o Urea cycle disturbances resulting in ammonia accumulation (very rare)[12] • Prepartum–may have maternal report of abnormal intrauterine movements (fluttering or hiccoughs)[38-40] • Myoclonic semiology–multifocal and generalised myoclonic jerks often intermixed with tonic signs, abnormal eye movement, grimacing or irritability • Can have progressive clinical and EEG worsening–asymptomatic after birth and then clinical deterioration[26] (depending on the disorder) with time, fasting, or after baby starts feeding[37] • Time of onset is disorder dependent: o Disorders resulting in key metabolite deficiency may present very early (e.g. pyridoxine dependent seizures) o Disorders resulting in accumulation of a toxic product may present late o May vary with severity and timing (e.g. hypoxia, infection) • Imaging can be normal or show prominent brain atrophy, apparent hypoxic-ischaemia injury without history of insult, or diffuse cerebral oedema • Refractory to conventional treatment • Seizure activity may be accompanied by: o Metabolic acidosis, electrolyte disturbance, abdominal distension, feed intolerance, hypoglycaemia
Developmental/ congenital	• Abnormality of brain development[12] o Includes schizencephaly, lissencephaly, holoprosencephaly and hydranencephaly
Genetic epileptic syndromes	• Present as seizures that have no identified precipitant and are recurrent • May be self-limited or persistent with a range of neurodevelopmental outcomes • Classified as: o Self-limited epilepsy syndromes o Developmental and epileptic syndromes • Refer to Appendix C: Genetic epilepsy syndromes presenting in neonates or infants
Other	• Drug withdrawal or intoxication[41] • Refer to Queensland Clinical Guidelines *Perinatal substance use—neonatal*[42] and *Perinatal substance use—maternal*[43]

3 Seizure description

Clinically a seizure is a paroxysmal alteration in neurological function.[12,33] Seizures can have clinically evident manifestations that can be motor, autonomic or dissociative (that is affecting state of consciousness) or combinations of these.[44] Underlying the clinical manifestation is an electrographic seizure.[13] Normal paroxysmal neonatal behaviours and movements, (e.g. myoclonus during sleep, nonconjugate eye movements and sucking without associated eye abnormalities) can be difficult to distinguish from seizures.[13].Video recording the seizure event can assist review by other clinicians. Refer to Appendix A: Abnormal movements.

3.1 Classification

Neonatal seizures may be classified as either clinical (clinical signs with EEG abnormalities) or electrographical (only evident on EEG) or described by their mode of onset or their predominant clinical feature.

Table 4. Classification

Aspect	Consideration
Neonatal seizures	• Mostly are all focal at onset[2]as bilateral brain networks are not sufficiently developed for generalised onset seizures to occur[2] • Sometimes in rare conditions (e.g. inborn errors of metabolism) seizures may rapidly engage bilaterally distributed networks and be generalised[2] • Described according to predominant clinical feature–motor, non-motor, sequential[2] • Motor–automatisms, clonic, epileptic spasms, myoclonic and tonic[2,13] • Non-motor–autonomic and behavioural arrest[2,13] • Sequential–seizure type events with a sequence of clinical signs and EEG changes evident at different times[2] • Unclassified–inadequate or unusual clinical features not able to classify[2]
Epilepsy[2,45]	• At least two unprovoked seizures more than 24 hours apart • One unprovoked seizure and probability of further seizures after two unprovoked seizures • Diagnosis of epilepsy syndrome

3.2 Clinical presentation

3.2.1 Motor presentation

Table 5 Seizure description–motor presentation

Motor presentation		
Type	**Description**	**Comment**
Automatism[2,13,34]	• Co-ordinated motor activity usually when there is impaired cognition (e.g. HIE, preterm)–typically oral • Ocular–tonic horizontal eye deviation, or sustained eye opening with ocular fixation or cycle fluttering • Oral-facial-lingual movements–chewing movements, tongue thrusting, lip smacking • Limb movements–cycling, paddling, boxing jabs	• More common in term babies but also identified with preterm babies • Apnoea • A rare manifestation of seizures, usually without accompanying bradycardia (unless prolonged hypoxaemia) • Require EEG confirmation • May be unilateral, bilateral symmetrical, bilateral asymmetrical
Clonic[2,13,34]	• Recurrent rhythmic movements of same muscle group–jerking o Usually slow, at a rate of one to three per second o Fast contraction phase followed by slow relaxation of muscle • May involve face, arms, legs or trunk	• More reliably diagnosed by clinical observation • More likely in babies who have: o Stroke o HIE • Primarily occur in term babies • May be: o Focal, multifocal or bilateral o Symmetric or asymmetrical
Myoclonic[2,13,34]	• Non-rhythmical, random, sudden brief (lasting less than 100 millisecond) involuntary single or multiple contractions of muscles or muscle groups–typically not repetitive or may recur at a slow rate • Tendency to affect flexor muscles or muscle groups–variable topography (axial, proximal limb, distal) • More likely to have EEG changes o Include burst suppression, focal sharp waves and hypsarrhythmia	• More likely in babies who: o Are preterm o Have inborn error of metabolism • Difficult to differentiate from non-epileptic myoclonus without EEG • Occur rarely but carry worst prognosis • May be: o Focal, multifocal o Bilateral asymmetrical o Bilateral symmetrical
Tonic[2,13,34]	• Sustained increase in muscle contraction–lasts a few seconds to minutes • Generalised o Tonic extension (resemble decerebrate posturing) or o Tonic flexion of arms and extension of legs (mimics decorticate posturing) o May involve one extremity or whole body axial musculature in a opisthotonic fashion • Focal involves one extremity and especially associated with eye deviation	• Rare–developmental and epileptic encephalopathy • More common in preterm babies who have poorer prognosis o Presumed pathophysiology is non-epileptic • May be: o Focal, unilateral o Bilateral asymmetrical, bilateral symmetrical

3.2.2 Non-motor and other presentation

Table 6. Seizure description–non-motor and other presentation

Type	Description	Comment
Epileptic spasm[2]	• Flexion of proximal and truncal muscles • More sustained than myoclonic movement, not as sustained as tonic seizure • May have grimacing, head nodding, subtle eye movements	• Motor presentation • Brief and rare in newborn babies • Seen in inborn errors of metabolism
Non-motor presentation		
Autonomic[2]	• Distinct alteration of autonomic nervous system function (cardiopulmonary, pupillary, gastrointestinal, sudomotor, vasomotor, thermoregulatory) • May present as apnoea	• Requires EEG to confirm • Rare in isolation • Seen in IVH, occipital or temporal lobe lesions
Behavioural arrest[2]	• Pause in activities/immobilisation	• Requires EEG to confirm
Sequential presentation		
Sequential seizure[2]	• Several seizure manifestations occurring in sequence (not necessarily simultaneously) in a given seizure event • Sequence of signs, symptoms and EEG changes o May manifest as tonic, clonic, automatisms, and autonomic features (including apnoea) o Show varying lateralisation during single seizure	• Often seen in genetic epilepsies
Unclassified		
Unclassified seizure type[2]	• Inadequate information or unusual clinical features • Unable to classify	• Diagnosed from EEG/aEEG

3.3 Non seizure activity in babies

Table 7. Non-seizure activity

Aspect	Consideration
Jitteriness[2,46]	• Generalised, short duration very rapid tremulous movements • Recurrent and symmetrical tremulousness of all limbs or just one limb • Does not affect the face and not associated with eye deviation or autonomic change • Commonly seen in many of the same conditions that are associated with neonatal seizures and more commonly are associated with neuronal hyperactivity, (e.g. HIE, drug withdrawal from maternal drug ingestion, hypocalcaemia, and hypoglycaemia) • Reducible by tactile stimuli, gentle restraint (by holding the baby) to distinguish from seizure activity o Also lacks associated features, (e.g. tachycardia or apnoea)
Excessive startles[46-48]	• Markedly excessive startles relative to the stimulation, (e.g. auditory, touch and tonic stiffening) • Can be a sign of an encephalopathy and also seen in hyperekplexia • May be secondary to drug withdrawal (e.g. neonatal abstinence syndrome)
Benign neonatal sleep myoclonus[46-51]	• Benign condition in which the infant has myoclonic jerks during sleep • Involves one or more limbs–more commonly observed in arms • Limb movements in slow wave sleep often just after falling asleep or waking up, and can occur in rapid succession • Focal or bilateral fast rhythmic myoclonic jerks • Can be quite dramatic–whole body may twitch or jerk o Ceases immediately when the baby awakens • May worsen if baby is held
Tremor[47,51]	• Involuntary generalised movement • Rhythmical oscillating around a fixed axis • If pathological related to underlying condition, (e.g. HIE, intracranial haemorrhage, hypoglycaemia, sepsis, drug withdrawal)
Clonus[47,52,53]	• Rhythmical oscillating stretch reflex–involuntary muscle contractions and relaxation in muscle around a joint • Can be stopped by change of position of joint • Can be provoked by quick movements of joint (e.g. ankle dorsiflexion) • Repetitive muscle contraction can be normal • If abnormal amount of clonus, suspect upper motor neuron lesion
Hyperekplexia[50,51]	• Rare genetic disease also known as startle disease • General stiffness while awake, nocturnal myoclonus and an exaggerated startle reflex • Hypertonia or tonic spasms occur on awakening or from auditory or tactile stimuli • If severe may interfere with breathing

3.3.1 Jitteriness versus seizures

Table 8. Jitteriness versus seizures

Clinical feature	Jitteriness	Seizure
Abnormal gaze or eye movement[48]	No	Yes
Predominant movement[48]	Tremor, rapid, oscillatory	Repetitive clonic, jerking, tonic
Movements cease with passive flexion[48]	Yes	No
Stimulus provoked movements[48]	Yes	May have, if hyperirritability (e.g. HIE, encephalopathy)
Conscious state/ autonomic change[48]	Awake or asleep/no change	Altered

4 Diagnosis and management

Seizures can be difficult to diagnose. The aetiologies of neonatal seizures are broad and includes encephalopathic, structural, metabolic, infectious and genetic causes.[33] Recurrent and prolonged seizures are harmful to the developing brain, and require rapid recognition and assessment[12,15] to identify and treat underlying causes, prevent further brain injury and stop seizure activity.

Abnormal movements in the newborn baby may either be seizure activity (with seizures shown on an EEG), or abnormal movements due to another reason.[54] However, electrographical seizures may not be associated with abnormal movements or other clinical correlates.[14,32] Approximately one third of neonatal seizures have a clinical correlate with simultaneous video EEG recordings. The likelihood of electroclinical dissociation is increased with prematurity, prior anti-seizure medication and in the setting of TH.

4.1 Initial management

Table 9. Initial assessment and management

Aspect	Consideration
Resuscitation	• Establish adequate airway, ventilation and perfusion[14,55-57] o Minimise additional potential contributory factors such as hypoxaemia and hyper- or hypocapnia • Commence cardio-respiratory, oxygen saturation and blood pressure monitoring • Obtain umbilical venous (UV) or intravenous (IV) access • Refer to Queensland Clinical Guideline *Neonatal resuscitation*[58]
Assessment/ examination	• Undertake comprehensive history and assessment of baby: o If possible, perform before any ASM or sedating medication is administered o Refer to Table 10. and Table 11. Initial investigations
Treat underlying causes	• HIE[14]–refer to Queensland Clinical Guideline *Hypoxic ischaemic encephalopathy*[29] • Biochemical causes (e.g. hypoglycaemia[14]) o Refer to Queensland Clinical Guideline *Newborn hypoglycaemia*[59] • If suspected bacterial infection manage according to local protocols or with empirical antibiotic therapy o Commence: ▪ Benzyl penicillin IV **OR** ▪ Amoxicillin/ampicillin IV **AND** ▪ If bacterial meningitis is suspected, **ALSO** commence cefotaxime IV o Refer to *NeoMedQ Benzylpenicillin*[60], *Ampicillin*[59], *Amoxicillin*[60] and *Cefotaxime*[61] • If suspected herpes simplex virus (HSV) infection commence acyclovir IV until cerebrospinal fluid (CSF) polymerase chain reaction (PCR) for HSV is known to be negative o Refer to *NeoMedQ Aciclovir*[62] • Treat other common biochemical derangements[14] such as: o Hypocalcaemia–with 10% calcium gluconate IV ▪ Refer to *NeoMedQ Calcium gluconate 10%*[59] o Hypomagnesaemia–with 50% magnesium sulphate ▪ Refer to *NeoMedQ Magnesium sulphate*[59] • If neonatal abstinence syndrome is suspected manage with an appropriate replacement or sedative medication o Refer to Queensland Clinical Guidelines *Perinatal substance use–neonatal*[42] and *Perinatal substance use–maternal*[43]

4.2 Assessment of baby

Table 10.History and examination

Aspect	Consideration
History	• Maternal antenatal history including[12]: o Previous miscarriages o Gestational diabetes (causing neonatal hypoglycaemia) o Infections and any treatment received (including sexually transmitted diseases) particularly HSV, syphilis, CMV and toxoplasmosis o Use of prescription and other substances (e.g. opioids, alcohol, serotonin-reuptake inhibitors (SSRI), serotonin-nonreuptake inhibitors (SNRI), benzodiazepines, barbiturates, amphetamines)[41] [refer to Queensland Clinical Guidelines *Perinatal substance use-maternal*[43] and *Perinatal substance use-neonatal*[42]] o Clotting or bleeding tendencies o Pre-eclampsia o Hiccoughing or fluttering in-utero as a clue to seizure activity[38,40] • Family history[12] of epilepsy especially and consanguinity[33] • Perinatal history including type of birth and resuscitation and any: o Fetal compromise as indicated by cardiotocograph (CTG) or cord blood gases o Birth trauma o Perinatal asphyxia
Examination	• Physical examination[12,33] : o Congenital anomalies o Head circumference as microcephaly or macrocephaly may be indicative of underlying brain malformation o Facial capillary malformation (e.g. large birthmark) o Somatic abnormalities o Facial dysmorphology o Refer to Queensland Clinical Guideline *Newborn baby assessment (routine)*[63] • Abnormal neurological examination (e.g. abnormal mental status, level of alertness, reflexes, spontaneous movement or tone)[12,33,48] • Refer to Appendix B: Abnormal neurological examination of term/near term baby • Signs of sepsis (e.g. systemic signs, lethargy, bulging fontanelle caused by meningitis, or rash or lesions suggestive of infection)[33] o Refer to Queensland Clinical Guideline *Early onset Group B streptococcal disease*[64]
Observations	• Monitor and record vital signs[12] including heart rate, respiratory rate and effort, oxygen saturations, temperature, colour, blood glucose level (BGL), tone, blood pressure as indicated (e.g. if phenytoin administered) o Refer to Queensland Clinical Guideline: *Hypoglycaemia-newborn*[59] • Consider time of presentation of seizures[32] • Observe and record seizure activity[12] o Date, time and duration of any event o Whether seizures are stereotypical with clear onset and offset o Type of seizure activity including location o Abnormal eye movements o Progression of events o Autonomic changes (e.g. apnoea, hypotension, hypertension) o Any provoking stimuli (e.g. handling, noise) o Whether activity can be stopped or modified with posture or restraint o EEG correlate if concurrent monitoring in place • Video recording of seizure event(s) for later diagnostic confirmation • Document response to medications administered

4.3 Investigations

The choice of investigations is dependent on the individual baby and circumstances including the likely cause of the seizures. Consider the maternal history, and the baby's history including presentation and type of seizures, and response to treatment. Initial investigations are undertaken when a baby presents with neonatal seizures. Further investigations are stratified according to possible cause. If the baby's presentation and family history are consistent with self-limited familial neonatal-infantile epilepsy, manage as guided by paediatric neurologist.

Table 11. Initial investigations

Aspect	Consideration
Blood[12,33,34,65]	• BGL • Urea, electrolytes including calcium and magnesium • Full blood count • Liver function tests (LFTs) and total bilirubin (TBR) • Blood cultures • Blood gas for pH and lactate • Newborn bloodspot screening test (NBST) • Toxoplasmosis, other (e.g. syphilis), rubella, cytomegalovirus, herpes simplex (TORCH)
CSF[12,33,65]	• If infection suspected: o Microscopy and bacterial culture o PCR (bacterial and viral), (e.g. HSV, enterovirus) o Glucose o Protein o Blood o Colour
Urine[54]	• If infection suspected–microscopy and culture
Imaging[32-34,46,54,66]	• Ultrasound scan (USS) for detection of intra-ventricular and parenchymal haemorrhage • Magnetic resonance imaging (MRI): o Optimal neuroimaging modality o Preferable to computed tomography or USS–availability and clinical condition of baby may indicate USS first (e.g. baby is receiving TH and MRI may be difficult to accomplish before 4–10 days of age) o Can be useful for identifying structural causes of seizures o Greater sensitivity than CT or USS in identifying brain malformations, intracranial haemorrhage and ischaemic damage o May show cerebral dysgenesis, lissencephaly and other neuronal migration disorders that are not visible on CT or USS o Timing is dependent on suspected cause of seizures (e.g. as soon as possible for serious intracranial haemorrhage and day 4–10 for baby with HIE o Refer to Queensland Clinical Guideline *Hypoxic ischaemic encephalopathy (HIE)*[29] o Indicated if aetiology is not identified and seizures resistant to usual ASMs
EEG	• EEG (12 lead) is the gold standard for seizure investigation[2,14,22] o Well-established, non-invasive real time monitor[67] o Provides accurate diagnosis of seizures o Identifies area of seizure origin in brain o Determines frequency, duration and propagation[68] o Has prognostic value as identifies babies at risk of abnormal outcome • Access and availability in Queensland may be limited to higher level services • Refer to Appendix D: EEG and subsequent investigations
Subsequent investigations	• Usually undertaken with specialist guidance and/or at a level 5/6 neonatal/paediatric facility • Are guided by the baby's clinical features and presentation of the seizures • Discuss any subsequent investigations with a neonatologist, by contacting RSQ to facilitate potential transfer or retrieval of baby. • Refer to Appendix D: EEG and subsequent investigations

4.4 Management and treatment

The principles for acute provoked neonatal seizure management includes[15,55,69]:

- Rapid and accurate identification of seizures[15]
  - Under treatment may lead to additional seizures, and add to existing brain injury and alter seizure thresholds in the brain[33,69]
  - Overtreatment results in exposure to neurotoxic medicines and prolonged intensive care with associated risks (e.g. separation from parents, complications from procedures, sedation)[33,69]
- ASM management:
  - Appropriate for seizure type[15] and aetiology
  - Early discontinuation once seizures have ceased[23]
- Prevention of secondary problems by maintaining normal physiological temperature, blood glucose, oxygenation, ventilation and blood pressure[55]

4.5 Continuing care

Table 12. Continuing care

Aspect	Consideration
Medications	• Refer to Section 5 Medications
Model of care	• Provide family centred care • Establish early and ongoing communication with parents o Repeat information as often as required • Involve support services (e.g. Indigenous health worker (as indicated), social worker, psychologist) to support parents and family o Long term sequelae from the underlying cause may have profound impact on quality of life for the baby and family
Parents	• Discuss baby's condition, and options for care and treatment with parents • Discuss prognosis with honesty and sensitivity o If relevant, include specialist advice about prognosis • Document discussions in healthcare record • Refer to 7.1 Discharge planning
Documentation	• Video (if available) abnormal movements simultaneously with recording of cardiorespiratory monitoring
Environment[70]	• Provide developmentally supportive care • Provide comfortable and supportive positioning • Reduce and manage pain and stress during procedures (e.g. analgesia, containment) • If seizures provoked by stimuli, reduce noise, light, invasive treatment and care activities

5 Medications

While pharmacological options for treatment of neonatal seizures have increased there is limited evidence regarding the optimal pharmacological treatment strategy.[15,34] Consider whether seizures are acute provoked (where underlying cause predicts whether seizures will remit, continue or worsen), or epileptic (with recurring risk), or have specific aetiology (e.g. KCNQ2 responds only to sodium-channel blockers but may have a self-limited seizure period). Phenobarbital is recommended as the first line medication. Due to insufficient evidence, there is variation in practice for the preferred medication(s) for second line treatment except for in certain specific aetiologies. If seizures appear refractory then confirmation of their epileptic nature is essential.

Table 13. Principles

Aspect	Consideration
Context	• Consider benefits and risks of initiating antiseizure medications including o Potential efficacy o Potential toxicity and side effects o Anticipated rapidity of response[71] • Evidence based recommendations from randomised controlled trials (RCT) is lacking regarding the relative benefits versus the risk of harm from ASMs used to treat neonatal seizures[15,34,72] • TH and the re-warming phase of HIE management alters ASM pharmokinetics[73] o Refer to Queensland Clinical Guideline *Hypoxic ischaemic encephalopathy (HIE)*[29]
Expert recommendation	• Treat clinical seizures and electrographical seizures if recurrent, frequent or prolonged • Benefit from treating brief infrequent electrographical seizures with no clinical correlate is unclear[72] • Phenobarbital is the preferred first line medication[74] o Refer to Table 14. Phenobarbital
Principles	• Treating specific underlying causes of seizures (e.g. metabolic) is critical to prevent clinical deterioration, further brain damage and poor long term neuro-developmental outcomes[75] • Commence treatment when: o Clinically apparent seizure lasts more than five minutes o Repeated seizure events occur
Maintenance and duration of treatment	• Optimal duration of treatment with ASM is unknown • Consider contact with Retrieval Services Queensland (RSQ) for discussion with paediatric neurologist before introducing second line ASM • Duration of treatment considerations include[23]: o Neurological status o EEG o Underlying aetiology[76] • Unless a persistent epileptic condition is suspected cease ASM when o Free of seizures for 72 hours o Neurological examination is normal[66,76] o Only one ASM medication required to control seizure activity–if two or more agents used consider the elimination half life of each medication in evaluating the seizure free interval • Targeted maintenance treatment for genetic and metabolic disorders may be lifelong[66,76], but is dependent on underlying aetiology and diagnosis • Treatment usually continued if progression to epilepsy is considered highly likely (e.g. structural brain malformations and neonatal epilepsy syndromes)

5.1 Anti-seizure medications

5.1.1 Phenobarbital

Table 14. Phenobarbital

Phenobarbital	Consideration
Context	• Preferred first line agent[24,77] • More effective in achieving initial control of neonatal seizures than levetiracetam[74,78] • A second line drug may be required • Consider ceasing phenobarbital if: o Baby is stabilised on second line ASM or • Baby is seizure free and second line ASM not required If relevant, refer to Queensland Clinical Guideline *Hypoxic ischaemic encephalopathy*[29]
***Phenobarbital administration**	• Loading dose required to achieve therapeutic levels (due to long half life) o Additional loading does may be given for refractory seizures • Maintenance dose o Commence only if seizures continue after the loading doses • Refer to *NeoMedQ Phenobarbital*[79] • If used for acute provoked seizures, discontinue after three days from last seizure, if: o Only medication used o Normal neurological examination, EEG and MRI[80] • If used for epilepsy, follow advice of paediatric neurologist

5.1.2 Other ASM

Consider second line ASM in discussion with paediatric neurologist.

Table 15. Other ASM

ASM	Consideration
***Levetiracetam** [74,77,81]	• Consider for seizures refractory to phenobarbital or where longer term medication is needed • Not as effective as phenobarbital as a first line medication in treating neonatal seizures • Associated with lower risk of adverse events o Data regarding adverse effects in neonates is limited to case reports and abstracts • Loading dose not required but may be given if urgent seizure control required • Refer to *NeoMedQ Levetiracetum*[79]
***Midazolam**[82]	• Consider for seizures refractory to phenobarbital • Refer to *NeoMedQ Midazolam*[83]
^*Topiramate[54]	• Consider for seizures refractory to phenobarbital • Refer to *NeoMedQ Topiramate*[84]
***Clonazepam**[84,85]	• Consider for seizures refractory to phenobarbital • Drug of choice for hyperekplexia • Refer to *NeoMedQ Clonazepam*[86]
***Lidocaine**[54]	• Consider for severe recurrent or prolonged seizures not responding to earlier line treatment • Refer to *NeoMedQ Lidocaine*[86]
***Phenytoin**	• Consider for seizures refractory to phenobarbital with neonatologist/paediatric neurologist advice (contact via RSQ) • Effective for treating some genetic epilepsies • Refer to *NeoMedQ Phenytoin*[81]

*Refer to an Australian pharmacopoeia for complete drug information
^Not on List of Approved Medicines (LAM) in Queensland

5.2 Pyridoxine (vitamin B6) deficiency

Table 16.Pyridoxine

Pyridoxine (vitamin B6)	
Comment[85,87-90]	• Vitamin B6 is a required enzyme in the biosynthesis of dopamine and serotonin • Used for diagnosis and treatment of pyridoxine dependent seizures o Babies with pyridoxamine5'-phosphate oxidase (PNPO) deficiency may also partially respond
Dose and administration	• Manage and administer with expert advice o Contact RSQ for discussion with neonatologist, paediatric neurologist and metabolic specialist • Cardio-respiratory monitoring is required • Ventilator support may be necessary • Best administered while EEG monitoring, but absence of EEG should not delay administration • If responsive, maintenance therapy required for life[80] • Refer to *NeoMedQ Pyridoxine*[91]

*Refer to an Australian pharmacopoeia for complete drug information

6 Ongoing care

6.1 Discharge planning

Document discussions with parents, including emergency seizure management at home, prognosis, follow-up and parental decisions to facilitate consistency of information. Provide the parents with appropriate discharge information and documentation including:

- A seizure emergency management plan[23] including contacting Queensland Ambulance Service (QAS)
- Prescriptions for medications required after discharge (as per local arrangements)
- Information and education about medications including potential duration of treatment
- A copy of the discharge summary including the type of seizures and medications
- Contact details of available support services in the local area or online[92]
- Copies of referrals to other services
- Confirmed, timely follow up appointments if baby is having or is at risk of having ongoing seizures or seizure emergency (e.g. paediatric neurologist), or has underlying genetic aetiology for seizures (e.g. Genetic Health Queensland)

6.2 Follow up

Table 17. Follow up

Aspect	Consideration
Context[26,67]	• Babies who are preterm or have other neurodevelopmentally vulnerable causes of seizures (e.g. metabolic disorders, CNS malformations and HIE) are at greater risk of poor outcomes • Follow up by multidisciplinary team to assess developmental outcomes o Dependent on cause of seizures and response to treatment
Follow up care	• Facilitate follow up with verbal and written communication, and assistance with appointments as required • Consider: o General practitioner and child health nurse o Paediatrician in local area o If baby discharged home on ASMs paediatrician, paediatric neurologist or neonatologist according to local arrangements–if available telehealth may be used
Early intervention[26,93]	• Consider multi-disciplinary team to identify any motor and cognitive deficits, and timely neuro-developmental early invention using simple tools such as the General Movements Assessment (GMA), parent screening and use of Ages and Stages questionnaire o Abnormal fidgety GMA at three months of age is predictive for cerebral palsy and other neurodevelopmental delay o GMA and has been validated in term (with HIE) and preterm babies as a predictor of cerebral palsy o GMA requires 15 minutes of observation of the baby by video and analysis by a trained observer in the fidgety movements stage (three months corrected age)

6.3 Prognosis and outcomes

Table 18. Prognosis

Aspect	Consideration
Prognosis [21,28,32,41,94]	• Neonatal seizures especially if high seizure burden are associated with poor outcomes • The aetiology of seizures determines the outcomes and prognosis • Strongest predictors of outcome–underlying cause and background EEG activity • Seizures due to some causes may have a good long term prognosis
High risk of poor outcome [25,32,41,95,96]	• HIE • Prematurity especially those with most serious life threatening illnesses o Early onset (within 48 hours of birth) o Repeated seizures of greater than or equal to one hour in duration o Recurrent seizures of greater than 48 hours • Cerebral malformations especially if extensive • CNS infection • Severe IVH or intercranial haemorrhage • Severely abnormal EEG inter-ictal activity (isoelectric pattern, paroxysmal, burst-suppression and low voltage background)-requires gestational age appropriate interpretation • Persistence of a diffuse EEG abnormality • More than one ASM to control seizures • Other factors: o Severely abnormal neurological examination o Severely abnormal neuroimaging o High seizure burden o Presence of status epilepticus
Associated with favourable outcome [18,93,97]	• Normal neurological examination • Focal clonic seizures o Transient metabolic disturbance (e.g. hypocalcaemia) o Focal lesions (e.g. brain haemorrhage or stroke) on MRI o Lesion confined to relatively circumscribed areas of the brain • Brief or rarely recurring seizures • Normal inter-ictal EEG • Normal EEG (if performed) within 24 hour of birth generally has good prognosis • Neonatal sleep myoclonus • Normal or rapid normalisation of background pattern on aEEG. • Self-limited familial neonatal epilepsy conditions • Normal MRI • Normal GMA at age 3 months
Morbidity and mortality [21,25,28,32,72,97-100]	• Risk of acute effects and long term sequelae including long term morbidity and neonatal mortality o Associated with underlying aetiology of seizures • Acute and long term adverse effects result from energy failure, excitotoxicity, neuronal death, apoptosis and status epilepticus • Complications include cognitive, motor and behavioural problems: o Neurodevelopmental disability (e.g. cerebral palsy, spasticity, learning difficulties, intellectual disability) o Cerebral atrophy o Hydrocephalus ex-vacuo o Microcephaly o Epilepsy o Feeding difficulties o Behavioural problems, (e.g. autism, attention deficit disorder) o Cognitive dysfunction, (e.g. memory deficit) • Headache

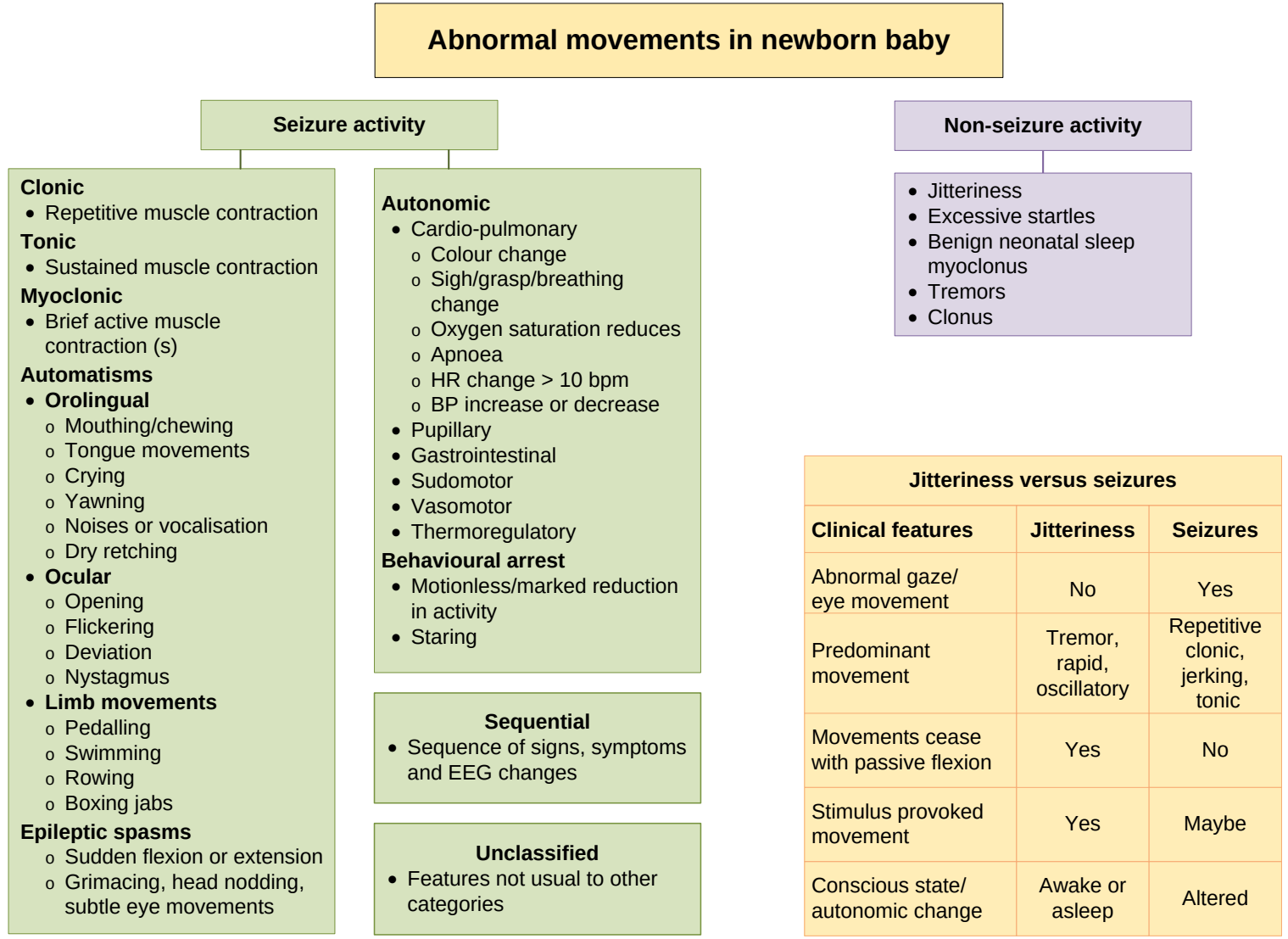
References

1. National Human Genome Research Institute. Apoptosis. [Internet]. 2021 [cited 2022 January 27]. Available from: https://www.genome.gov/genetics-glossary.
2. Pressler RM, Cilio MR, Mizrahi EM, Moshé SL, Nunes ML, Plouin P, et al. The ILAE classification of seizures and the epilepsies: modification for seizures in the neonate. Position paper by the ILAE Task Force on Neonatal Seizures. Epilepsia 2021;62(3):615-28.
3. John Hopkins Medicine. Hydrocephalus. [Internet]. 2022 [cited 2022 June 2]. Available from: http://www.hopkinsmedicine.org.
4. Monteagudo A. Holoprosencephaly. American Journal Obstetrics and Gynecology 2020;223(6):B13-B6.
5. Habek D. Peripartum cephalocenthesis in a large fetal hydranencephaly. Journal Perinatal Medicine 2022;50(5):634-5.
6. Merriam-Webster Medical Dictionary. Hypsarrhythmia. [Internet]. 2022 [cited 2022 June 9]. Available from: http://c.merriam-webster.com.
7. John Hopkins Medicine. Diagnosing seizures and epilepsy. [Internet]. 2022 [cited 2022 June 9]. Available from: https://www.hopkinsmedicine.org.
8. National Institute of Neurological Disorders and Stroke. Lissencephaly. [Internet]. 2022 [cited 2022 June 9]. Available from: https://www.ninds.nih.gov.
9. MedlinePlus. Pyridoxal 5'-phosphate dependent epilepsy. [Internet]. 2008 [cited 2022 October 7]. Available from: https://medlineplus.gov.
10. Griffiths PD. Schizencephaly revisited. Neuroradiology 2018;60(9):945-60.
11. MedlinePlus. Spasticity. [Internet]. 2022 [cited 2022 June 2]. Available from: https://medlineplus.gov.
12. Krawiec C, Muzio M. Neonatal seizure. Stat Pearls. [Internet]. 2021 [cited 2022 February 11]. Available from: https://www.ncbi.nlm.nih.gov.
13. Nguyen T, Wusthoff CJ. Clinical manifestations of neonatal seizures. Pediatrics International 2021;63(6):631-5.
14. Kaminiów K, Kozak S, Paprocka J. Neonatal seizures revisited. Children (Basel) 2021;8(2):155.
15. Falsaperla R, Scalia B, Giugno A, Pavone P, Motta M, Caccamo M, et al. Treating the symptom or treating the disease in neonatal seizures: a systematic review of the literature. Italian Journal of Pediatrics. [Internet]. 2021 [cited May 14 2022]; 47(1):85- DOI:10.1186/s13052-021-01027-2.
16. Soul JS, Bergin AM, Stopp C, Hayes B, Singh A, Fortuno CR, et al. A pilot randomized, controlled, double-blind trial of bumetanide to treat neonatal seizures. Annals of Neurology 2021;89(2):327-40.
17. Nickels K. Neonatal seizures: providing care with evidence, not just experience. Epilepsy Currents 2021;21(6):427-9.
18. Uria-Avellanal C, Marlow N, Rennie J. Outcome following neonatal seizures. Seminars in Fetal & Neonatal Medicine 2013;18:224-32.
19. Queensland Clinical Guidelines. Standard care. Guideline No. MN18.50-V1-R23. [Internet]. Queensland Health. 2018. [cited 2022 June 3]. Available from: https://www.health.qld.gov.au/qcg.
20. Queensland Clinical Guidelines. Neonatal stabilisation for retrieval. Guideline No. MN18.18-V4-R23. [Internet]. Queensland Health. 2018. [cited 2022 June 3]. Available from: https://www.health.qld.gov.au/qcg.
21. Chalia M, Hartmann H, Pressler R. Practical approaches to the treatment of neonatal seizures. Current Treatment Options in Neurology 2022;24(3):111-27.
22. Santarone ME, Pietrafusa N, Fusco L. Neonatal seizures: when semiology points to etiology. Seizure 2020;80:161-5.
23. Glass HC, Shellhaas RA. Safety of early discontinuation of antiseizure medication after acute symptomatic neonatal seizures—reply. Journal of the American Medical Association Neurology 2022;70(1):91-2.
24. Ziobro JM, Eschbach K, Shellhaas RA. Novel therapeutics for neonatal seizures. Neurotherapeutics 2021;18(3):1564-81.
25. Glass HC, Shellhaas RA, Wusthoff CJ, Chang T, Abend NS, Chu CJ, et al. Contemporary profile of seizures in neonates: a prospective cohort study. Journal of Pediatrics. 2016 [cited Jul]; 174:98-103.e1. Available from: https://doi.org/10.1016/j.jpeds.2016.03.035. DOI:10.1016/j.jpeds.2016.03.035.
26. Martin M, Querubin J, Hagen E, Lim J. Evaluation of the neonate with seizures. Pediatric Annals 2020;49(7):e292-e8.
27. Ramantani G, Ikonomidou C, Walter B, Rating D, Dinger J. Levetiracetam: safety and efficacy in neonatal seizures. European Journal of Paediatric Neurology 2011;15:1-7.
28. Sheth R, Hobbs J, Mullett M. Neonatal seizures: incidence, onset, and etiology by gestational age. Journal of Perinatology 1999;19(1):40-3.
29. Queensland Clinical Guidelines. Hypoxic ischaemic encepathalopathy. Guideline No. MN21.11-10-R26. [Internet]. Queensland Health. 2021. [cited 2022 June 3]. Available from: https://www.health.qld.gov.au/qcg.
30. McKee-Garrett T. Neonatal birth injuries. [Internet]. Waltham NA: UpToDate Inc; 2021 [cited 2022 June 2]. Available from: https://www.uptodate.com.
31. De Vries LS. Germinal matrix hemorrhage and intraventricular hemorrhage (GMH-IVH) in the newborn: pathogenesis, clinical presentation, and diagnosis. [Internet]. Waltham MA: UptoDate; 2022 [cited 2022 May 29]. Available from: https://www.uptodate.com.
32. Ramantani G, Schmitt B, Plecko B, Pressler RM, Wohlrab G, Klebermass-Schrehof K, et al. Neonatal seizures—are we there yet? Neuropediatrics 2019;50(5):280-93.
33. Shellhaus R. Clinical features, evaluation, and diagnosis of neonatal seizures. [Internet]. Waltham MA: UpToDate; 2021 [cited 2021 December 8]. Available from: https://www.uptodate.com.
34. Soul JS. Acute symptomatic seizures in term neonates: etiologies and treatments. Seminars Fetal & Neonatal Medicine 2018;23(3):183-90.
35. Palasanthiran P, Starr M, Jones C, Giles M. Management of Perinatal Infections. Sydney: Australasian Society for Infectious Diseases; 2014.
36. Edwards MS, Baker CJ. Bacterial meningitis in the neonate: treatment and outcome. [Internet]. Waltham MA: UptoDate; 2020 [cited 2022 May 17]. Available from: www.uptodate.com.
37. Olson DM. Neonatal Seizures. Neoreviews. [Internet]. 2012 [cited 2022 May 24]; 13(4):e213-e23 DOI:10.1542/neo.13-4-e213.
38. Rahman S, Footitt E, Varadkar S, Clayton P. Inborn errors of metabolism causing epilepsy. Developmental Medicine & Child Neurology 2012;55:23-36.
39. Sandoval Karamian A, Wusthoff C. Current and future uses of continuous EEG in the NICU. Frontiers in Pediatrics. [Internet]. 2021 [cited 2022 May 27]; 9:768670-. Available from: https://www.frontiersin.org DOI:10.3389/fped.2021.768670.
40. Surtees R, Wolf N. Treatable neonatal epilepsy. Archive of Diseases in Childhood 2007;92:659-61.
41. Shellhaus R. Etiology and prognosis of neonatal seizures. [Internet]. Waltham MA: UpToDate Inc; 2021 [cited 2021 December 8]. Available from: http://www.uptodate.com.
42. Queensland Clinical Guidelines. Perinatal substance use: neonatal Guideline No. MN21.38-V3-R26. [Internet]. Queensland Health. 2021. [cited 2022 June 3]. Available from: https://www.health.qld.gov.au/qcg.
43. Queensland Clinical Guidelines. Perinatal substance use: maternal Guideline No. MN21.37-V2-R26. [Internet]. Queensland Health. 2021. [cited 2022 June 2]. Available from: https://www.health.qld.gov.au/qcg.

44. Fisher RS, Cross JH, French JA, Higurashi N, Hirsch E, Jansen FE, et al. Operational classification of seizure types by the International League Against Epilepsy: position paper of the ILAE commission for classification and terminology. Epilepsia 2017;58(4):522-30.
45. Fisher RS, Cross JH, D'Souza C, French JA, Haut SR, Higurashi N, et al. Instruction manual for the ILAE 2017 operational classification of seizure types. Epilepsia 2017;58(4):531-42.
46. Sivaswamy L. Approach to neonatal seizures. Clinical Pediatrics 2012;51(5):415-25.
47. Hart A, Pilling E, Alix J. Neonatal seizures—part 1: not everything that jerks, stiffens and shakes is a fit. Archive of Diseases in Childhood: Education and Practice Edition 2015;100:170-75.
48. Volpe JJ. Neurological examination: normal and abnormal features. In: Volpe JJ, Inder TE, Darras BT, de Vries LS, du Plessis AJ, Neil JJ, et al, editors. Volpe's Neurology of the Newborn (Sixth Edition). [Internet]: Elsevier; 2018. p. 191-221.e8. Available from: https://www.sciencedirect.com.
49. Losito E, Eisermann M, Vignolo P, Hovhannisyan S, Magny JF, Kaminska A. Benign neonatal sleep myoclonus evokes somatosensory responses. Journal of Clinical Neurophysiology 2017;34(6):484-91.
50. Nguyen T, Kaplan P, Wilfong A. UpToDate, editor. Nonepileptic paroxysmal disorders in infancy. [Internet]. Waltham MA 2021 [cited 2021 December 8]. Available from: https://www.uptodate.com.
51. Orivoli S, Facini C, Pisani F. Paroxysmal nonepileptic motor phenomena in newborn. Brain and Development 2015;37(9):833-9.
52. Hawes J, Bernardo S, Wilson D. The neonatal neurological examination: improving understanding and performance. Neonatal Network 2020;39:116-28.
53. Zimmerman B, Hubbard J. Clonus. Stat Pearls. [Internet]. 2021 [cited 2022 May 18]. Available from: https://www.ncbi.nlm.nih.gov.
54. Chalia M, Austin T. Practice guide to neonatal seizures. Paediatrics and Child Health 2018;28(10):488-91.
55. Glass H. Neonatal seizures: advances in mechanisms and management. Clinics in Perinatology 2014;41(1):177-90.
56. Murray D, Boylan G, Ali I, Ryan C, Murphy B, Connolly S. Defining the gap between electrographic seizure burden, clinical expression and staff recognition of neonatal seizures. Archives of Disease in Childhood - Fetal and Neonatal Edition 2008;93:F187-89.
57. Roland E, Hill A. Neurological problems of the newborn. In: Daroff R, Mazziotta J, Pomeroy S, Jankovic J, editors. Bradley's Neurology in Clinical Practice. 7th ed. London: Elsevier 2016.
58. Queensland Clinical Guidelines. Neonatal resuscitation. Guideline No. MN22.5_V6_R27. [Internet]. Queensland Health. 2022. [cited 2022 November 1]. Available from: https://www.health.qld.gov.au/qcg.
59. Queensland Clinical Guidelines. NeoMedQ Calcium guconate 10%. Guideline No. NMedQ19.008-V1-R24. [Internet]. Queensland Health. 2019. [cited 2022 June 3]. Available from: https://www.health.qld.gov.au/qcg.
60. Queensland Clinical Guidelines. NeoMedQ Benzylpenicillin. Guideline No. NMedQ20.13-V3-R25. [Internet]. Queensland Health. 2020. [cited 2022 June 2]. Available from: https://www.health.qld.gov.au/qcg.
61. Queensland Clinical Guidelines. Cefotaxime. Guideline No. nMedQ19.011-V2-R24. [Internet]. Queensland Health. 2019. [cited 2022 June 2]. Available from: https://www.health.qld.gov.au/qcg.
62. Queensland Clinical Guidelines. NeoMedQ Ampicillin. Guideline No. NMedQ19.012-V5-R24. [Internet]. Queensland Health. 2019. [cited 2022 June 3]. Available from: https://www.health.qld.gov.au/qcg.
63. Queensland Clinical Guidelines. Newborn baby assessment (routine). Guideline No. MN21.4-V6-R26. [Internet]. Queensland Health. 2022. [cited 2022 June 3]. Available from: https://www.health.qld.gov.au/qcg.
64. Queensland Clinical Guidelines. Early onset Group B Streptococcal disease. Guideline No. MN16.20-V4-R22. [Internet]. Queensland Health. 2016. [cited 2022 June 3]. Available from: https://www.health.qld.gov.au/qcg.
65. Hellström-Westas LK, Levene M. Neonatal seizures. In: Neonatology. Cham: Springer International Publishing; 2018. p. 2287-93.
66. World Health Organisation. Guidelines on neonatal seizures. [Internet]. 2011 [cited 2022 June 3]. Available from: www.who.int.
67. Kong AHT, Lai MM, Finnigan S, Ware RS, Boyd RN, Colditz PB. Background EEG features and prediction of cognitive outcomes in very preterm infants: a systematic review. Early Human Development 2018;127:74-84.
68. Ryan MA, Mathieson S, Dempsey E, Boylan G. An introduction to neonatal EEG. The Journal of Perinatal & Neonatal Nursing 2021;35(4):369-76.
69. Rennie JM, de Vries LS, Blennow M, Foran A, Shah DK, Livingstone V, et al. Characterisation of neonatal seizures and their treatment using continuous EEG monitoring: a multicentre experience. Archive of Diseases in Childhood: Fetal & Neonatal 2019;104(5):F493-F501.
70. Webster D. Neonatal brain injury In: Boxwell G, Petty J, Kaiser L, editors. Neonatal Intensive Care Nursing 3rd ed. Milton: Routledge; 2020.
71. Slaughter LA, Patel AD, Slaughter JL. Pharmacological treatment of neonatal seizures: a systematic review. Journal of Child Neurology 2013;28(3):351-64.
72. Hunt RW, Liley HG, Wagh D, Schembri R, Lee KJ, Shearman AD, et al. Effect of treatment of clinical seizures vs electrographic seizures in full-term and near-term neonates: a randomized clinical trial. Journal of American Medical Association Network Open. [Internet]. 2021 [cited 2022 May 12]; 4(12):e2139604-e. Available from: https://doi.org.
73. Donovan M, Griffin B, Kharoshankaya L, Cryan J, Boylan G. Pharmacotherapy for neonatal seizures: current knowledge and future perspectives Drugs 2016;76:647-61.
74. Sharpe C, Reiner GE, Davis SL, Nespeca M, Gold JJ, Rasmussen M, et al. Levetiracetam versus phenobarbital for neonatal seizures: a randomized controlled trial. Pediatrics 2020;145(6):e20193182.
75. Shetty J. Neonatal seizures in hypoxic–ischaemic encephalopathy – risks and benefits of anticonvulsant therapy. Developmental Medicine and Child Neurology 2015;57(Supplement 3):40-3.
76. Nagarajan L. Treatment of neonatal seizures In: Nagarajan L, editor. Neonatal Seizures: Current Treatment and Future Challenges. London: MacKeith Press; 2016.
77. Xu Z-E, Li W-B, Qiao M-Y, Cui H-T, Zhao L-Z, Chen Q-X, et al. Comparative efficacy of anti-epileptic drugs for neonatal seizures: a network meta-analysis. Pediatrics & Neonatology 2021;62(6):598-605.
78. Sharpe C, Reiner GE, Davis SL. For the neolev2 investigators. Levetiracetam versus phenobarbital for neonatal seizures: a randomized controlled trial Pediatrics 2021;147(1).
79. Queensland Clinical Guidelines. NeoMedQ Phenobarbital (phenobarbitone). Guideline No. NMedQ21.062-V1-R26. [Internet]. Queensland Health. 2021. [cited 2022 June 3]. Available from: https://www.health.qld.gov.au/qcg.
80. Therapeutic Guidelines. Neonatal seizures. [Internet]. 2021 [cited 2022 April 20]. Available from: https://tgldcdp.tg.org.au.
81. Micromedex Solutions. Levetiracetam. [Internet]. 2022 [cited 2022 June 3]. Available from: https://www.micromedexsolutions.com.
82. Micromedex Solutions. Midazolam. [Internet]. 2022 [cited 2022 June 3]. Available from: https://www.micromedexsolutions.com.
83. Queensland Clinical Guidelines. NeoMedQ Midazolam. Guideline No. NMedQ20.033-V1-R25. [Internet]. Queensland Health. 2020. [cited 2022 June 3]. Available from: https://www.health.qld.gov.au/qcg.

84. Micromedex Solutions. Clonazepam. [Internet]. 2022 [cited 2022 June 3]. Available from: https://www.micromedexsolutions.com.
85. Ainsworth SB. Neonatal formulary 7. Drug use in pregnancy and the first year of life. Wiley Blackwell BMJI Books 2015.
86. Queensland Clinical Guidelines. NeoMedQ Clonazepam. Guideline No. NMQ 22.085-V1-R27. [Internet]. Queensland Health. 2022. [cited 2022 September 26]. Available from: https://www.health.qld.gov.au/qcg.
87. Ghatge MS, Al Mughram M, Omar AM, Safo MK. Inborn errors in the vitamin B6 salvage enzymes associated with neonatal epileptic encephalopathy and other pathologies. Biochimie 2021;183:18-29.
88. Shellhaas R, Glass H, Chang T. Neonatal seizures. In: Swaiman K, Ashwa lS, Ferriero D, Schor N, Pearl P, Shevell M, editors. Swaiman's Pediatric Neurology 6th edition Edinburgh: Elsevier; 2018. p. 129-37.
89. Shellhaas R, Chang T, Wusthoff C, Soul J, Massey S, Chu C, et al. Treatment duration after acute symptomatic seizures in neonates: a multicenter cohort study. Journal of Pediatrics. 2016; 181:298-301.e1. Available from: https://www.jpeds.com DOI:10.1016/j.jpeds.2016.10.039.
90. Micromedex Solutions. Pyridoxine. [Internet]. 2022 [cited 2022 June 3]. Available from: https://www.micromedexsolutions.com.
91. Queensland Clinical Guidelines. NeoMedQ Pyridoxine. Guideline No. NMedQ22.089-V1-R27. [Internet]. Queensland Health. 2022. [cited 2022 September 26]. Available from: https://www.health.qld.gov.au/qcg.
92. Lemmon ME, Glass HC, Shellhaas RA, Barks MC, Bansal S, Annis D, et al. Family-centered care for children and families impacted by neonatal seizures: advice from parents. Pediatric Neurology 2021;124:26-32.
93. Seesahai J, Luther M, Church PT, Maddalena P, Asztalos E, Rotter T, et al. The assessment of general movements in term and late-preterm infants diagnosed with neonatal encephalopathy, as a predictive tool of cerebral palsy by 2 years of age—a scoping review. Systematic Reviews 2021;10(1):1-226.
94. Glass HC, Shellhaas RA, Tsuchida TN, Chang T, Wusthoff CJ, Chu CJ, et al. Seizures in preterm neonates: a multicenter observational cohort study. Pediatric Neurology 2017;72:19-24.
95. Ghosh S, Cabassa Miskimen AC, Brady J, Robinson MA, Zou B, Weiss M, et al. Neurodevelopmental outcomes at 9–14 months gestational age after treatment of neonatal seizures due to brain injury. Child's Nervous System 2019;35(9):1571-8.
96. Kharoshankaya L, Stevenson NJ, Livingstone V, Murray DM, Murphy BP, Ahearne CE, et al. Seizure burden and neurodevelopmental outcome in neonates with hypoxic–ischemic encephalopathy. Developmental Medicine and Child Neurology 2016;58(12):1242-8.
97. Glass HC, Li Y, Gardner M, Barkovich AJ, Novak I, McCulloch CE, et al. Early identification of cerebral palsy using neonatal MRI and general movements assessment in a cohort of high-risk term neonates. Pediatric Neurology 2021;118:20-5.
98. Lombroso C. Neonatal seizures: gaps between the laboratory and the clinic. Epilepsia 2007;48(Supplement 2):83-106.
99. Oh A, Thurman DJ, Kim H. Independent role of neonatal seizures in subsequent neurological outcomes: a population-based study. Developmental Medicine and Child Neurology 2019;61(6):661-6.
100. Padiyar S, Nusairat L, Kadri A, Abu-Shaweesh J, Aly H. Neonatal seizures in the U.S. national inpatient population: prevalence and outcomes. Pediatrics and Neonatology 2020;61(3):300-5.

Appendix A: Abnormal movements



Jitteriness versus seizures

Clinical features	Jitteriness	Seizures
Abnormal gaze/ eye movement	No	Yes
Predominant movement	Tremor, rapid, oscillatory	Repetitive clonic, jerking, tonic
Movements cease with passive flexion	Yes	No
Stimulus provoked movement	Yes	Maybe
Conscious state/ autonomic change	Awake or asleep	Altered

Abbreviations: **BP:** blood pressure, **EEG:** electroencephalogram, **HR** heart rate, **>** greater than

Queensland Clinical Guidelines F22.23-3-V2-R27 Neonatal seizures: Abnormal movements

Appendix B: Abnormal neurological examination of term/near term baby

Aspect/test	Consideration	Abnormal
History	• Family, maternal, antenatal, intrapartum and birth • Gestational age • Apgar score and resuscitation • Morphology scan or fetal MRI findings	• Family history of: o Genetic disorders o Birth defects o Seizures, developmental delay, thromboembolic or coagulation disorders o Stillbirths or early unexpected deaths • Difficult birth
General	• Normal resting posture–moderate flexion of four limbs, held off bed	• Constant tight flexion • Full extension, flaccid or forced
	• Observations (temperature, heart rate, respirations, oxygen saturation, colour) and growth	• Apnoea • Small for gestational age or fetal growth restriction
	• General appearance–normal features, facial symmetry, eye movement symmetry	• Dysmorphic features, facial asymmetry, eye movement asymmetry, facial palsy
	• Examination of the skin (presence of lesions or rashes), and spine	• Hair tufts, tracts along spine • Pale pink macular lesion (portwine stain); petechiae
Head shape, size and facial features	• Fontanelles, sutures • Facial features	• Caput succedaneum • Cephalhaematoma • Subgaleal haemorrhage • Bulging fontanelles • Widely separated, open sutures • Abnormal head shape with rigid sutures • Facial dysmorphism • Facial palsy • Abnormal eye movements • Sunset sign
	• Head circumference (10th–90th percentile)	• Microcephaly • Macrocephaly
Level of alertness	• Normal response to arousal	• Irritability, lethargy
Behavioural state (state of consciousness)	• Light sleep, drowsiness, quiet alert, active alert, crying	• Stupor, coma • Irritable • Lethargic
Cry	• Loud, strong	• High pitched • Weak or monotonous
Muscle tone and development	• Normal	• Hypotonic • Hypertonic

Aspect/test	Consideration	Abnormal
Reflexes–Moro, suck, grasp, gag	• Present	• Absent or asymmetric; pooling of secretions • Moro: o No response or opening of hands only o No abduction or adduction; only forward extension of arms from the shoulders; marked adduction only
Movements	• Tremor	• Continuous
	• Spontaneous limb movements	• Only stretches • Mouthing • Jerky or other abnormal movements • Fisted hand(s) • Cramped synchronised (rigid and lack normal smooth and fluent character) as seen in general movements assessment
Posture	• Position of limbs at rest	• Arms and legs extended or very slightly flexed • Abnormal posture–opisthotonos; arm flexed, leg or foot extended
Arm traction	• Normal resistance • Arm recoil	• Arms remain straight, no resistance • Flexion of arms less than 100° maintained when body lifts up
Leg traction	• Normal resistance • Leg recoil	• Leg straight, no resistance • Knee flexion remains when back and buttocks raised
Head control	• Raises head • Head: o Drops forward or back (36–37 weeks gestational age) o May wobble (after 37 weeks gestational age)	• No attempt to raise head from flexion • No attempt to raise head from extension–head remains upright or neck extended; cannot be passively flexed
Head lag	• Resistance to gravity	• Head drops and stays extended • Head in front of line of body
Ventral suspension	• Back slightly curved, limbs flexed (36–37 weeks gestational age) • Back straight, limbs flexed (after 37 weeks gestational age)	• Back curved, heads and limbs hang straight • Back straight, head above line of body

Adapted from: Hawes J, Bernardo S, Wilson D. The neonatal neurological examination: improving understanding and performance. Neonatal Network 2020;39:116-28; Kaur S, Pappas K. Genetic etiologies of neonatal seizures. Neoreviews 2020;21(10):e663-e72; Kotagal S. Neurological examination of the newborn. 2020. Available from: https://www.uptodate.com; Mercuri E, Ricci D, Pane M, Baranello G. The neurological examination of the newborn baby. Early Human Development 2005;81(12):947-56

Appendix C: Genetic epilepsy syndromes presenting in neonates or infants

	Self-limited (familial) neonatal epilepsy (SeLNE)[1,2]	Self-limited familial neonatal-infantile epilepsy (SeLFNIE)[1,2]	Self-limited familial (familial) infantile epilepsy (SeLIE)[1,2]	Early infantile developmental and epileptic encephalopathy (EIDEE)[1,2]
Presen-tation	• Seizure activity starts from day 2–7 o If preterm, within days of being term by corrected age • Can be repetitive over hours to days • Remits by 4–6 months of age (most by 6 weeks of age)	• Day 1 to 23 months of age	• Seizure activity starts from 3–20 months • Remit within one year of onset	• Present from birth to 3 months of age (adjusted for prematurity) • Onset of epilepsy in first 3 months of life • Frequently medication resistant seizures
Seizure type	• Focal tonic at onset affecting head, face and limbs • May progress in sequential pattern with tonic, clonic, myoclonic and autonomic features following each other without single dominant feature • May alternate sides • Tonic features, vocalisations, autonomic features and/or automatisms • May be focal to bilateral tonic-clonic • Brief (up to three minutes) • Apnoea and cyanosis in one third • Clusters over days or hours may occur with normal behaviour between	• Varies from a few to clusters of many seizures • Initially focal tonic with head and eye deviation • May have apnoea and staring • Duration from 20 seconds to four minutes • Ceases by 12–24 months of age	• Focal seizures with behavioural arrest, staring with impaired awareness, automatisms (e.g. cyanosis), head/eye version, and clonic movements • May alternate sides • May progress to hemiclonic or focal to bilateral tonic-clonic seizure • Usually brief (less than three minutes), but can be frequent	• Focal tonic, generalised tonic, myoclonic, focal clonic and epileptic spasms–may be sequential • If tonic (focal or asymmetric in neonatal period) frequently occurring in isolation or cluster of 10–20 per day, independent of sleep cycle • Focal or multifocal clonus may be predominant o Can occur in face or extremities, or restricted to an eyebrow, finger or lip o Occurs during wakefulness and sleep o Commonly associated with metabolic aetiology • Epileptic spasms: usually occur: o Beyond first month of life o In clusters often on wakening • Sequential seizures o Several manifestations occurring in sequence (e.g. may begin with focal tonic, then focal clonic, then epileptic spasms without predominant manifestation)
History	• Family history • Pregnancy and birth history normal	• Unremarkable perinatal history	• Pregnancy, birth and neonatal history normal • Family history of self-limited familial neonatal epilepsy	• Family, pregnancy and birth history usually normal
Neuro exam	• Normal except immediately postictal or if baby is sedated	• Normal	• Normal	• Abnormal tone (frequently central hypotonia), posture, motor behaviour with cortical visual impairment
Neuro-imaging	• Normal	• Does not show cause	• Typically normal	• May show structural brain abnormalities
EEG	• Normal or show minor non-specific abnormalities	• Typically normal	• Normal or show focal slowing postictally	• Abnormal
Common aetiology	• Autosomal genetic patterns including de novo pathogenic variants	• Genetic origin	• 80% genetic	• Metabolic or other genetic
Neuro-develop-ment	• Milestones usually normal	• Usually normal	• Milestones usually normal	• Impairment prior to or shortly after seizure onset • Abnormal including intellectual disability - from underlying aetiology independent of epileptiform activity or epileptic encephalopathy • Moderate/profound impairment with poor prognosis

1. International League Against Epilepsy. Diagnostic manual: self limiting neonatal seizures. 2022. Available from: https://www.epilepsydiagnosis.org. **2.** Zuberi SM, Wirrell E, Yozawitz E, Wilmshurst JM, Specchio N, Riney K, et al. ILAE classification and definition of epilepsy syndromes with onset in neonates and infants: Position statement by the ILAE Task Force on Nosology and Definitions. Epilepsia 2022;63(6):1349-97.

Appendix D: EEG and subsequent investigations

Aspect	Consideration
Monitoring[1]	• If available and suggested by paediatric neurologist, commence 12 lead EEG monitoring to confirm clinical event is seizure activity • More accurate than clinical observation alone • Most accurately interpreted with knowledge of medical history and overall clinical condition
aEEG[1]	• Amplitude integrated EEG (aEEG) o Convenient bedside tool using limited number of channels o Useful for monitoring background brain activity (e.g. identifying variability as a sign of neurological wellbeing) o Can be continued for several days o Lower sensitivity and specificity than EEG for brief, focal seizures o Filtered, processed and displayed in time-compressed scale • Has prognostic value in assessing general neurological well-being
EEG[2,3]	• EEG–conventional, video EEG (duration specific to clinical condition) o Recommended for babies at high risk for seizures and/or paroxysmal events (if available) o Initiated and may be extended based on risk stratification by neurologist
Seizure diagnosis[4]	• Seizures are diagnosed if EEG: o Has a pattern that evolves in frequency, voltage morphology and/or field with time o Is abnormal with repetitive and evolving pattern, voltage greater than two microvolts and duration greater than 10 seconds

Subsequent investigations

Aspect	Consideration
Clinical[5]	• Pyridoxine test (if baby's seizures refractory and unresponsive to conventional ASM)
Blood[5,6,7]	• Ammonia • If metabolic or other genetic cause suspected o Genetic tests with neurologist and or metabolic specialist consultation—phenotyping and advice in relation to optimising test type and result (e.g. SNP array)
CSF[5]	• If congenital or acquired infection suspected and lumbar puncture not previously done o Culture and PCR for CSF pathogens • Amino acids • Neurotransmitters
Urine[5]	• CMV • If metabolic cause of seizures suspected, metabolic screen for amino acids and organic acids • Alpha aminoadipic semialdehyde • Sulfites • Creatinine
Other[7]	• Congenital infection screen (in addition to initial TORCH screen) o Varicella zoster o Parvovirus B19
Neuroimaging[5]	• MRI imaging • MRI spectroscopy

1 Ryan MA, Mathieson S, Dempsey E, Boylan G. An introduction to neonatal EEG. The Journal of Perinatal & Neonatal Nursing 2021;35(4):369-76.
2 Pisani F, Spagnoli C. Diagnosis and management of acute seizures in neonates. In: Perlman JM, Cilio MR, editors. Neurology (Third Edition). Philadelphia: Elsevier; 2019. p. 111-29.
3. Macdonald-Laurs E, Sharpe C, Nespeca M, Rismanchi N, Gold JJ, Kuperman R, et al. Does the first hour of continuous electroencephalography predict neonatal seizures? Archives of Diseases in Childhood: Fetal & Neonatal 2021;106(2):162-7.
4. Pressler RM, Cilio MR, Mizrahi EM, Moshé SL, Nunes ML, Plouin P, et al. The ILAE classification of seizures and the epilepsies: modification for seizures in the neonate. Position paper by the ILAE Task Force on Neonatal Seizures. Epilepsia 2021;62(3):615-28.
5. Shellhaus R. Clinical features, evaluation, and diagnosis of neonatal seizures. Waltham MA: UpToDate; 2021. Available from: https://www.uptodate.com
6. Soul JS. Acute symptomatic seizures in term neonates: etiologies and treatments. Seminars Fetal & Neonatal Medicine 2018;23(3):183-90.
7. Chalia M, Austin T. Practice guide to neonatal seizures. Paediatrics and Child Health 2018;28(10):488-91.

Acknowledgements

Queensland Clinical Guidelines gratefully acknowledge the contribution of Queensland clinicians and other stakeholders who participated throughout the guideline development process particularly:

Working Party Clinical Leads

Professor Paul Colditz, Consultant Neonatal Paediatrician Royal Brisbane and Women's Hospital, Director Perinatal Research, University of Queensland
Ms Anndrea Flint, Neonatal Nurse, Caboolture Hospital

QCG Program Officer

Ms Stephanie Sutherns

Working Party Members

Mrs Seija Argyros, Neonatal Nurse Practitioner, Royal Brisbane and Women's Hospital
Associate Professor Ravi Bala, Paediatrician, Gold Coast University Hospital, Pindara, Gold Coast Private Hospitals, University of Queensland
Ms Maxine Ballinger, Clinical Nurse Consultant, Rockhampton Hospital
Miss Chase Becker, Clinical Nurse/Registered Midwife, Pournara Refugee Centre, Cyprus
Miss Zoe Collins, Clinical Nurse, Mater Mothers' Hospital
Ms Eileen Cooke, Consumer Representative, Preterm Infants Parents Association Incorporated
Dr Emily Cripps, Neonatal Fellow, Mater Mothers' Hospital
Professor Mark Davies, Neonatologist, Royal Brisbane and Women's Hospital
Dr Jocelyn Domingo-Bates, Neonatologist, Mater Mothers' Hospital
Miss Tania Dowdle, Clinical Nurse, Mater Mothers' Hospital
Mrs Anne-Marie Feary, Clinical Research Nurse, Varsity Lakes Day Hospital
Ms Carla Finch, Consumer Representative, Maternity Choices Australia
Mrs Cerys Firth, Registered Nurse, Rockhampton Hospital
Dr Corey Forrest, Neonatal Fellow, Royal Brisbane and Women's Hospital
Dr Deborah Gilmour, Neonatologist, Mater Mothers' Hospital
Dr Kristen Haakons, Neonatal Fellow, Mater Mother's Hospital
Dr Shivanand Hebbandi, Paediatrician, Redland Hospital
Dr Lisa Hong, Neonatal Fellow, Royal Brisbane and Women's Hospital
Ms Karen Hose, Neonatal Nurse Practitioner, Royal Brisbane and Women's Hospital
Mrs Kristin Hughes, Clinical Nurse Consultant, Royal Brisbane and Women's Hospital
Dr Meghan Jones, Neonatal Senior Registrar, Royal Brisbane and Women's Hospital
Dr Victoria Kain, Senior Lecturer, School of Nursing and Midwifery, Griffith University
Dr Lisa Kane, Paediatrician, Caboolture Hospital
Ms Yanna Klaassen, Registered Nurse/Midwife, Bundaberg Hospital
Dr Melissa Lai, Neonatologist, Royal Brisbane and Women's Hospital
Professor Helen Liley, Neonatologist, Mater Mothers' Hospital
Associate Professor Kate Riney, Paediatric Neurologist/Epileptologist, Queensland Children's Hospital/University of Queensland
Dr Jamie Ross, Paediatrician, Redlands Hospital
Ms Kahli Sanders, Paediatric Speech Pathologist, Royal Brisbane and Women's Hospital
Dr Philip Scott, Neonatal Registrar, Mater Mothers' Hospital
Mrs Anu Surendran, Registered Nurse/Nurse Practitioner , Atherton Hospital
Mrs Elizabeth Upton, Clinical Pharmacist, Sunshine Coast University Hospital
Dr Michaela Waak, Senior Medical Officer, Queensland Children's Hospital
Dr Lizelle Weber, Director of Neonatology, Sunshine Coast University Hospital
Dr Christina White, Paediatrician, Bundaberg Hospital
Mrs Deborah Wright, Clinical Nurse, Sunshine Coast University Hospital

Queensland Clinical Guidelines Team

Professor Rebecca Kimble, Director
Ms Jacinta Lee, Manager
Ms Stephanie Sutherns, Clinical Nurse Consultant
Ms Cara Cox, Clinical Nurse Consultant
Ms Emily Holmes, Clinical Nurse Consultant
Ms Janene Rattray, Clinical Nurse Consultant
Steering Committee

Funding

This clinical guideline was funded by Healthcare Improvement Unit, Queensland Health.