Clinical Guidance

Neonatal Manual Chapter 5: Nervous System

Summary
This manual contains clinical guidelines developed by the Neonatal Unit multidisciplinary team over recent years. This chapter contains guidelines on management of babies with various problems reacted to the nervous system. It is linked to and should be used in conjunction with the completed neonatal manual details of which are contained in the introductory chapters.

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5.1 NEONATAL ENCEPHALOPATHY (NE)

Definition
Neurological manifestations of acute disturbance of brain function manifested by difficulty with
initiating and maintaining respiration, depression of tone and reflexes, subnormal level of
consciousness and often seizures.

Incidence
2-6/1000 live births (1-2/1000 severe)

Aetiology
NE can result from a wide variety of conditions and occasionally remains unexplained.

• Hypoxic-ischaemic encephalopathy (HIE) is a major cause of NE. However, only 5-10% of the
cases have identifiable evidence of intrapartum events that may have caused this. Therefore,
terms such as birth asphyxia should be avoided until a thorough clinical evaluation is made
• Perinatal stroke (infarction)
• Metabolic conditions
• Sepsis
• Intracranial hemorrhage
• Intra-ventricular hemorrhage (in term infants this commonly accompanies sinus venous
thrombosis)
• Other causes

Clinical features and staging
Common presentations of infants with acute brain dysfunction are

• Neonatal seizures
• Irritability
• Abnormal tone
• Poor feeding
• Coma and stupor
• Respiratory insufficiency

Staging:

<table>
<thead>
<tr>
<th>Stage 1 – mild</th>
<th>Stage 2 – moderate</th>
<th>Stage 3 – severe</th>
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</thead>
<tbody>
<tr>
<td>Conscious level</td>
<td>Hyperalert</td>
<td>Lethargic</td>
</tr>
<tr>
<td>Muscle tone</td>
<td>Minor disturbances</td>
<td>Significant disturbances</td>
</tr>
<tr>
<td>Feeding</td>
<td>Slight difficulties</td>
<td>Poor</td>
</tr>
<tr>
<td>Seizures</td>
<td>No</td>
<td>Yes</td>
</tr>
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<td>---------</td>
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<tr>
<td>Able to maintain vital functions</td>
<td>Yes</td>
<td>Yes</td>
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Sarnat and Sarnat originally described 3 states of HIE with baby assigned to the maximum level reached.

Levene’s staging scheme may be more practical as the categories are more mutually exclusive.

Other systems are also commonly involved as a further consequence of inadequate organ perfusion. This may be manifested as:

- Acute tubular necrosis leading to acute renal failure (see separate protocol)
- Hypoglycaemia secondary to glucose and glycogen consumption during compensatory anaerobic metabolism, which is energy inefficient. This may cause hypoglycaemic convulsions
- Necrotising enterocolitis or dysmotility with ileus and dilated loops
- (Transient) myocardial ischaemia, which may result in systemic hypotension, possibly exacerbated by poor peripheral vasomotor tone
- Disseminated intravascular coagulation (DIC)
- Shock lung/acute RDS
- Pulmonary hypertension
- Hepatic failure
- Acute cold injury to skin

**Management**

- Adequate resuscitation should be instituted at birth. If difficult or prolonged resuscitation inform the attending neonatal consultant immediately
- Check glucose level as soon as possible after resuscitation. Hypoglycemia is considered an important risk factor for perinatal brain injury particularly in depressed infants
- Keep baby’s temperature in the normal range. Take care not to overheat infants under the resuscitaire
- Make a detailed record of the resuscitation to include:
  - The time when respiration established,
  - The initial heart rate and when it recovered.
  - The neurological state
  - Interventions required.
  - The arterial and venous cord gas
- Request the placenta to be sent for histological examination
• Consider the diagnosis and aetiology and investigate as appropriate. Always check blood cultures and early arterial (not capillary) blood gas.

• Discuss all cases with attending consultant. If encephalopathy is secondary to HIE then therapeutic hypothermia is indicated and should be instituted as soon as possible after birth (see separate protocol)

On going management is mainly supportive:

• Ventilation: ventilation may be required if there is inadequate respiratory effort or frequent apnoeas. Remember most of these babies do not have lung pathology and may only require minimal support. Avoid hyperventilation which will lead to cerebral vasoconstriction and exacerbate the brain injury, therefore maintain PaCO₂ in normal range between: 5-7 kPa.

• Circulation:
  - Maintain normal blood pressure. Cerebral blood flow as cerebral perfusion pressure (difference between mean arterial pressure and intracranial pressure) is dependent on MAP.
  - Carefully monitor fluid balance. Fluid restriction may be appropriate if there is concurrent acute renal failure (see separate protocol) but there is no evidence that fluid restriction/diuretics are beneficial for encephalopathy per se, unless there is fluid overload or cerebral oedema. Cerebral oedema is very difficult to diagnose: full or tense fontanelle with invisible ventricles on cranial ultrasound can (but ventricles are normally very small in term infants). In established cerebral oedema, fluid restrict as tolerated (start at 40ml/kg/24 hours and increase according got urine output, daily weight etc)
  - Blood Glucose: it is imperative to maintain blood glucose levels above 2.6mmols/L. In fluid restricted patients the glucose flow rate should be increased accordingly. If more than 12.5% dextrose required central venous access should be established (UVC or long line )

• Neurology:
  - Commence therapeutic hypothermia if indicated (see separate protocol)
  - Treat seizures: all electrographic seizures should be treated (see seizure protocol). Consider anticonvulsants even if baby is very irritable but not obviously seizing
  - Apply aEEG as soon as practical
  - Request a full EEG
  - Imaging:
    - Obtain cranial US scan including cerebral artery Doppler measurements. Although not very detailed, US images may reveal parenchymal lesions, presence of infarctions, basal ganglia changes and presence of cerebral oedema.
    - Brain MRI can offer further prognostic information and may help to clarify the aetiology of NE. It should be performed in the first 5 days of life in most patients. If therapeutic hypothermia was applied then MRI scan should be slightly delayed and performed between 7-10 days
• Treat other organ failure as appropriate
• Consider the diagnosis and aetiology and investigate as appropriate. Always check blood culture and early arterial (not capillary) blood gas
• Keep parents well informed. Ensure documentation of all management decisions and parent discussions.

Prognosis and follow-up:
• The baby’s staging (based on either Levene or Sarnat system) according to their worst clinical condition is probably the best guide to prognosis.
  - Stage 1: up to 100% full recovery with normal neurological follow-up
  - Stage 2: approximately 75% full recovery with normal neurological follow-up
  - Stage 3: up to 100% die or have significant neurodevelopmental abnormalities
• The rate of improvement in the baby’s neurological condition may also have prognostic value but note that anticonvulsants and therapeutic hypothermia may modify the initial neurological assessments.
• Results of brain imaging and EEG may also provide valuable prognostic information. These should be considered along with the baby’s neurological examination findings and discussed with the relevant sub-specialist on a case by case basis.
• Arrange outpatient follow-up at the Mary Sheridan Centre for all Lambeth and Southwark babies with appreciable encephalopathy (i.e. stage 2 or 3).

Further reading
• Denis Azzopardi, Clinical management of the baby with hypoxic ischaemic encephalopathy, Early Human Development, Volume 86, Issue 6, June 2010,
• Donna Ferriero, Neonatal Brain Injury, New England Journal, 351;19, 1994
5.2 NEONATAL SEIZURES

Neonatal seizures are often subtle and difficult to recognize, often including repetitive movements, staring and apnoea.

Incidence

Term infants 2.6 / 1000 live births
Preterm infants 11.1 / 1000 live births

Causes of Neonatal seizures

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Contribution (%)</th>
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<tbody>
<tr>
<td>HIE</td>
<td>46</td>
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<tr>
<td>CNS infection</td>
<td>17</td>
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<td>Intracranial Infection</td>
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<td>Cerebral Artery Infarction</td>
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<tr>
<td>Acute metabolic disorder</td>
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<tr>
<td>Inborn error of metabolism</td>
<td>4</td>
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<tr>
<td>CNS malformation</td>
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<tr>
<td>Maternal drug intoxication</td>
<td>?</td>
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<tr>
<td>Benign neonatal Seizures</td>
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<tr>
<td>Idiopathic</td>
<td>2</td>
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Clinical features

- Onset
  - 36% in 1st 24 hours
  - 64% in 1st 48 hours
  - 83% in 1st week

- Type - may be:
  - multifocal or generalized
  - clonic, myoclonic or tonic
  - electrographic with no clinical signs

Investigations

- Initial investigations / monitoring:
  - Oxygen saturation, heart rate and apnoea monitoring
  - Blood glucose
  - Neonatal biochemistry profile including Ca, Mg
  - FBC
  - Blood gas
- Septic screen including lumbar puncture
- Cranial ultrasound scan
- Apply aEEG, request EEG

- If seizures persist or if otherwise indicated from results of initial investigations
  - Brain MRI
  - Urine for toxicology
  - Investigations for congenital infection
  - Plasma ammonia
  - Plasma amino acids
  - Urine organic and amino acids
  - Plasma and CSF lactate and pyruvate
  - CSF glycine

- EPR has a ‘Neonatal Unit – Encephalopathy’ order set for reference

Treatment:
- Treat the underlying cause
- Treat if more than 2 seizures/hour or one seizure of more than 3 minutes duration
  - Phenobarbitone 18-20mg/kg IV over 30 minutes loading dose. If seizures uncontrolled, give up to 2 further IV doses of 10 mg/kg over 30 minutes (i.e. max. total dose of 40 mg/kg). Obtain phenobarbitone level and if in normal range commence maintenance dose 5mg/kg/day (starting 24 hours after loading dose)
- If seizures uncontrolled with phenobarbitone, use 2nd line treatment of phenytoin or lidocaine
  - Phenytoin – 20mg/kg (slow IVI over ~20 mins.). If seizures not controlled, give 2nd dose of 10mg/kg (slow IVI over ~20 mins.), then maintenance dose 2-4mg/kg BD (IV or oral)
  - Lidocaine – loading dose 2 mg/kg dose (0.2 ml/kg of a 1% solution of adrenaline-free lidocaine IV over 10 minutes), followed by a maintenance infusion of 6 mg/kg per hour for 6 hours. Then tail treatment off over the next 24 hours (4 mg/kg per hour for 12 hours followed by 2 mg/kg per hour for 12 hours). ECG monitoring is necessary.
- If IV access is problematic, paraldehyde (0.4ml/kg, diluted 50:50 with olive oil to make 0.8ml/kg of solution ) may be given
- Clonazepam (IV infusion 10-60micrograms/kg/hr.) may be considered if Phenobarbitone and either Phenytoin or Lidocaine have failed to adequately control seizures. However, there is no evidence that Clonazepam has any effect in reducing the burden of electrographic seizures.
- Refer to Paediatric Neurology team if seizures difficult to control
• Aim to stop anticonvulsant treatment early if seizures well controlled, within 1st week of life if possible

NOTE: In infants treated with therapeutic hypothermia:
• The serum concentrations of medications may remain higher for longer than in non-cooled infant
• Infants may manifest further seizures when therapeutic hypothermia discontinued and rewarming commences

Further Reading
• Neonatal Seizures: an update on mechanisms and management, Jensen FE Clin Perinatol. 2009 Dec;36(4)
• Neonatal Seizures, Silverstein FS, Jensen FE. Ann Neurol. 2007 Aug;62(2)
5.3 THERAPEUTIC HYPOTHERMIA (TH)

Therapeutic hypothermia is the only proven neuroprotective therapy that may benefit infants with hypoxic ischemic encephalopathy (HIE).

A recent meta-analysis has confirmed that TH:

- significantly reduced the combined rate of death and severe disability at 18 months (risk ratio 0.81, 95% confidence interval 0.71 to 0.93, P=0.002; risk difference −0.11, 95% CI −0.18 to −0.04), with a number needed to treat of nine (95% CI 5 to 25)
- increased survival with normal neurological function (risk ratio 1.53, 95% CI 1.22 to 1.93, P<0.001; risk difference 0.12, 95% CI 0.06 to 0.18), with a number needed to treat of eight (95% CI 5 to 17)
- in survivors reduced the rates of severe disability (P=0.006), cerebral palsy (P=0.004), and mental and the psychomotor developmental index of less than 70 (P=0.01 and P=0.02, respectively).
- Mortality was significantly reduced (1320 infants; relative risk 0.78, 95% CI 0.66 to 0.93, P=0.005; risk difference −0.07, 95% CI −0.12 to −0.02), with a number needed to treat of 14 (95% CI 8 to 47)

At St Thomas TH is applied by total body cooling using the Criticon cooling system. (Details on the use of the device are attached to the cooling system)

Selection of patients

Standard Criteria

The standard selection criteria are based on the published trials and are outlined below. It is important to identify the patients that may benefit from TH early. The therapeutic window for commencing this treatment is 6 hours from the time of birth.

The selection criteria are:

A. Infants ≥ 36 weeks gestation admitted to the neonatal unit with at least one of the following:
   - Apgar score of ≤ 5 at 10 minutes after birth
   - Continued need for resuscitation, including endotracheal or mask ventilation at 10 minutes after birth
   - Acidosis within 60 minutes after birth (defined as any occurrence of umbilical cord, arterial or capillary pH< 7.00)
   - Base deficit ≥ 16 mmol/L in umbilical cord or any blood sample (arterial, venous or capillary) within 60 minutes of birth

Infants that meet the criteria A should be assessed if they meet the neurological abnormality entry criteria outlined in B.
B. Seizures or moderate to severe encephalopathy, consisting of one of the following:

- Altered state of consciousness (reduced or absent response to stimulation)
- Abnormal tone (focal or general hypotonia, or flaccid)
- Abnormal primitive reflexes (weak or absent suck or Moro response)

Criteria for helping define moderate and severe encephalopathy are listed in the table:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Moderate encephalopathy</th>
<th>Severe encephalopathy</th>
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<tbody>
<tr>
<td>Level of consciousness</td>
<td>Reduced response to stimulation</td>
<td>Absent response to stimulation</td>
</tr>
<tr>
<td>Spontaneous activity</td>
<td>Decreased activity</td>
<td>No activity</td>
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<tr>
<td>Posture</td>
<td>Distal flexion, complete extension</td>
<td>Decerebrate</td>
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<tr>
<td>Tone</td>
<td>Hypotonia (focal or general)</td>
<td>Flaccid</td>
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<tr>
<td>Suck</td>
<td>Weak</td>
<td>Absent</td>
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<tr>
<td>Moro</td>
<td>Incomplete</td>
<td>Absent</td>
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<tr>
<td>Pupils</td>
<td>Constricted</td>
<td>Constricted</td>
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<tr>
<td>Heart rate</td>
<td>Bradycardia</td>
<td>Variable</td>
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<tr>
<td>Respiration</td>
<td>Periodic breathing</td>
<td>Apnoea</td>
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All infants that meet the criteria A and B should be considered for therapeutic hypothermia.

Cooling and aEEG:

- aEEG should be applied in all infants treated with cooling but cooling need not be delayed until aEEG is initiated.
- The aEEG is not currently considered an entry criterion for hypothermia however a completely normal trace indicates a high probability of normal outcome and clinicians may consider that further treatment with cooling is not required. (Please discuss this with the attending consultant)
- However apparent improvement of the aEEG trace after 6 hours of age may not be an indication to discontinuing therapeutic hypothermia.
- Continued aEEG recording during the treatment period is helpful to assess the occurrence of seizures and to monitor the severity of encephalopathy.

Therapeutic hypothermia: key points

- Start treatment as early as possible and not more than 6 hours from birth
- The target rectal temperature is 33.5°C
- If there is a sudden change in the displayed rectal temperature check that the temperature probes (axilla and rectal) are correctly placed
- Cooling treatment duration is 72 hours
• Cooling may be applied to non ventilated infants but if appear distressed adequate sedation should be provided

• Effects of treatment
  - Cooling reduces the HR: most babies will have a HR around 100 bpm
  - A mild increase in BP can also be seen. Remember it is important to keep mean BP>40mm Hg
  - Cooling may lead to increase concentration of medications (i.e: anticonvulsants, gentamicin)
  - Cooling may lead to a decrease of platelets and slight coagulation abnormalities but no significant bleeding problems have been reported in the clinical trials

• Re-warming
  - Following completion of treatment it is important that baby is rewarmed to normal temperature very slowly
  - The rate of increase in rectal temperature should not be more than 0.3°C/ hour
  - Be aware that cooling suppresses seizure activity, therefore increased seizures may occur during rewarming
  - Rewarming may cause hypotension secondary to peripheral vasodilatation that should be sought and treated if occurs

Patients referred to Evelina Children's Hospital Neonatal Unit for cooling:
• Patients referred to St Thomas to receive therapeutic hypothermia will have cooling instituted by the transport team provided they are able to retrieve the baby within 6 hours from birth.
• If that is the case the baby should be placed on the cooling system when arrives at our unit irrespective of the time of birth. Usual monitoring should be placed including aEEG.
• The time of treatment will start from the time the infant was placed on the cooling system after arrival to St Thomas’ and cooling should be continued for 72 hours.

Use of Therapeutic Hypothermia outwith TOBY guidelines

The use of TH for HIE in the following babies remains in the unstudied realm for cooling therapy and is not supported by clinical trials:
• Infants > 6 hours old
• Infants < 36 weeks gestation
• Infants with sudden unexpected postnatal collapse (when the clinical deterioration is considered to be primarily secondary to hypoxia ischaemia)
However it is possible that TH may be of benefit in selected cases. These cases should be discussed with the attending neonatal consultant. TH may be considered if the mechanism of neonatal encephalopathy is considered to be primarily due to hypoxia-ischaemia.

Consent should be obtained if TH in these babies is being considered as the use of TH in this setting is outside current standard of care. Discussion with parents should outline the proposed treatment, potential benefits and the lack of efficacy and safety data.

Further Reading

- Neurological outcomes at 18 months of age after moderate hypothermia for perinatal hypoxic ischaemic encephalopathy: synthesis and meta-analysis of trial data Edwards et al, BMJ 2010;340:c363
- Moderate Hypothermia to Treat Perinatal Asphyxial Encephalopathy, Azzopardi et al NEJM, 2009; 361:1349-1358
- https://www.npeu.ox.ac.uk/toby/protocol
5.4 SUDDEN UNEXPECTED POSTNATAL COLLAPSE (SUPC)

Sudden and unexpected postnatal collapse of a healthy newborn is a rare event but carries a high risk of mortality and significant neurodisability in survivors. It may occur in 0.03-0.08/1000 livebirths (BPSU data: incidence in the first 12 hours after birth in the UK 1/20000). As these infants are considered to be well in the immediate period after birth, they are frequently nursed on the labour ward and/or postnatal ward and the timing of the deterioration or the sequence of events leading to the episode may not be immediately apparent or witnessed.

Infants who suffer SUPC are defined as:
- Term or near term (>35 weeks gestation)
- Well at birth (normal 5 minute APGAR scores and considered for routine postnatal care)
- Unexpected collapse – required resuscitation with IPPV ± cardiac compressions
- Postnatal age < 7 days
- Infant dies or requires intensive care admission or develops encephalopathy

The cause of the collapse may not be immediately apparent and we should avoid attributing a cause until thorough review of the events preceding the event and results of detailed investigations are available.

In this group of infants underlying congenital anomalies and conditions arising through pregnancy and birth are common. If the baby dies, a cause of the SUPC can be determined in 60% following post-mortem examination.

From the data collated by the BPSU (2008-2009) in the UK of infants with SUPC presenting in the first 12 hours after birth, 26.6% died and an underlying disease or abnormality was detected in 33.3%. In the infants where no cause was found there was a clinical/pathological diagnosis of airway obstruction during breast feeding or prone position in 80%.

Immediate management
- Respond to the cardiac arrest call
- Resuscitate the infant in accordance to Resuscitation Council/ILCOR guidance
- Admit the infant to NICU and institute intensive care treatment as appropriate
- Apply aEEG monitoring as soon as practical
- Inform the attending consultant
- Avoid ascribing a cause for the collapse before completing detailed diagnostic work up
- Update the parents as soon as possible. Ensure documentation of all management decisions and parent discussions.
- Although not evidence-based, some of the infants that suffer SUPC may be considered for the use of Therapeutic Hypothermia (see TH guideline). Discuss this with the attending consultant.
**Investigation**

This should be directed by the history and clinical findings. Possible causes and diagnostic investigations are outlined in the table below. BAPM has endorsed the guidelines for investigation of newborn infants that suffer SUPC and they are available from [http://www.bapm.org/publications/documents/guidelines/SUPC_Booklet.pdf](http://www.bapm.org/publications/documents/guidelines/SUPC_Booklet.pdf)

Causes of SUPC and proposed investigations

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<thead>
<tr>
<th>Condition</th>
<th>Range of investigations to detect conditions in each category</th>
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<tbody>
<tr>
<td><strong>Infection</strong></td>
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<tr>
<td>- Systemic: Bacterial infection – various</td>
<td>- Placenta: histopathology, bacteriology</td>
</tr>
<tr>
<td>- Viral infection – Echovirus, Coxsackie, Respiratory Syncytial Virus, Parvovirus, Herpes</td>
<td>- Maternal blood: viral titres</td>
</tr>
<tr>
<td>- Meningitis: bacterial, viral</td>
<td>- Maternal high and low vaginal swabs: bacteriology</td>
</tr>
<tr>
<td><strong>Cardiac anomalies</strong></td>
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<tr>
<td>- Cyanotic heart disease: Transposition of the great arteries, Truncus arteriosus, Univentricular heart, Pulmonary stenosis/ataresia, Tricuspid atresia</td>
<td>- Blood: culture, viral titres, storage</td>
</tr>
<tr>
<td>- Left sided obstructive lesions: Coarctation/interruption of the aorta, Hypoplastic left heart, Aortic stenosis</td>
<td>- CSF: bacteriology, virology, biochemistry, glucose</td>
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<td>- Cardiac conduction problems: Long QT syndrome, Atrial fibrillation</td>
<td>- Urine: bacteriology, virology</td>
</tr>
<tr>
<td>- Other: Total anomalous pulmonary venous drainage, Myocardial infarction, Cardiomyopathies, Barth syndrome, Congenital coronary artery aneurysm, Anomalous coronary artery</td>
<td>- Surface swabs: bacteriology</td>
</tr>
<tr>
<td><strong>Respiratory Conditions</strong></td>
<td>See investigations for infection</td>
</tr>
<tr>
<td>- Pneumonia</td>
<td>- Chest X-ray</td>
</tr>
<tr>
<td>- Pulmonary hypertension</td>
<td>- Echocardiogram</td>
</tr>
<tr>
<td>- Pulmonary haemorrhage</td>
<td>- Endotracheal secretions: bacteriology</td>
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<tr>
<td>- Aspiration</td>
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<tr>
<td>Haematological</td>
<td>Endocrine</td>
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<td>Accidental smothering</td>
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<td>Full blood count and film</td>
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### Neurological
- Any metabolic cause of seizures/apnoea
- Drug withdrawal
- Perinatal infarction
- Intracranial bleed
- Antenatal injury
  - Hypoplasia of brainstem nuclei
  - Hyperekplexia: apnoea/tonic
  - Congenital hypoventilation syndrome
  - Rett syndrome variants
  - Joubert syndrome
- Metabolic as above
  - EEG/aEEG/cerebral function monitor
  - Maternal and infant urine and blood toxicology
  - Coagulation screen
  - Cranial ultrasound scan
  - MRI brain
  - Blood
  - skin biopsy
  - PHOX2B sequencing (congenital hypoventilation syndrome)
  - MECP2 sequencing and copy estimation (Rett syndrome variants)

### Further Reading
- Unexpected collapse in apparently healthy newborns – a prospective national study of a missing cohort of neonatal deaths and near-death events,,Julie-Clare Becher et al *Arch Dis Child Fetal Neonatal Ed* 2012;97:1
5.5 PERI/INTRAVENTRICULAR HAEMORRHAGE (PIVH), POST-HAEMORRHAGIC VENTRICULAR DILATATION (PVHD) AND HYDROCEPHALUS (PHH)

Definitions
PIVH is bleeding limited to the subependymal area, into the cerebral ventricles, or involving the adjacent cerebral parenchyma. It commonly occurs silently and is diagnosed ultrasonographically. There are several grading schemes, but description of the morphology of the IVH is recommended using the table below.

Ventricular dilatation (ventricular index) is measured by comparison of the width of the lateral ventricle (measured on the coronal view in the plane of the 3rd ventricle – midline to most lateral part of lateral ventricle on either side) to the normal width for corrected gestational age. Conventional wisdom is to define PHVD as growth of the lateral ventricles to above 4 mm wider than the 97th centile for corrected gestational age following PIVH.

Hydrocephalus is defined as abnormally rapid head growth as a consequence of impaired CSF absorption.

<table>
<thead>
<tr>
<th>Morphology</th>
<th>Grade</th>
<th>Location of blood</th>
<th>Immediate symptoms</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subependymal haemorrhage (SHE), germinal matrix haemorrhage (GMH)</td>
<td>I</td>
<td>Adjacent and just below the lateral ventricle, over the head of the caudate nucleus, outside the ependyma</td>
<td>Silent</td>
<td>No long term effects</td>
</tr>
<tr>
<td>Uncomplicated intraventricular haemorrhage (IVH)</td>
<td>II</td>
<td>Within the lateral ventricle but without change in its size or shape</td>
<td>Silent</td>
<td>No long term effects</td>
</tr>
<tr>
<td>IVH with ventricular dilatation</td>
<td>III</td>
<td>Within and distending the lateral ventricle</td>
<td>Occasionally shock from blood volume depletion</td>
<td>Risk of CP and cognitive delay. Sometimes hydrocephalus</td>
</tr>
<tr>
<td>Haemorrhagic parenchymal infarction (HPI)</td>
<td>III (Levene), IV (Papile)</td>
<td>IVH plus echogenic wedge-shaped echolucency adjacent to the lateral ventricle (usually superolateral)</td>
<td>Occasionally shock. Very rarely neurological signs (fits, hypotonia, reduced level of consciousness, raised ICP)</td>
<td>Very high risk of CP (spastic hemiplegia), and maybe cognitive delay. Sometimes hydrocephalus</td>
</tr>
</tbody>
</table>
PIVH with dilatation and HPI are termed “complicated PIVH” and lesser degrees of PIVH as “uncomplicated”.

**Pathogenesis**
It is thought that PIVH is usually a consequence of bleeding from the vascular but immature germinal layer, which overlies the head of the caudate nucleus in the fetus until about 30-32 weeks gestation. The bleeding comes about mainly because of unstable perfusion, a consequence of cardiovascular immaturity in the preterm infant, aggravated if they are sick/unstable.
HPI probably occurs due to venous infarction due to compromised venous drainage, rather than direct extension of bleeding from the germinal matrix.

**Aetiology**
The major factor is prematurity
Others correlate to the degree of sickness and instability of the infant:
- Hypoxia
- Hypercapnia (or rapid changes in PCO₂)
- Acidosis; need for bicarbonate
- Hypothermia
- Unstable blood pressure; hypotension
- Maternal smoking
- Asphyxia
- Coagulopathy (this is also an important predisposing factor in the rare term baby with IVH)

**Prophylaxis**
- Stop prematurity!
- Antenatal steroids
- Scrupulous maintenance of physiological stability (blood gases, BP, clotting etc.)
- Minimal handling, gentle handling

**Clinical presentation**
- Majority of PIVH is clinically silent
- Acute hypovolaemia (rare)
- Neurological symptoms – apnoea, seizures, hypotonia, reduced level of consciousness, signs of raised ICP)

There is a very high mortality rate in babies presenting with shock or neurological signs due to PIVH.
Acute management of PIVH

Maintain normal physiological parameters (blood gases, BP, clotting etc.)
Treat hypovolaemia and neurological manifestations symptomatically
Repeat cranial ultrasound scan within 24-48 hours and then weekly in any baby with complicated PIVH
Measure OFC twice weekly in babies with PIVH complicated by ventricular dilatation
More frequent ultrasound scans and OFC measurement may be necessary in babies with rapidly progressing abnormalities.

Complications

- Intracranial hypertension – this occasionally occurs in babies with developing PHH and may be symptomatic with hypotonia, drowsiness, poor feeding and tense fontanelle
- Outcome of PHVD
  - ~ 1/3 normalise spontaneously
  - ~ 1/3 retain ventriculomegaly – likely to represent cerebral atrophy
  - ~ 1/3 require CSF diversion (shunt)
- Infants with IVH + dilatation and HPI are at risk of cerebral palsy. This is usually evident by 1 year, occasionally later, and may be of variable severity because of brain plasticity. There is also an increased risk of learning difficulties, which may vary in severity from almost indiscernible to very severe. The longer the delay before presentation, the less severe the neurodevelopmental impairment. Other less common complications include epilepsy, hearing impairment and impaired visual motor coordination.

Management of PHVD and hydrocephalus

- Indications for CSF removal are:
  Ventricular index >4mm over 97th centile for corrected gestational age in association with:
  - OFC with abnormal growth acceleration (i.e. increasing relative to centiles)
  - symptoms suggesting raised intracranial pressure (apnoea, vomiting, lethargy, seizures etc.)
- Always attempt lumbar puncture (LP) as initial choice for CSF removal, regardless of ultrasound evidence of CSF communication, as this is less invasive and may reduce the risk of iatrogenic brain injury. If LP unsuccessful, do ventricular tap.
- Always measure pressure prior to removing CSF by LP or ventricular tap
  - if pressure ≤6mmHg (8cmH2O), remove CSF for microbiological investigation only
  - if pressure >6mmHg, remove 10-15ml/kg CSF, send samples for microbiological investigation and protein concentration
- Repeat CSF removal as in indications above. Any baby requiring CSF removal more than once should be discussed with the neurosurgery team at KCH. Temporary measures to drain CSF via a reservoir may be considered in babies requiring very frequent CSF removal by LP or
ventricular tap prior to permanent CSF drainage via a shunt. The timing of shunt insertion may be determined by the baby’s general condition, weight and CSF protein concentration and cell count.

- CSF drainage from a reservoir should be carried out according to the baby’s symptoms or as directed by the neurosurgical team (see separate protocol)
- It is likely that permanent CSF diversion via a shunt is appropriate more for cosmetic and practical reasons than for improvement in neurodevelopmental outcome

Further Reading

- Management of posthaemorrhagic ventricular dilatation A Whitelaw, K Aquillina, Arch Dis Child Fetal Neonatal Ed doi:10.1136/adc.2010.190173
5.6 PERIVENTRICULAR LEUCOMALACIA

Definitions

PVL refers to injury of the cerebral white matter. It is generally more severe in the deep rather than the superficial white matter (WM). It consists of two major components

a) focal necrosis involving all cellular elements deep in the WM

b) diffuse component in the central WM with injury to premyelinating oligodendrocytes, marked astrocytosis and microgliosis

The focal necrotic lesions may evolve to cysts readily visualised on cranial ultrasound and occur in <5% of VLBW infants. More commonly the focal necrotic lesions are microscopic and evolve into small glial scars that may not easily be visualised by cranial ultrasound.

PVL may be manifested ultrasonographically by periventricular echogenicity (“flare”), which evolves into obvious cysts (usually multiple), but occasionally the “flare” remits and it is unclear whether this is genuine.

PVL is commonly accompanied by neuronal/axonal disease that Volpe terms “Encephalopathy of Prematurity” and consists of a complex amalgam of primary destructive and secondary developmental disturbances. This leads to the multifaceted developmental impairments that present in some preterm infants.

25-50% of very low birth weight (VLBW) babies that survive have subsequent cognitive, behavioural, attentional and socialization deficits and 5-10% major motor defects.

Coronal sections of cerebrum show focal necrotic and diffuse components of PVL. In cystic PVL (left) the focal necrotic lesions (circles) are macroscopic in size and evolve primarily to cysts, and in noncystic PVL, the focal necrotic lesions (dots) are microscopic in size and evolve primarily to glial scars. The diffuse component (shaded) is characterized by pre-oligodendrocyte injury, astrogliosis and microgliosis.
Abbreviations: SVZ, subventricular zone; GE, ganglionic eminence; T, Thalamus; P, putamen; GP, globus pallidus. (from Volpe 2009)

Pathogenesis
It has recently become clear that PVL is brought about by cytokine damage to the watershed areas of the brain (i.e. those areas functionally furthest from the cerebral blood supply and which are therefore first to show the damage brought about by impaired perfusion). The cytokine release is provoked most obviously by infection, intrauterine or postnatal. It is also brought about by hypoxia/ischaemia, but PVL is not primarily a hypoxic-ischaemic lesion.

Natural history
After a precipitating event (e.g. chorioamnionitis, cardiac arrest), periventricular echogenicity may be noted on ultrasound scan virtually immediately or up to a few days later. This flare may persist for a few days or up to about 14 days. Evolution to cysts may be seen from within a few days of the echogenicity up to 14 (rarely up to 28) days later - i.e. cystic PVL occurs within a few days to a few weeks after the precipitating event; a baby born with these appearances has suffered an intrauterine insult. The ultimate appearance is irregular dilatation of the cerebral ventricle(s) as the cysts collapse but there is no regeneration of the damaged brain tissue, and cerebral atrophy may be marked.

Clinical features
- clinically silent initially, although the baby may have signs of underlying cause
- hypotonicity then occurs, lasting until approximately term corrected age
- hypertonicity, possibly with irritability and poor feeding
- subsequent very high risk (>90%) of CP – most commonly spastic diplegia, moderate risk of learning difficulties and some risk of infantile spasms and visual impairment
- infants with subcortical leucomalacia are severely impaired with spastic tetraplegia, severe learning difficulties, cortical blindness and epilepsy. Early mortality is high.

Management
- There is no specific treatment available to reverse or reduce the extent of PVL
- Parents should be informed of the diagnosis by a senior doctor. Ensure documentation of all management decisions and parent discussions.
- Supportive care is important. Patients with PVL may benefit from multidisciplinary input and should be referred to the team of Neonatal therapists.

Further reading
- The encephalopathy of prematurity- Brain Injury and impaired Brain development Inextricably Intertwined, J Volpe, Seminars Ped Neurol. 2009; 16 (4)
5.7 INVESTIGATION OF HYPOTONIA

History

• Review maternal history – ? fetal movements, polyhydramnios, maternal myotonia (direct questions re. grip, screwing up eyes etc.), maternal myasthenia

• Review perinatal history – ? HIE, birth trauma – particularly cervical spine injury, history of respiratory problems, feeding problems etc.

Examination:

• Any dysmorphism

• Deep tendon reflexes

• Fasciculations (including tongue)

• Contractures

Investigations:

• If neuro-muscular problem is suspected, liaise with Paediatric Neurology team before arranging detailed investigations. These may include:
  - Cervical spine x-rays
  - Cranial U/S scan
  - MRI brain / spinal cord
  - CK (N.B. may be high for a few days after vaginal delivery)
  - DNA analysis for SMA, Congenital Myotonia (2 ml in EDTA bottle)
  - EMG
  - Nerve conduction study
  - Muscle biopsy
  - Myasthenia antibodies (baby and maternal)
  - Edrophonium test

• If metabolic disorder is suspected, liaise with Paediatric Metabolic team
  - Blood gas
  - Blood sugar
  - Serum lactate
  - Serum ammonia
  - Serum amino acids
  - Urine organic acids
  - Serum carnitine and acyl carnitines
  - CSF – lactate, glycine
• If Genetic disorder is suspected liaise with Clinical Genetics Department
  - DNA analysis for Prader-Willi Syndrome
  - Karyotype
• If Endocrine problem is suspected – TFTs (TSH +/- T₄)
5.8 CRANIAL ULTRASOUND SCANNING

The following babies should have a cranial ultrasound scan

Initial cranial ultrasound scan should be done within the first 12 hours if possible, particularly in babies admitted to NNU.

- < 32 weeks gestation or birth weight < 1500g
- Any baby with seizures or other neurological signs or symptoms (e.g. neonatal encephalopathy)
- Antenatally detected intra-cranial anomalies
- Babies with craniofacial anomalies or other syndromic features associated with intracranial anomalies
- Babies born to mothers known to use cocaine
- Suspected congenital infection (e.g. CMV, toxoplasmosis)
- Monochorionic twins
- Any baby with congenital cardiac disease

Schedule for routine cranial ultrasound scans in babies < 32 weeks gestation or birth weight < 1500g

- As soon as possible after stabilisation and ensuring clinical stability
- At 2-3 days or sooner if unstable
- At 7 days
- Then 2-4 weekly depending on the baby’s condition
- At term (i.e. 40 weeks corrected gestational age) or prior to discharge whichever comes first

N.B. further scans should be done:

- After any adverse event – e.g. major cardiovascular collapse, NEC etc.
- Depending on results of previous scans – schedule needs to be individualized if significant abnormalities are detected

All scans should be carried out under the supervision of an experienced SpR or consultant. All scans will be reviewed by the attending consultant on the ward rounds to finalise the report. Any scan with major abnormalities should also be reviewed at the monthly radiology meeting, or sooner if necessary by a consultant radiologist. The results of the cranial ultrasound scans should be entered in the Badger/SEND database

Images to save

- Coronal view through frontal horns
- Coronal view through 3rd ventricle
- Coronal view through posterior horns
- Mid line sagittal
- Left and right parasagittal through lateral ventricles
- Left and right parasagittal view of parenchyma lateral to the lateral ventricles
- Any abnormality

If any abnormality is found, a hard copy should be printed and filed in the correct part of the in-patient hospital notes. Write a provisional report in the correct sheet in the in-patient hospital notes. Ensure images are saved on PACS.

Cranial Ultrasound scan findings and prediction of cerebral palsy

For indication we provide the results of the NEURO cohort of the SUPPORT trial of a cohort of infants <28 weeks gestation

<table>
<thead>
<tr>
<th>Predictive associations of CUS findings with 18-22 mo outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>CUS finding</td>
</tr>
<tr>
<td>Cognitive&lt;70</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Mod/severe cerebral palsy</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Death or neurodevelopmental impairment</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Unimpaired</td>
</tr>
</tbody>
</table>

Early adverse cranial ultrasound (CUS): IVH grade 3/4 or PVL; Late adverse CUS: cystic PVL, porencephalic cyst, moderate-severe ventriculomegaly or shunt

Further reading:
5.9 AMPLITUDE INTEGRATED EEG (aEEG)

Amplitude integrated EEG is a method of continuous monitoring of brain function. The method is based on filtered and compressed EEG that enables evaluation of long-term changes and trends in electrocortical background activity by relatively simple pattern recognition.

The signal is written out in slow speed (6cm/h) and as it is compressed may not reveal easily, acute changes such as short seizures. It is important to interrogate the underlying EEG as well as the aEEG trace to exclude presence of seizure activity.

We use the 2 channel “BRAINZ” aEEG (a guide to applying the device is attached to the device). This will display two waveforms, one for each hemisphere enabling detection of unilateral changes.

We also use the NICOLET aEEG monitor (a guide to applying the device is attached to the device) which can monitor 2-10 channels

Interpretation
It is important to record the following when reporting an aEEG trace:

a) The background pattern:
   - Continuous normal voltage:
     - Upper margin > 10 μV (usually 10-50μV)
     - Lower margin > 5 μV (usually 5-7μV)
     - Discontinuous normal voltage:
     - Upper margin > 10 μV
     - Lower margin < 5 μV
   - Burst suppression (BS): discontinuous background with
     - Lower margin without variability 0-2 μV and bursts >25 μV
     - BS+: burst density > 100 bursts/hour
     - BS-: burst density <100 bursts/hour
   - Low voltage (LV)
     - Continuous background pattern of very low voltage (around or below 5μV)
   - Inactive, flat (FT)
     - Primarily inactive (isoelectric) background<5μV

b) Presence of Sleep-wake cycling:
   - Normal term babies should exhibit sleep-wake cycling on aEEG consisting of smooth sinusoidal variations.
• Broader bandwidth represents activity during quiet sleep
• Narrow bandwidth represents wakefulness or active sleep

c) Presence of Seizures:
• Single seizures: a solitary seizure
• Repetitive seizures: Single seizures appearing more frequently than at 30 minute intervals
• Status epilepticus: continuous ongoing seizure activity for ≥ 30 minutes

Further reading:
• Amplitude-integrated EEG Classification and Interpretation in Preterm and Term Infants
  Hellström-Westas, Neoreviews 2006
5.10 OPHTHALMOLOGICAL ASSESSMENT

Ophthalmologic assessments are carried out by the Paediatric Ophthalmology team. In order to detect retinopathy of prematurity (ROP) in premature babies, other pathological changes or congenital abnormalities at the earliest opportunity, babies with the following problems should have an ophthalmological assessment:

- Prematurity - gestational age <32 weeks or birth weight <1500g.
- Congenital infection (TORCH).
- Congenital abnormalities of the eye or face.
- Disease associated with eye problems, e.g. Sturge-Weber, candida septicaemia.
- Concern about vision.

Schedule

- For ROP screening, the age for the first examination depends on the gestational age at birth (see table)

<table>
<thead>
<tr>
<th>Gestational age</th>
<th>Age of first examination</th>
<th>Corrected Gest. Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>23 weeks</td>
<td>7 weeks</td>
<td>30 weeks</td>
</tr>
<tr>
<td>24 weeks</td>
<td>6 weeks</td>
<td>30 weeks</td>
</tr>
<tr>
<td>25 weeks</td>
<td>5 weeks</td>
<td>30 weeks</td>
</tr>
<tr>
<td>&gt;26 weeks</td>
<td>First exam at 4 weeks of age</td>
<td></td>
</tr>
</tbody>
</table>

- In order to remind medical staff which babies are due for screening, a diary is kept at the SCBU Nursing Station. Any baby requiring ophthalmologic examination should be entered onto the appropriate page in advance.
- The ophthalmologists will decide scheduling of subsequent examinations.
- If a baby requires their 1st ROP screening examination after discharge is planned, the Neonatal Outreach Team will arrange a Tuesday morning outpatient appointment on the Neonatal Unit. Out-patients should arrive in the Neonatal Unit reception for eye drops to be instilled 1 hour before examination.
- Babies needing long term outpatient ophthalmology follow-up should be seen in Paediatric Ophthalmology outpatient clinic
- For inpatients, ophthalmological examinations will routinely be done weekly on Tuesday mornings, unless otherwise arranged by the ophthalmologists.

Procedure

- From one hour before examination (i.e. from 1pm) the following should be instilled:
  - Phenylephrine 2.5% - 1 drop in each eye every 15 minutes for 3 doses
- Cyclopentolate 0.5% - 1 drop in each eye every 15 minutes for 3 doses
- Occasionally, the pupils fail to dilate sufficiently using the above drops. If this occurs and if directed by the ophthalmology team, for subsequent examinations use Atropine 1% - 1 drop in each eye at 6am and 6pm for 48 hours prior to examination

- Results of the examination will be recorded on the special green ophthalmology sheet and kept in the baby's notes.
- Follow-up should be arranged as indicated by the ophthalmologists.
- Parents should be given an information sheet on screening for ROP if they require screening. They may also require an information sheet on severe ROP and treatment of ROP if necessary.
- The result of the eye examination should be entered in the relevant section on the Badger/SEND database

Further Reading
- Current update on retinopathy of prematurity: screening and treatment. Chen J, Curr Opin Pediatr, 2011 Apr;23(2)
5.11 AUDIOLOGICAL ASSESSMENT

- Moderate to severe hearing loss = >30-40dB loss at 500-4000Hz in better ear. This degree of hearing loss impairs normal conversation.
- Incidence – 1-3/1000 live births.
- Incidence in high risk group – 2-4/100
- High risk group contribute 50% of all cases of hearing loss
- If diagnosis and intervention in cases of hearing loss is delayed until >6months, there is usually delay in language, cognitive and emotional development

Automated auditory brainstem responses (AABR)

- Test whole auditory pathway
- Are reliable in the first 24 hours of life
- Have 98% concordance with standard auditory brainstem responses (ABR)

Universal Neonatal Hearing Screening (UNHS)

All babies are screened soon after birth by oto-acoustic emissions (OAE), which is an easier and quicker test than AABR but one with a higher rate of false positive results. Those failing twice will then have an AABR. In Guy’s and St Thomas’ this will all take place on the postnatal wards (home deliveries will be referred in) 7 days a week by a dedicated, trained screener. Babies on SCBU will be screened in the week prior to discharge.

Babies at increased risk of hearing loss

- Parental consanguinity
- Family history of permanent childhood hearing impairment
- Syndrome known to include hearing impairment
- Dysmorphology of head, neck or ear (including pits and skin tags)
- Clinical suspicion of hearing impairment
- NICU for 48hrs, therefore including:
  - prematurity <32weeks and/or birthweight <1500g
  - IVH grade 3 and/or parenchymal infarction
  - PVL
  - hypoxic-ischaemic encephalopathy (moderate to severe)
  - bacterial meningitis
  - congenital infection
  - hydrocephalus
  - hyperbilirubinaemia (>=exchange level)
aminoglycosides – toxic levels (especially + furosemide)

NB Babies presenting with severe jaundice at term may have already had UNHS and will require a repeat screen or referral to Audiology

Please enter the result of the Hearing screen in the Badger / SEND database