



Title:	Guidelines for Management of the Acute Collapsed Neonate/Infant	
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Supersedes:	N/A	
Application:	ation: The guideline is intended for use by any hospital team caring for neonates and infants	
	across the Paediatric Critical Care Network in the North West & North Wales region.	

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Responsibility of:	Clinical lead North West & North Wales Paediatric Critical Care Network &
	NWTS guideline lead consultant & lead nurse

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EqIA Registration Number:	2021-16	





# 1. Detail of Procedural Document

Guidelines for Management of Guidelines for Management of the Acute Collapsed Neonate/Infant This guideline is for use by staff working in the District General Hospitals of the North West (England) and North Wales region and NWTS team to use when caring for an acutely collapsed neonate or infant. It focuses on acute management and diagnostic categories that need to be considered.

This does not replace an acute referral to NWTS team for advice on management, but is designed to help both NWTS and the referring team throughout the acute stabilisation period.

# 2. Equality Impact Assessment

EqIA registration Number for RMCH: 2021-16
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# 3. Consultation, Approval and Ratification Process

This guideline was developed with input from:

- North West (England) and North Wales Paediatric Transport Service (NWTS) medical & nursing
- Representatives from the District General Hospitals within the North West (England) & North Wales Paediatric Critical Care operational delivery network; includes medical, nursing and AHP (paediatrics, anaesthetics, and emergency medicine teams)
- Representatives from both Paediatric Critical Care Units (Royal Manchester Children's Hospital and Alder Hey Children's Hospital) medical and nursing

These guidelines were circulated amongst the North West and North Wales Paediatric Critical Care Network for comments on 20.07.2020

All comments received have been reviewed and appropriate amendments incorporated.

These guidelines were signed off by the Network's Joint Clinical Leads

For ratification process for network guidelines see appendix 1.

# 4. Disclaimer

These clinical guidelines represent the views of the North West (England) and North Wales Paediatric Critical Care Transport Service (NWTS) and the North West and North Wales Paediatric Critical Care Operational Delivery Network (PCCN). They have been produced after careful consideration of available evidence in conjunction with clinical expertise and experience.

It is intended that trusts within the Network will adopt this guideline and educational resource after review and ratification (including equality impact assessment) through their own clinical governance structures.

# The guidance does not override the individual responsibility of healthcare professionals to make

# decisions appropriate to the circumstances of the individual patient.

Clinical advice is always available from NWTS on a case by case basis.

Please feel free to contact NWTS (01925 853 550) regarding these documents if there are any queries





# **GENERAL MANAGEMENT**

RESUSCITATION: for details see below Structured ABCDE assessment High flow oxygen, IV/IO access – send investigations 10-20mL/kg crystalloid fluid bolus if features of shock

**IMMEDIATE INVESTIGATIONS:** FBC, U&Es, LFTs, CRP, Ca<sup>2+</sup>, Mg<sup>2+</sup>, clotting, blood gas + lactate, glucose, group & save. Ammonia. Cultures: blood, urine + respiratory secretions. CXR +/- AXR; consider ECG if tachycardic.

# **EVIDENCE OF CARDIAC DISEASE?** See page 5-6

<u>Duct-dependent condition?</u> - Pre/post ductal SpO<sub>2</sub> discrepancy, cyanosis in FiO<sub>2</sub> 1.0, abnormal femoral pulses or 4 limb BP, cardiac murmur?

CXR: cardiomegaly; metabolic acidosis/raised lactate <u>Heart failure</u>?: gallop rhythm, hepatomegaly, peripheral oedema, FTT

# STILL SHOCKED AFTER 20mL/KG FLUID?

Further 10-20mL/kg crystalloid fluid bolus URGENT senior paediatric + anaesthetic review

# EVIDENCE OF SEIZURES / ENCEPHALOPATHY?

Convulsions, unexplained episodes of tachycardia/ hypertension, apnoeas, drowsy or irritable child, vomiting, cycling movements, lip smacking, other subtle movements

### STILL SHOCKED AFTER 40mL/KG FLUID?

Further 10-20 mL/kg bolus if no evidence fluid overload (hepatomegaly, gallop rhythm) Consider using blood products if appropriate Start inotrope infusion via IO/PVL: **NWTS sepsis guideline** Central/arterial lines: if less 1 week old consider umbilical venous and arterial access

Urinary catheterisation

Intubation triggers: see NWTS intubation guidelines Respiratory failure, impending CVS collapse, fluid refractory shock, reduced/fluctuating GCS, ↑ammonia.

## Suggested induction: ketamine, fentanyl + NMB<sup>7</sup>.

Have **dilute adrenaline** (take 0.1 mL/kg from Minijet 1:10,000 adrenaline + make this up to 10mL) ready even if not shocked. If shocked: start inotrope infusion before intubation.

# POSSIBLE SURGICAL CAUSE?

Abdominal distension, bilious vomits/aspirates, high lactate, history of abnormal stools +/- blood in stools

# **ELECTROLYTE ABNORMALITIES?**

lonised calcium <1?Correct with calcium gluconate as per crashcall.net. Consider giving sodium bicarbonate if pH <7 and not improving with fluid bolus and inotropic support

# FLUID/INOTROPE RESISTANT SHOCK?

Add **adrenaline** if peripheral pulses are poor Add **noradrenaline** if peripheral pulses are bounding Add **hydrocortisone** 1mg/kg if requiring >2 inotropes

# **SPECIFIC CAUSES**

SEPSIS: page 5 & NWTS sepsis guidelines Broad spectrum antimicrobials: cefotaxime, amoxicillin & high dose aciclovir<sup>3</sup>

High risk HSV if contact or rash, seizures, hypoglycaemia, abnormal LFTs, coagulopathy, encephalopathy Consider adding antibiotics for specific suspected infections

## DUCT DEPENDENT SYSTEMIC or PULMONARY CIRCULATION:

Consider limiting  $FiO_2$  + aim lower  $SpO_2$  e.g. 75-85% Start **Dinoprostone** (Prostin) at 50-100 nanogram/kg/minute<sup>4</sup> High doses required to re-open duct in sick neonate. Beware apnoeas & hypotension: doses higher than 10 nanogram/kg/minute – need I&V

### NON-DUCT DEPENDENT CARDIAC CONDITION:

Evidence arrhythmia on ECG – treat as per APLS  $^5$ Evidence heart failure: cautious fluid resus, stop if liver $\uparrow$ . Discuss with cardiology. Urgent ECHO if available in DGH

### METABOLIC: page 6-7

Check: blood gas, glucose, ammonia and ketones sent. Treat ↓glucose (**2mL/kg 10% glucose**), stop feeds + start 10% glucose-containing maintenance fluids. If ammonia >150 but <200 repeat ammonia urgently

If >200 at any point: repeat ammonia, + do not wait for result – call NWTS ASAP

See NWTS guidelines: Management of hyperammonaemia<sup>6</sup>

### **INTRACRANIAL BLEED / TRAUMA: page 8**

Signs: focal neurology, bulging AF, anaemia, bruising or other injuries, ↑OFC (check baseline), unequal or unreactive pupils If baby did not receive **vitamin K** at birth give 1mg IV<sup>4</sup> URGENT CT head if stable: if evidence bleed contact ChMTC & TTL (AHCH 0151 252 5401; RMCH 0161 701 9191) Organise time critical transfer (**use STOPP document**) **Initiate safeguarding procedures** 

POISONING: page 9 Consider in any unexplained collapse Take 10mL urine for toxicology studies ASAP Treat known poisoning as per TOXBASE advice Initiate Safeguarding procedures

### SURGICAL: page 9

Ensure NBM and NGT on free drainage. Check abdo x-ray Consider metronidazole.

Discuss with tertiary paediatric surgeon and share x-rays via PACS. May need time critical transfer to tertiary centre.

#### ENDOCRINE: page 8 Examine genitalia.

?Congenital adrenal hyperplasia: hypoglycaemia, hyponatraemia, hyperkalaemia, shock. Check cortisol + 17-OHP

Isolated hyponatraemia? Consider ingestion dilute feeds/ excess water

?Adrenal crisis: consider IV hydrocortisone 1mg/kg Check blood for random cortisol

?Difficulty maintaining normal glucose, send blood for hypoglycaemia screen, increase maintenance infusion glucose





# INTRODUCTION

Although collapse of a neonate (aged  $\leq$  44 weeks post gestational age) or infant is rare, it is associated with significant morbidity and mortality, and can cause severe long term neurodevelopmental sequelae<sup>1</sup>. Treatment targeted at the cause is important, but the cause is often not clear at presentation, and general supportive measures are important to improve outcomes. Please see algorithm for a stepwise management plan, and refer to Crashcall.net or the BNFc for drug dosages.

Signs and symptoms of neonatal illness can be vague and non-specific, and include:

- Respiratory distress including grunting
- Hypoxia
- Apnoeas
- Poor feeding
- Tachycardia
- Poor volume or difficult to feel pulses
- Prolonged capillary refill
- Mottling
- Temperature instability (i.e. high or low temperature)
- Reduced responsiveness
- Seizures
- Low blood pressure (late finding)

The main categories of neonatal collapse are:

- Sepsis
- Cardiac disease
- Metabolic disease or endocrine disorders
- Safeguarding Non accidental injury (NAI) / abusive head trauma / poisoning
- Abdominal surgical conditions

Important features to include in the history taking are:

- Exact circumstances surrounding collapse (including position of baby, persons present, timeline of events)
- Antenatal and birth history including risk factors for sepsis
- Family history, including history of congenital cardiac disease or unexplained sudden deaths
- Feeding history
- Drugs/medications present in the household
- Social history including any safeguarding red flags

The initial assessment of a collapsed neonate should follow a structured ABCDE approach. The signs and symptoms, investigations and management of each category are discussed below. However it is important to note that conditions can co-exist (for example sepsis AND metabolic disease). Always consider non-accidental injury within the differential diagnosis, particularly if history is inconsistent with the clinical presentation or there is no history

### **HYPOTHERMIA in ACUTELY SICK NEONATE / INFANT**

Hypothermia at presentation is associated with a worse outcome.

Common metabolic consequences hypothermia

- **Hypoglycaemia:** due to an increase in metabolic rate to produce heat which may rapidly use up glucose stores leading to hypoglycaemia.
- Hypoxia: secondary to a leftward shift in the oxyhaemoglobin dissociation curve, i.e. increased haemoglobin affinity
  for oxygen, impedes oxygen delivery to tissues. Also an increased metabolic rate increases oxygen needs, and aggravates any hypoxia
- Metabolic acidosis: anaerobic metabolism of glucose to produce heat causes an increase in lactic acid production.
- Coagulopathy: Disseminated intravascular coagulopathy is common in marked hypothermia

### Management

To reduce heat loss and avoiding hypothermia, use a woollen hat, socks and blankets. If intubated consider covering with a plastic sheet or bubble wrap, and using Mediwrap blanket or similar.

More advanced measures include using an overhead radiant warmer, warming blanket, or closed incubator (set at 37 °C)





# SEPSIS: See NWTS Sepsis Guidelines for management via www.nwts.nhs.uk

Sepsis is the leading cause of neonatal collapse. It is important to check for any risk factors<sup>2</sup>, such as:

- Maternal group B streptococcus (GBS) colonisation
- Previous sibling with invasive GBS disease
- Prolonged rupture of membranes (>24 hours prior to delivery if term, >18 hours if preterm)
- Maternal infection e.g. sepsis, chorioamnionitis, genital HSV
- Missed maternal antenatal influenza and pertussis immunisations

The absence of risk factors for neonatal sepsis, or the absence of raised inflammatory markers does not exclude sepsis. <u>ALL</u> babies presenting with collapse should be treated with broad spectrum antibiotics. The suggested initial combination is Cefotaxime, amoxicillin and aciclovir<sup>3</sup> to cover Group B streptococcus, E.Coli, Listeria and HSV. Please also refer to local antimicrobial guidelines.

Disseminated Herpes simplex can present in the same way as bacterial sepsis, and is associated with high mortality. High dose aciclovir should be included with broad spectrum antimicrobials, especially if there is a contact with known HSV infection, or evidence of liver dysfunction, hypoglycaemia, coagulopathy (including excessive bleeding from venepuncture sites) or rash. Disseminated HSV has been seen in those with no risk factors and following an elective LSCS. Blood samples for viral PCR testing should be sent as soon as possible to allow confirmation of diagnosis and prognostication.

Investigations:

- Blood and urine cultures should be sent for every neonate.
- Congenital pneumonia can present with sepsis without isolated chest findings, so a chest x-ray should be considered.
- Lumbar puncture should be considered only once the baby is stable, as long as there are no contraindications
  (cardiorespiratory instability, focal neurology, apnoeas, seizures, coagulopathy, suspected trauma or raised intracranial
  pressure). It should not delay resuscitation and treatment with appropriate antimicrobials. Viral CSF PCRs should be
  sent, in addition to microscopy, culture, protein, and paired CSF: serum glucose and lactate.
- Respiratory secretions are required to investigate for respiratory viral screen, COVID-19 or Pertussis.

As well as broad spectrum antimicrobials, additional targeted treatment may be needed if specific infections are suspected:

- Gentamicin if child is critically unwell, or if suspected urinary tract infection
- Clindamycin if concerns about invasive group A streptococcus, or Staph aureus infections (eg concurrent or recent Chicken pox)
- Macrolide if concerns about pertussis
- Metronidazole if surgical cause / intra-abdominal sepsis suspected

### CARDIAC DISEASE

Structural or congenital heart disease can be split into many different categories. For the purposes of this guideline they have been categorised as either pink, blue or grey. Both blue or grey lesions are duct dependent and require dinoprostone infusion. Echocardiogram may be available (paediatric or neonatal trainee / consultant) at the local hospital but treatment should not be delayed if echocardiogram is not available or delayed.

### DUCT DEPENDENT IE BLUE OR GREY CARDIAC LESIONS

These are conditions that rely on circulation through a patent ductus arteriosus for maintenance of either pulmonary or systemic blood flow. Once the duct begins to close the child will rapidly become unwell. These conditions include:

PINK: Congestive cardiac failure	BLUE: Persistent cyanosis	GREY: Low cardiac output
Increased pulmonary blood flow May present with difficulty feeding, FTT, recurrent LRTI, tachypnoea +/- increased WOB, hepatomegaly, gallop rhythm	Obstructed pulmonary outflow tract Or inadequate mixing	Obstructed systemic outflow tract—absent or weak femoral pulses
Increased shunt eg VSD, ASD, complete AVSD, PDA, Total anomalous pulmonary venous drainage (TAPVD)	Pulmonary atresia Critical pulmonary stenosis Tricuspid atresia Tetralogy of Fallot Transposition of Great Arteries with or without intact septum	Coarctation of aorta Critical Aortic Stenosis Hypoplastic left heart syndrome Interrupted aortic arch





# CARDIAC (continued)

If a duct-dependent cardiac lesion is suspected start Dinoprostone infusion (5-100 nanograms/kg/minute)<sup>4</sup>. ALWAYS ensure dedicated IV access for the infusion which should not be interrupted or flushed.

If the baby is well and non-acidotic (eg immediately post-partum with an ante-natal diagnosis of duct dependent congenital heart disease) start Dinoprostone at 5-10 nanogram/kg/min, as the duct is presumed not to have fully closed (d/w tertiary paediatric cardiologist). NB echocardiogram is needed to confirm whether ductus arteriosus is patent

Any dose above 5-10 nanograms/kg/minute may cause apnoea and therefore a period of observation (1-2 hrs) is advised before transfer. If the baby is having recurrent apnoeas, then intubation and ventilation for transfer is strongly advised.

If the baby is unwell or acidotic this implies that duct has closed, and start Dinoprostone at 50-100 nanograms/kg/minute to re-open the duct. If no improvement after approximately one hour at 50 nanograms/kg/minute or the baby remains in extremis, increase to 100 nanograms/kg/minute.

If starting Dinoprostone at higher infusion doses, it is essential to intubate and ventilate.

Be aware that higher doses of Dinoprostone may also cause hypotension requiring fluid resuscitation (in judicious aliquots e.g. 5mL/kg) and other vasoactive drugs e.g. adrenaline.

If the baby does not improve clinically or deteriorates despite maximum Dinoprostone infusion, then the duct may not have reopened, or diagnosis may be obstructed total anomalous pulmonary venous drainage . In either case the baby needs a time critical transfer to cardiac surgical centre by an experienced team e.g. NWTS or CONNECT NW.

Aims of treatment:

- Palpable femoral pulses
- Resolving acidosis
- Aim for oxygen saturations 75-85% for those presenting with cyanosis, titrate supplemental oxygen down to prevent over-oxygenation. Many duct dependent lesions have a degree of mixing, therefore SpO<sub>2</sub> 75-85% is appropriate.
- Caution: some duct dependent lesions are 'balanced circulations' whereby a single ventricle provides blood flow to both the pulmonary and systemic circulations. Increasing inspired oxygen concentrations may increase saturations to 90-100% but at the expense of pulmonary over-circulation and resultant poor systemic circulation with hypotension, poor peripheral perfusion / pulses and worsening lactic acidosis.
- Aim for normalisation of blood gases including lactate

#### **NON-DUCT DEPENDENT LESIONS**

Non-duct dependent structural cardiac lesions typically do not present in the first few days of life, but may present in the neonatal or early infancy period. Cardiomyopathy or myocarditis may present at any time.

If any evidence of heart failure (hepatomegaly, gallop rhythm) then fluid resuscitation should be done with cautiously (i.e. 5mL/kg aliquots), and avoided if increasing liver size. Discussion with NWTS and tertiary cardiac centre (via NWTS conference call) regarding further management is required.

#### ARRHYTHMIAS

Babies may present with either acute shock or heart failure due to arrhythmia. Manage as per APLS protocols<sup>5</sup> and print out rhythm strips and 12-lead ECG. Discuss with the local tertiary cardiac centre and NWTS for on-going management. Be aware that arrhythmias may co-exist with structural heart lesions e.g. Ebstein's anomaly. Keep magnesium, ionised calcium (check blood gas) and potassium levels are kept at the upper end of normal range, which may require calcium, magnesium and potassium supplementation.

Refer to NWTS arrhythmia guidelines.

### METABOLIC DISEASE & ENDOCRINE DISORDERS

Metabolic disease can present in the neonatal period with lethargy, coma, metabolic acidosis or alkalosis, and can be indistinguishable from sepsis. Sepsis can also cause metabolic derangement that can mimic metabolic disease, causing hypoglycaemia or hyperammonaemia. Many metabolic conditions are tested for on routine neonatal heel prick screening, but may present before the results are available.

#### Hyperammonaemia

Hyperammonaemia is an acute life-threatening condition that can lead to severe neurological impairment and cerebral oedema. All hospitals should have easy access on site to intravenous drugs used to treat hyperammonaemia ie Sodium Benzoate, Sodium Phenylbutyrate, L-Arginine and L-Carnitine.





# METABOLIC DISEASE & ENDOCRINE DISORDERS (continued)

Check plasma ammonia level on all patients presenting with acute collapse, abnormal neurology or behaviour. It is essential to ensure samples are taken in the correct bottle, handled appropriately (usually transported on ice and ideally arrive at the laboratory within 15 minutes of collection) and that the local biochemistry lab are contacted to ensure a result is available within 1-2 hours.

Hyperammonaemia is a medical emergency: follow NWTS guidelines on Management of Hyperammonaemia<sup>6</sup>.

- FIRST AMMONIA LEVEL IS GREATER than 150 micromol/L, but less than 200 micromol/L, send repeat ammonia urgently.
- Stop any enteral feeds / protein load and start IV fluids containing 10% glucose to drive an anabolic state.
- Ensure have 2<sup>nd</sup> peripheral IV access to use for metabolic drug infusions.
- Send investigations listed below.
- If 2<sup>nd</sup> ammonia result is less than 200 micromol/L, discuss with metabolic consultant +/- NWTS if clinically necessary.
- **FIRST AMMONIA IS GREATER than 200micromol/L**, send repeat sample immediately but escalate treatment without waiting for result.
- Contact NWTS who will organise a conference call with a metabolic consultant.
- Start treatment agreed with metabolic consultant within 30 minutes of the discussion.
- NB Hyperammonaemia causes cerebral oedema
- Urgent anaesthetic review and intubation and ventilation is required before transfer to tertiary unit.

Any delay in starting metabolic drug treatment and intubation and ventilation with neuroprotective measures, increases the risk of permanent neurological injury.

Investigations to perform:

- Liver function tests and clotting
- Glucose
- Ketones— both urine dipstick and point of care capillary blood test
- Blood gas and lactate
- Acylcarnitines
- Plasma amino acids
- Urine amino and organic acids can be transferred with patient to PICU

### OTHER METABOLIC DISORDERS

If a neonate is found to have E.Coli sepsis, consideration should be given to an underlying diagnosis of galactosaemia. Send a blood sample for Gal-1-PUT activity is recommended (more specific than urine reducing substances).

A persistent unexplained metabolic acidosis (particularly with a high anion gap) or high lactate should also prompt discussion with metabolic team.

### HYPOGLYCAEMIA

Hypoglycaemia (defined as blood sugar <2.6 mmol/L) can be caused by a variety of pathologies, including reduced feeding in the hours prior to collapse, sepsis, liver dysfunction, endocrine dysfunction, or metabolic disease.

Upon identification of low blood sugar, send a hypoglycaemia screen prior to treatment. Administer 2mL/kg 10% glucose IV bolus, and repeat blood sugar in 15 minutes to check for resolution.

In an emergency (if difficulty obtaining IV access) consider giving GlucoGel (fast acting glucose gel via buccal mucosa) or IM glucagon (see BNFc). Ensure child is placed on IV maintenance fluids containing 10% glucose (for example 0.9% sodium chloride + 10% glucose). Calculate how much glucose (mg/kg/minute) is required to maintain normoglycaemia (normal range: 6-8 mg/ kg/minute). Hypoglycaemia is severe if it persists despite infusion of >10 mg/kg/min.

Glucose intake (mg/kg/min) = <u>% glucose x hourly rate</u>

Weight (kg) x 6

Hypoglycaemia screen should include, in order of priority:

- Lab glucose
- Bedside ketones (using ketone meter), blood gas and lactate
- U&Es, LFTs, ammonia
- Insulin & C-peptide
- 3-OH butyrate, free fatty acids
- Cortisol, growth hormone, thyroid function
- Acyclcarnitines (blood spot sample), plasma amino acids
- Urine organic and amino acids
- 17-OHP (17-Hydroxyprogesterone)

The child may require discussion with metabolic or endocrine teams if hypoglycaemia is recurrent, or above results abnormal. All non-ketotic hypoglycaemia should be discussed with the tertiary teams.



# ENDOCRINE DISORDERS

North Wale

Neonates may present with abnormal electrolytes for a variety of reasons, but endocrine disorders must be considered.

Congenital adrenal hyperplasia (CAH) may present as acute collapse in the neonatal period, with hypoglycaemia, hyponatraemia, hyperkalaemia and shock. Ambiguous genitalia may or may not be present. If any concerns regarding CAH then cortisol and 17-OHP levels should be taken prior to administration IV hydrocortisone 1mg/kg.

One differential diagnosis for electrolyte abnormalities should include the administration of diluted feeds to the baby. This may be due to miseducation regarding the making up of formula feeds, or deliberate dilution of the feeds due to financial issues. Some babies are given water to drink when they are perceived to be unwell, for example with vomiting, and this can cause hyponatraemia and collapse. In addition to clinically treating the hyponatraemia, it is important to trigger safeguarding investigations, as part of the review of potential causes.

For more information on management of electrolyte disturbances – see NWTS / PCCN guidelines.

# SAFEGUARDING

Non-accidental injury and poisoning are important causes of neonatal / infant collapse and should always be considered.

### **INTRACRANIAL BLEED / TRAUMA**

Neonates and infants are at high risk of intracranial bleeds associated with trauma, due to the relatively large head and fragile bridging vessels between dural spaces.

NB this can be a cause of major haemorrhage/shock due to size of head/expanding fontanelles, and appropriate resuscitation including blood products may be required.

Causes of severe intracranial bleeds:

- Abusive head trauma: there may be no other external signs of trauma, but findings of bruising elsewhere, or rib, skull or other bone fractures will increase suspicion.
- Non-traumatic bleeds: associated with congenital vascular malformations, meningitis, glutaric aciduria type 1, or vitamin K deficiency/haemorrhagic disease of the newborn.
- Accidental trauma: it is important to take a thorough history from any family members/carers present.

### Signs of possible intracranial bleeds:

- Reduced GCS / drowsiness / irritability / vomiting
- Seizures may be focal or generalised
- Focal neurology
- Bulging fontanelle
- Unexplained bradycardia / hypertension
- Unexplained anaemia

NB an intracranial bleed can be a cause of major haemorrhage/shock due to size of head/expanding fontanelles.

Management:

- Urgent CT head (after appropriate resuscitation including intubation & ventilation if required)
- An intracranial bleed is a time critical neurosurgical emergency. ALWAYS refer via trauma team leader
- Alder Hey TTL: 0151 252 5401; RMCH TTL: 0161 701 9191
- Discuss with trauma team leader (TTL) and organise an immediate time critical transfer to one of the children's major trauma centre (ChMTC). Transfer to be undertaken by the local team, use the STOPP (safe transfer of paediatric patients) document available on the NWTS website for all transfers plus NWTS transfer resources (see page 9).
- Seizures should be managed as per APLS / NWTS seizure guidelines.<sup>5</sup>
- If evidence of raised intracranial pressure 3-5mL/kg hypertonic (2.7-5%) sodium chloride should be given; this can be repeated as required.
- In addition, consider intubation and ventilation with neuroprotective measures
  - Maintain SpO<sub>2</sub>>96%
  - ETCO<sub>2</sub> 4-4.5kPa
  - Maintain age appropriate blood pressure ie mean BP 45-50 (with inotropes as needed via PVL/IO) Ensure adequate sedation
- If the baby did not receive vitamin K at birth, give 1mg IV immediately<sup>4</sup>.

Safeguarding procedures should be initiated by the local paediatric team, and all documentation copied and transferred with the baby to the tertiary unit. This includes contacting the local social services emergency duty team, and/or police and ensuring any siblings are safeguarded.

See https://nwchildrenstrauma.nhs.uk/clinical-guidelines





In a neonate / infant with unexplained collapse, poisoning must be considered. Send urine for toxicology as soon as possible. Ensure any drugs that may have been given in hospital or are reported to be available in the household are documented in the notes and on the test request (e.g. antibiotics or ketamine and fentanyl for RSI) to avoid any confusion with results. NB it is advisable to send a chain of custody form with urine toxicology samples if there are safeguarding concerns. If poisoning has occurred with a known agent, use Toxbase to guide management.

Initiate safeguarding procedures in the local hospital, and all documentation should be copied and transferred with the baby to the tertiary unit. This includes contacting the local children's services emergency duty team, and/or police.

# **SURGICAL**

Bowel obstruction can lead to severe illness and a collapsed presentation due to acute fluid losses and bowel wall oedema. Translocation of bacteria across compromised bowel wall can lead to sepsis, and all babies should receive broad spectrum antibiotics with consideration of metronidazole and/or gentamicin (always check renal function). Abdominal compartment syndrome is a rare complication but can cause compression of blood vessels, ischaemia of intraabdominal organs, and loss of perfusion to lower limbs.

Ensure the baby is nil by mouth and has a nasogastric tube inserted which is on free drainage and regularly aspirated every 1-2 hours. Perform an abdominal x-ray and check blood gas, lactate and electrolytes. These children may have profound fluid and electrolyte losses.

Early discussion with the surgical registrar at tertiary paediatric surgical centre is crucial. Images should be shared via PACS, but if this is not possible then all images should be transferred with the child on CD. The neonatal acute surgical abdomen is a time critical emergency. Responsibility for transfer will usually rest with the local hospital with support and advice from NWTS as required. Delayed transfers may lead to loss of gut and an increased mortality risk.

Potential surgical causes of collapse include:

- Necrotising enterocolitis (even in term babies)
- Malrotation and volvulus
- Hirschsprung's disease
- Meconium ileus
- Bowel atresia or hypoplasia
- Congenital diaphragmatic hernia
- Imperforate anus

# TRANSFER RESOURCES FOR ALL CAUSES OF NEONATAL COLLAPSE

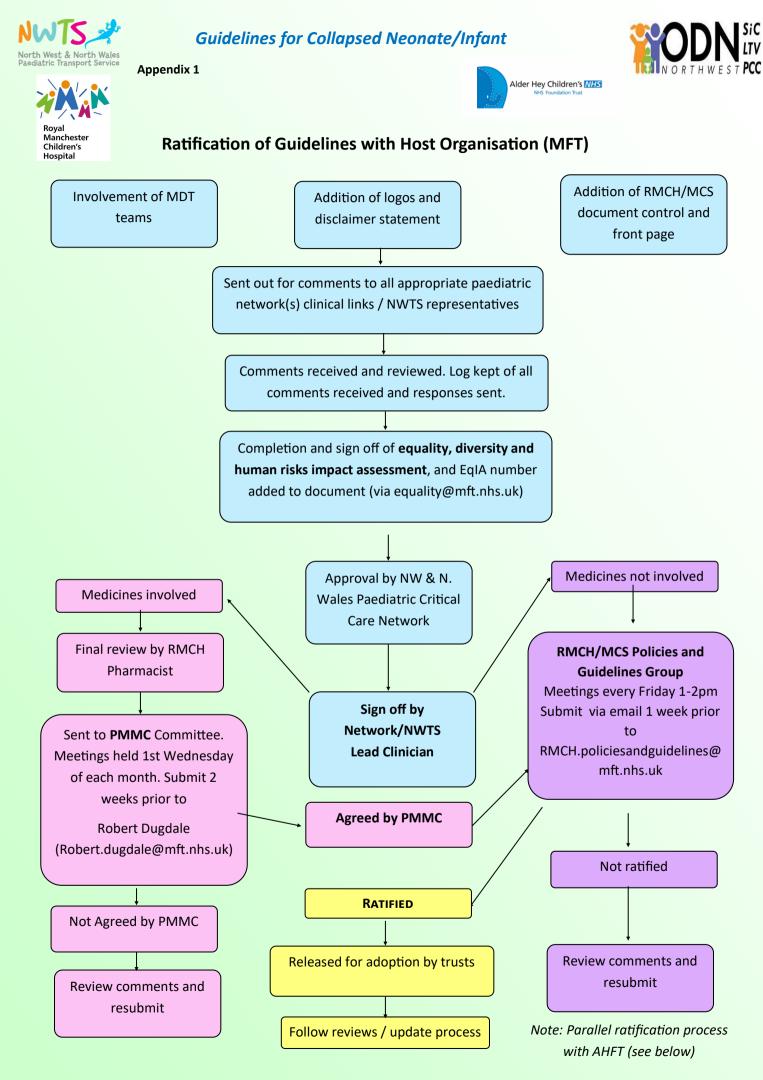
### NWTS website: www.nwts.nhs.uk/guidelines

Useful tools available to support local team time-critical transfers on NWTS regional guidelines page include:

- The Safe Transfer Of the Paediatric Patient (STOPP)
- The Major Trauma Quick Reference Guide
- NWTS regional acute abdomen guideline
- NWTS regional transport guideline
- NW Children's Major Trauma Network guidelines: https://nwchildrenstrauma.nhs.uk/clinical-guidelines

### REFERENCES

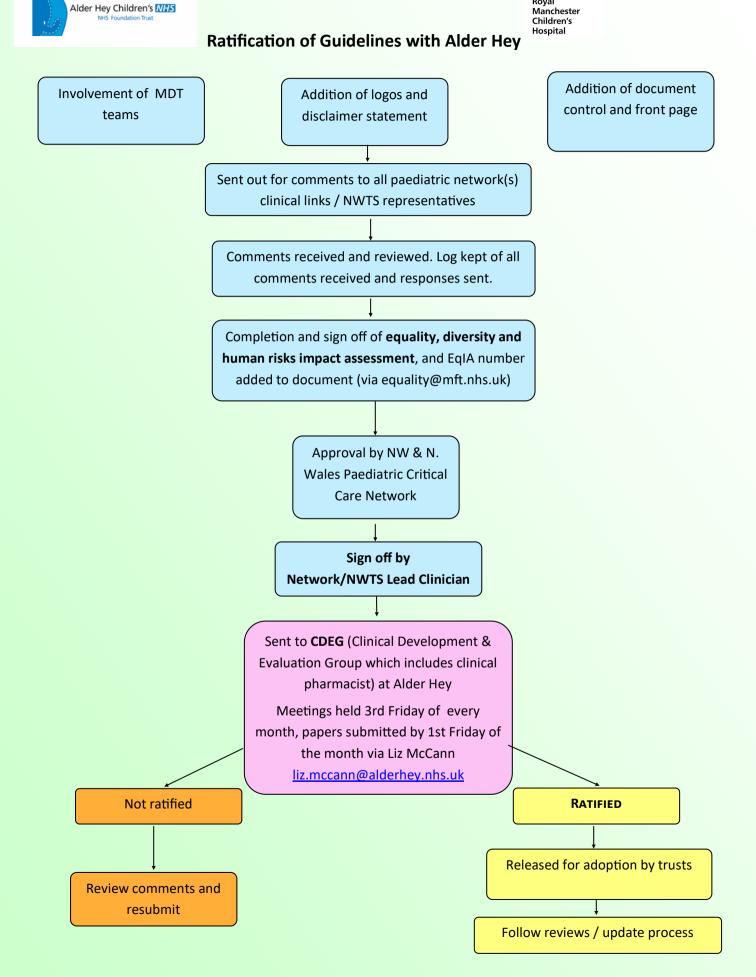
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**Appendix 1 continued** 









# Resources

www.crashcall.net - for intubation drugs / sedation regime

# **Contact numbers:**

NWTS (North West (England) & North Wales Paediatric Transport Service) referral number: 08000 84 83 82 NWTS (North West (England) & North Wales Paediatric Transport Service) office: 01925 853 550 Regional Paediatric Intensive Care Unit Alder Hey Children's Hospital 0151 252 5241 Regional Paediatric Intensive Care Unit Royal Manchester Children's Hospital 0161 701 8000

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# Next Review Due: January 2025

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For the most up to date version of this guideline visit: www.nwts.nhs.uk

