Update in Anaesthesia

Neonatal anaesthesia

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INTRODUCTION

Neonates present a challenge to the anaesthetist. They have unique physiology as they transition from intrauterine to extrauterine life, limited physiological reserve and immature drug handling. The goals of anaesthesia are to provide stable conditions for surgery, minimise physiological disturbance, reduce pain, and support the neonate during the postoperative period. This article will describe general considerations for anaesthesia in term and preterm neonates, and anaesthesia for some specific neonatal conditions.

PREOPERATIVE ASSESSMENT OF THE NEONATE

As for any child undergoing anaesthesia, it is important to take a detailed history and examination, together with relevant investigations to assess the current physiological status and the impact of any associated congenital abnormalities, which may or may not be related to the surgical condition. This helps to plan when best to proceed with the surgery, and the level of postoperative support required.

History

The history should include the gestational age, birth history, current age and weight, and significant peri-natal events such as low APGAR scores, respiratory distress requiring respiratory support, hypoglycaemic episodes, NICU admissions, evidence of sepsis or any antenatal concerns such as maternal illness. The anaesthetist should check whether intramuscular vitamin K has been given to prevent haemorrhagic disease of the newborn. The fasting status should be established if the child is receiving feeds - ideally 2 hours for clear fluids, 4 hours for breast milk, 6 hours for formula feed.

Examination

Examine the child carefully. In particular, it is important to look for signs of respiratory distress (respiratory rate, nasal flare, subcostal recession), and cardiovascular compromise (check heart rate, blood pressure, peripheral perfusion and capillary refill). Check the oxygen saturation – low oxygen saturation may be associated with respiratory disease, or in some cases with cyanotic congenital heart disease.

Investigations

Relevant investigations will be guided by the clinical findings and the underlying condition, although resources may limit investigations that can be performed. They may include the following:

Laboratory investigations:

- Full blood count and haemtocrit
- Blood glucose
- Urea and electrolytes
- Coagulation studies
- Liver function tests and bilirubin
- Capillary blood gas.

Radiological investigations:

- CXR, AXR
- Echocardiogram
- Cranial/spinal/renal ultrasound.

Finally, the anaesthetic plan, including risks, should be discussed with the parent(s) or guardian(s), and consent taken for anaesthesia including regional anaesthesia and blood transfusion if indicated.

DEFINITIONS

- Neonate is aged up to 28 days
- Term neonate is born between 37 to 40 weeks post conception
- Preterm neonate is born at <37 weeks post conception
- Extreme preterm neonate is born <28 weeks post conception
- Low birthweight <2.5kg
- Very low birthweight <1.5kg

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GENERAL PRINCIPLES OF ANAESTHESIA IN NEONATES

It is important to prepare and check all equipment that may be required, prior to the start of anaesthesia (see Figure 1).

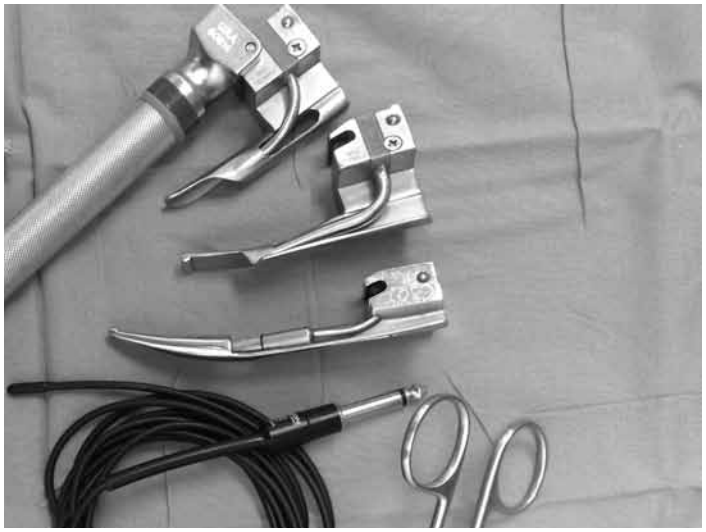

Figure 1. *Airway and monitoring equipment*

Monitoring

Standard monitoring must be applied prior to induction of anaesthesia. This includes oxygen saturation, ideally pre-ductal (right hand) and post-ductal (other limbs). A low post-ductal oxygen saturation may be a sign of low pulmonary blood flow, for instance due to significant pulmonary hypertension in a septic neonate (see transitional circulation below).

ECG and non-invasive blood pressure measurement should be used. The lower limit of mean arterial blood pressure can be estimated to be equivalent to the gestational age in weeks; by about 6 weeks of age, the normal mean arterial pressure is 50-60 mmHg. Basic intra-operative monitoring should ideally also include a precordial or oesophageal stethoscope and, if available, capnography must be used.

Airway equipment

Intubation and ventilation will be required unless it is an extremely short procedure. The size of the tracheal tube will depend on the weight of the neonate; most term babies require a size 3.5 tracheal tube (see Table 1). Make sure that strapping is available. Precut the tape to fix the tracheal tube firmly in place immediately after intubation. An appropriately sized oral airway (preterm 000 – 00 and term neonate 0) and face mask should be available. Dead space within the apparatus is kept to a minimum with the appropriate sized breathing circuit and filter.

Warming

Neonates are extremely vulnerable to heat loss and hypothermia. Hypothermia (core temperature <36°C) is associated with postoperative apnoeas, coagulopathy and poor wound healing, and worsens outcomes. The theatre environment should be warmed (or air conditioning turned down) to at least 20-23°C and the baby kept covered as much as possible. A forced air warmer and a radiant heater should be used if available. Warmed packs should be considered if other sources of warming are not available; take care not to place warmed packs directly in contact with the skin. Fluids and blood products should be warmed. The temperature of the baby should be measured unless the procedure is very quick.

Preparation of drugs

The first thing to be drawn up is a saline flush so that the IV line can be flushed immediately after a drug is given. Calculate the correct dose of analgesics, muscle relaxants and antibiotics and draw these up. Double check dose calculations – it is easy to make 10-fold errors in neonatal practice.

Emergency drugs should be drawn up in the appropriate doses. These include atropine (20mcg.kg^{-1}), suxamethonium (1-2mg.kg^{-1}) and adrenaline (10mcg.kg^{-1}, i.e. 0.1ml.kg^{-1} 1:10,000 adrenaline).

Induction of anaesthesia

Inhalational induction is ideally with sevoflurane although halothane can also be used. The MAC of volatile agents is lower in neonates than in older children, and the onset of anaesthesia is relatively fast due to the rapid respiratory rate and high cardiac output. However, the neonatal myocardium is extremely sensitive to the negative inotropic effects of volatile

Table 1. *Uncuffed tracheal tube sizes and lengths in neonates*

Weight	Tube Size (ID) (MM)	Oral Length (cm)	Nasal Length (cm)
<0.7	2.0	5	5
<1.0	2.5	5.5	7
1.0	3.0	6	7.5
2.0	3.0	7	9
3.0	3.0	8.5	10.5
3.5	3.5	9	11

agents, so deep volatile anaesthesia must be avoided.

Sevoflurane can cause apnoea at high concentrations and the induction concentration should not exceed 6%. The neonate might require assisted mask ventilation until an airway is secured as they may hypoventilate during induction; take care to turn the inspired concentration of volatile agent down if assisted ventilation is used, otherwise the child will become very deep, very quickly. Halothane is more likely to cause myocardial depression and the induction concentration should be kept less than 2%. Halothane can cause arrhythmias in high concentrations, especially if the CO_2 is also high. Atropine (20mcg.kg^{-1} IM or IV) should be considered prior to induction to reduce bradycardia, particularly if halothane is used.

Alternatively, intravenous induction with ketamine (2mg.kg^{-1}) or thiopentone (2-4mg.kg^{-1}) can be performed; induction of anaesthesia will be rapid (the anaesthetist must be confident they can manage the airway), and recovery may be delayed. Ketamine is particularly useful for the critically unwell neonate as cardiovascular depression is minimised. Use glycopyrrolate (10mcg.kg^{-1} IV) or atropine (20mcg.kg^{-1} IV or IM) to minimise the secretions caused by ketamine.

Maintenance of anaesthesia

Anaesthesia is maintained with volatile, oxygen and air or nitrous oxide. A ketamine infusion run at 2-4mg.kg.hr^{-1} is a useful alternative in unstable neonates.

Attention must be paid when positioning the patient and pressure points must be protected. Whenever the child is moved, check the position of the tracheal tube as it is very easy to displace the tracheal tube in neonates, which could have potentially catastrophic consequences.

Pain management

It is important to consider pain management in all neonates. Pain pathways are fully developed before birth, and neonates display the physiologic, hormonal, and metabolic markers of the stress response.[1] Preterm infants have been shown to have an increased sensitivity to pain and even non-painful stimuli may be perceived as painful.[2] Long-term effects on pain responses have been documented in neonatal boys who were circumcised without analgesia.[3] However, immature metabolic pathways for drugs and immature respiratory control mean that neonates are more sensitive to the side effects of analgesics commonly used during surgery.

Multimodal analgesia should be used for all neonates. Options include paracetamol (7.5mg.kg^{-1} IV, or 20mg.kg^{-1} PR), opioids such as fentanyl or morphine titrated to effect (fentanyl 1mcg.kg^{-1} IV, morphine 10-20mcg.kg^{-1} IV). Regional anaesthesia or infiltration of local anaesthetics should be used where possible. Non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided because of the immature renal system. Non nutritive sucking, sucrose and breast milk have also been shown to be safe and effective for reducing pain associated with procedures such as cannulation.[4,5]

Invasive monitoring

Invasive monitoring (intra-arterial and central venous pressure) may be indicated depending on the type of surgery and the physiological status of the patient. Invasive monitoring is mandatory in circumstances such as cardiac surgery where there is the potential for rapid changes in blood pressure, use of inotropes or potential for large volume blood loss. In other circumstances, for instance neonatal laparotomy, the risks/benefits should be considered. Invasive monitoring is time consuming to insert, associated with complications and may delay the start of surgery. If the surgery is sufficiently urgent it may be necessary to proceed without. 24G or 22G catheters may be inserted into radial or femoral arteries for arterial monitoring, but distal limb perfusion must be checked. 4Fr or 5Fr central lines may be inserted into the femoral or internal jugular vein, ideally with ultrasound guidance. Near infrared spectroscopy (NIRS) can be used, if available, as a non-invasive monitor of tissue perfusion.[6]

Oxygen

Unmonitored oxygen therapy leading to hyperoxia in neonates is associated with retinopathy of prematurity, bronchopulmonary dysplasia and damage to the developing brain. Neonatal exposure to 100% oxygen is rarely necessary, and should be avoided except prior to interventions such as intubation. Hypoxia is also harmful, so targeting oxygen saturation levels between 91% and 95% is probably the safest practice.[7-9] In low income countries where it may not be possible to deliver a variety of inspired oxygen mixtures, an air/oxygen mix should be used if possible and oxygen saturations should be monitored before, during and after anaesthesia.

Postoperative apnoea

Apnoea can be defined as a pause in breathing of more than 20 seconds or cessation of respiration of any duration accompanied by bradycardia or oxygen desaturation. Preterm infants are particularly at risk apnoeas due to an immature respiratory control centre. This effect is potentiated by general anaesthetic agents, and all term neonates <44 weeks post-conceptual age (PCA) and pre-term neonates (<60 weeks PCA) are at risk of postoperative apnoea. Infants with multiple congenital abnormalities, a history of apnoea and bradycardia, chronic lung disease and anaemia (Hb <10g.dl^{-1}) are at particular risk for postoperative apnoeas.[10]

Prophylactic caffeine (10mg.kg^{-1} orally) can be given to prevent

post-operative apnoea in premature neonates.[11] Intravenous aminophylline (5mg.kg^{-1}) is an alternative although it has more side effects including tachycardia, jitteriness, irritability, feed intolerance, vomiting and hyperglycaemia.

It is important to allow sufficient time for neonates to wake up at the end of the operation, and they should be closely monitored in recovery until the anaesthetist is happy that they have returned to their normal awake state. All neonates <44 weeks post-conceptual age (PCA), ex-preterm infants up to 60 weeks PCA and any patients with whom there is any concern regarding the possibility of post-operative apnoeas should have post-operative apnoea and oxygen saturation monitoring for 24 hours.

Hypoglycaemia and hyperglycaemia

Persistent, recurrent or severe hypoglycaemia (blood glucose <2.5mmol.l^{-1}) may lead to irreversible neurological injury in neonates. Preterm infants and those with intrauterine growth retardation (IUGR) are at particular risk of hypoglycaemia. Fasting times should be minimized, blood glucose should be monitored and glucose containing maintenance fluids should be continued if they have been required prior to surgery.

Treat hypoglycaemia with a bolus of 2ml.kg^{-1} of 10% dextrose. Hyperglycaemia (blood glucose >10mmol.l^{-1}) is also detrimental and is associated with increased mortality and sepsis in extremely low birth weight infants, so do not use boluses of 50% glucose.[12]

Perioperative fluids

Assessment of the fluid status of the neonate will help to guide peri-operative fluid replacement. It is helpful to consider preoperative maintenance fluids, intraoperative fluids and postoperative maintenance.

Preoperative maintenance fluids

A neonate may require preoperative maintenance fluids if they are unable to take fluids by mouth before surgery. In the first few days of life, the sodium requirement is not high, and typically 10% dextrose is recommended. After the post-natal diuresis has occurred at around day 3 of life, an isotonic fluid containing 5% dextrose and sodium should be used, and electrolytes and plasma glucose monitored (Table 2).

Intraoperative fluids

During surgery, isotonic fluids such as Hartmann's or Ringer's lactate must be used for resuscitation, replacement and maintenance to maintain intravascular fluid volume, replace fluid deficits and avoid hyponatraemia. Blood glucose should be monitored.

The decision whether to order or administer blood or blood products will depend on the cardiovascular status of the neonate, presence of haemorrhage, type of surgery, the most recent blood results and the normal expected values (Table 3). Once the decision to transfuse has been taken it may be worth transfusing to higher haemoglobin levels to avoid exposure to further donors. Ideally, the haematocrit should be measured during surgery using near-patient testing device such as a HemoCue® or a blood gas machine. The British Committee for Standards in Haematology (BCSH) has a suggested transfusion 'trigger' for neonatal top-up transfusion (Transfusion Guidelines for Neonates and Older Children (http://www.bcshguidelines.com) (see Table 4). Suggested transfusion doses for blood and blood products are described in Table 5.

Transitional circulation

In utero, the pulmonary vascular resistance is high and there is very little blood flow to the lungs as the placenta is the source of gas exchange. After birth as the neonate takes the first few breaths, a chain of events is set in place that results in the transition from the foetal circulation to the neonatal circulation with closure of the foetal shunts (foramen ovale, ductus venosus and ductus arteriosus). During the first few weeks of life the pulmonary vasculature is highly reactive; an increase in pulmonary vascular resistance can lead to reopening of the foetal shunts, in particular the arterial duct between the pulmonary artery and the aorta. As a result there is right-to-left shunting from the pulmonary artery (deoxygenated blood) to the aorta, causing profound hypoxia. The oxygen saturation measured in the right hand may be normal ('pre-ductal'); the oxygen saturation in the other limbs ('post-ductal') will be low.

During the perioperative period it is important to prevent factors that increase pulmonary vascular resistance such as sepsis, hypoxia, acidosis, hypercapnoea, pain and hypothermia. When post-ductal oxygen saturations drop in relation to preductal oxygen saturations it may indicate a return to a foetal circulation.

Table 2. *Routine maintenance fluids in neonates*

Day 1 of life	2ml.kg.hr^{-1} (50ml.kg.day^{-1})
Day 2 of life	3ml.kg.hr^{-1} (75ml.kg.day^{-1})
Day 3 of life and thereafter	4ml.kg.hr^{-1} (100ml.kg.day^{-1})

Table 3. *Normal haematological ranges for term and preterm babies (adapted from United Kingdom Blood Services Handbook of Transfusion Medicine, p54 4th Edition 2007 TSO)*

	Term	Preterm	Adult
Haemoglobin $g.l^{-1}$	140 - 240	140 -240	115 - 180
Platelets x $10^9.l^{-1}$	150 - 450	150 -450	150 - 400
PT (sec)	10 -16	11 - 22	11 - 14
APTT (sec)	31 - 55	28 - 101	27 - 40

Table 4. *The British Committee for Standards in Haematology (BCSH) transfusion 'trigger' for neonatal top-up transfusion - reproduced from British Committee for Standards in Haematology (BCSH) Transfusion Guidelines for Neonates and Older Children - http://www.bcshguidelines.com with permission*

Postnatal age	**Suggested transfusion threshold ($g.l^{-1}$)**		
	Ventilated	**On oxygen/CPAP**	**Off oxygen**
First 24 hours	<120	<120	<100
≤ week 1 (days 1-7)	<120	<100	<100
week 2 (days 8-14)	<100	<95	<75-85
≥ week 3 (days 15 onwards)		<85	Depending on clincial situation

Table 5. *Suggested transfusion doses for blood and blood products (Joint United Kingdom (UK) Blood Transfusion and Tissue Transplantation Services Professional Advisory Committee http://www.transfusionguidelines.org/transfusion-handbook/10-effective-transfusion-in-paediatric-practice/10-2-neonatal-transfusion)*

Product	**Suggested transfusion dose**
Packed cells	10-20$ml.kg^{-1}$
Platelets	10-20$ml.kg^{-1}$
FFP	12-15$ml.kg^{-1}$

Neurodevelopmental effects of anaesthetics in neonates

Inadequate anaesthesia and analgesia have been shown to be detrimental to neonates, and associated with increased mortality. However, many animal model studies have been published recently that have demonstrated accelerated neuronal cell death ('apoptosis') and long-term behavioural changes after animals are exposed to anaesthetic agents in the neonatal period. The situation in humans remains unclear.[13] The risks and benefits of surgery in neonates should be considered carefully, and non-essential elective surgery should be avoided in the neonatal period where possible.

Transfer of neonates

Neonatal surgery should ideally be undertaken in an environment where the facilities and expertise are available for definitive treatment and on-going care. In certain situations, if the baby is unstable and not suitable for transfer to theatre, it may be necessary to undertake surgery on the NICU itself. In certain situations the baby may need to be transferred to a specialist centre. In low-income countries this may not be an option and treatment may not always be possible.

Prior to transfer the appropriate personnel, equipment, drugs and fluids should be prepared and checked using a transfer checklist (Table 6). The neonate should be carefully assessed for stability for transfer or if necessary transfer may need to be delayed for further resuscitation and optimisation. Check that the monitoring is functional and the patient is adequately fluid resuscitated. Take time to ensure that the neonate is stable prior to transfer on the current drug infusions and mode of ventilation.

Careful monitoring during transfer is extremely important and will highlight clinical trends. A detailed handover is essential for good continuity of care.

SPECIFIC NEONATAL PATHOLOGIES

Inguinal hernia repair

Inguinal hernia is common in premature neonates. The timing of surgery depends on the risk of incarceration, bowel strangulation or testicular atrophy versus the risk of

postoperative apnoea and the potential harm to neurodevelopment. The major anaesthetic risk is post-operative apnoea, which has been shown to vary from 4.7% to 49% of patients.[10,14]

Some units prefer spinal anaesthesia for inguinal hernia repair, others use a balanced anaesthetic technique using general anaesthesia with intubation, supplemented with a regional technique. There is currently not enough evidence to show whether the incidence of apnoea is lower using spinal anaesthesia, and the choice is usually determined by local preference of the surgeon and anaesthetist.[15]

Editors' note: As this edition of Update goes to press, the editors are aware that the GAS study is reporting its preliminary findings on apnoea comparing GA and spinal in >700 neonates, publication of full results is expected late 2018.

Caudal anaesthesia using 0.25% bupivacaine 0.75ml.kg^{-1} provides excellent supplementary analgesia for inguinal hernia repair under general anaesthesia. Alternatively, an ilioinguinal block can be performed with 0.5-1.0ml.kg^{-1} 0.25% bupivacaine. These patients may require post-operative apnoea monitoring dependent on their PCA, as discussed earlier, and some premature infants will require post-operative ventilation or CPAP for treatment of apnoea. Paracetamol (7.5mg.kg^{-1} IV or 20mg.kg^{-1} rectal suppository) provides adequate post-operative analgesia.

Anorectal malformations

Anorectal malformations (ARM) occur in approximately 1:5000 live births. They represent a wide spectrum of disease, from a simple membrane involving the distal rectum and anus to more complex anomalies involving the genital and urinary tract. Spinal anomalies are frequently found in these patients. These include spinal dysraphism, low lying cord (LLC) and tethered cord.[16] Plain spinal Xrays and spinal ultrasound are used to screen for these abnormalities although they may be normal in occult dysraphism. Caudal anaesthesia may be beneficial and can be used in ARM if there is certainty that anomalies of the spine and spinal cord have been excluded.[17] ARM may be associated with other anomalies including Vertebral, Anorectal, Cardiac, Tracheoesophageal, Renal and Limb abnormalities, collectively known as the VACTERL association.

Primary surgical repair can be undertaken in the neonatal period although more commonly a colostomy is performed and a definitive repair is carried out at a later date.

If caudal anaesthesia is contraindicated an opioid-based technique is used (fentanyl 1-2mcg.kg^{-1} or morphine 20-50mcg.kg^{-1} [0.02-0.05mg.kg^{-1}], with infiltration with local anaesthetic. Rectal suppositories cannot be used but intravenous paracetamol is a useful adjunct if available. Standard monitoring is usually all that is required. Opioids should be carefully titrated as the usual aim is to extubate at the end of surgery.

The patient may be positioned supine or prone depending on the surgical technique. Prone positioning is associated with increased risk to pressure areas, abdominal compression resulting in difficulty with ventilation, endobronchial intubation or tracheal tube displacement. Long-term outcome is variable depending on the complexity of the anorectal malformation. These patients usually require serial anal dilatations following repair.

Intestinal malrotation

Malrotation occurs in approximately 1:500 live births. Normal intestinal rotation around the superior mesenteric artery (SMA) and fixation during foetal development is interrupted. It may also be associated with congenital diaphragmatic hernia, exomphalos and gastroschisis.

Nearly 50% of cases will present in the first week of life most commonly with bilious vomiting secondary to duodenal obstruction.[18] This may be due to a midgut volvulus, or physical compression secondary to peritoneal tissue bands or abnormal locations of the duodenum and its surrounding structures. If the condition is diagnosed early the neonate may be relatively well with only subtle abdominal signs. The neonate may present late with frank sepsis and peritonitis secondary to perforated or necrotic bowel. The gold standard radiological investigation is an upper GI contrast series. Plain X-rays are useful if there is concern of another diagnosis or to exclude visceral perforation.

These patients require adequate volume resuscitation and electrolyte replacement for ongoing fluid losses and should be taken to theatre as soon as is feasible. A nasogastric tube is inserted to suction the stomach. Prophylactic antibiotics such as co-amoxiclav or benzylpenicllin, gentamicin and metronidazole are required. Ideally invasive monitoring is inserted although it should not delay surgery in the sick neonate. If the gut has been compromised, inotropes may be needed and any coagulopathy will require correction. A central venous line may be required for ongoing total parenteral nutrition in the septic neonate. An opioid based technique can be used although a caudal may be considered if the patient is haemodynamically stable, there are no other contra-indications and extubation is anticipated. Post-operative NICU care and ventilation is often necessary.

Long-term outcomes depend on the extent of the necrotic bowel. Some patients will develop short bowel syndrome and if there is extensive bowel necrosis the mortality is 100%.

Necrotising enterocolitis (NEC)

Necrotising enterocolitis occurs in approximately 0.5 – 5:1000 live births. More than 90% of infants diagnosed with NEC are preterm.[19] Morbidity and mortality are inversely proportional to the infant's post-conceptual age (PCA) and birth weight.

The aetiology of NEC is multifactorial. Risk factors include vascular compromise of the gastrointestinal tract, commencement of enteral feeding, immature gastrointestinal immunity and sepsis. Hypoxia or ischaemia combined with reduced splanchnic blood flow can occur with patent ductus arteriosus (PDA), cyanotic heart disease, respiratory distress syndrome, shock, asphyxia and with the use of umbilical catheters.

NEC may present with subtle gastrointestinal signs including abdominal distension, intolerance of feeds, abdominal tenderness, blood in the stool and bilious vomiting or may present with perforation and peritonitis with systemic signs including shock, temperature instability, acidosis and disseminated intravascular coagulopathy. Supine and decubitus plain Xrays may show the presence of hepatic venous gas, free intraperitoneal air, dilated bowel loops, ascites and asymmetric bowel gas patterns along with pneumatosis intestinalis.

Initial management includes discontinuation of enteral feeds, insertion of a nasogastric tube and commencement of broad-spectrum antibiotics such as benzylpenicillin, gentamicin and metronidazole. Ongoing fluid and electrolyte management with parenteral nutrition will be required. Frequent clinical monitoring of systemic and abdominal signs together with radiographic examination, monitoring of laboratory values and acid-base status guides further management. The only absolute indication for surgery is bowel perforation although the decision to proceed to surgery may be made if there is a clinical deterioration.

The preoperative assessment should evaluate and optimise any cardiovascular instability, metabolic acidosis, coagulopathy and respiratory compromise. If the patient is too unstable it may be necessary to carry out surgery on the NICU.

These patients are often already intubated and ventilated. A high dose fentanyl technique (10-20mcg.kg^{-1}) may be used to promote cardiovascular stability and reduce the systemic stress response.[20] Nitrous oxide should be avoided because of the risk of bowel distension. Low cardiac output state, organ hypoperfusion and acidosis secondary to large fluid shifts is common, and large volumes of intravenous fluids are frequently required. Invasive monitoring is useful to guide fluid management and allow frequent arterial blood gas sampling although this must be balanced against the risk of limb ischaemia in the preterm neonate. Insertion of an arterial or a central line should not delay the start of surgery in the sick infant. There is a significant risk of coagulopathy and significant blood loss, and inotropes are often required. Packed red cells should be available and fresh frozen plasma and platelets are often indicated based on laboratory results or clinical evidence of bleeding. Hypothermia and glucose instability are common and should be managed appropriately.

Mortality remains significant and long term complications include short bowel syndrome and neurodevelopmental delay.

Oesophageal atresia and tracheoesophageal fistula

Congenital tracheoesophageal fistula (TOF) occurs in approximately 1:3,000 live births. It arises during foetal development as a result of incomplete separation of the oesophagus from the laryngotracheal tube. It is classified based on the site and presence of the fistula and whether there is oesophageal atresia (Figure 2). There may be other associated VACTERL anomalies.

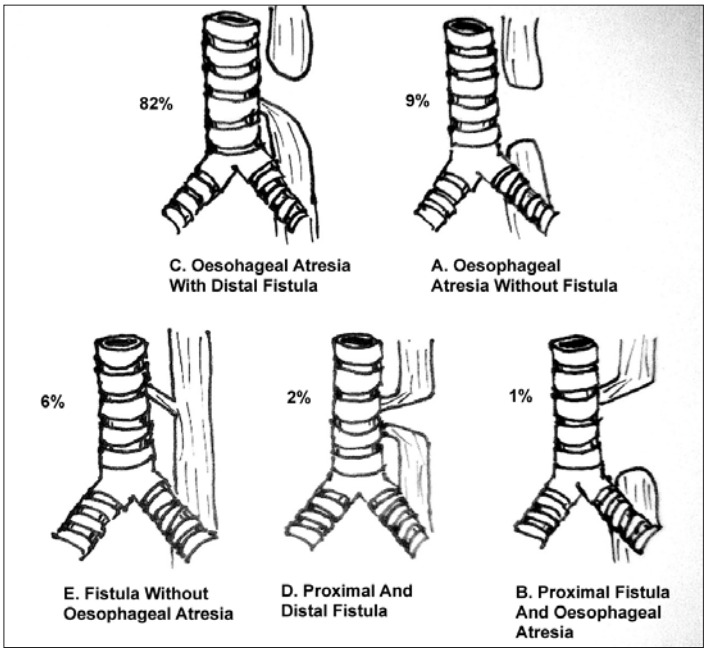

Figure 2. *Incidence and types of oesophageal atresia/tracheo-oesophageal fistula*

Neonates with TOF classically present within a few hours of birth with frothy sputum as they are unable to swallow oral secretions; delayed diagnosis is associated with episodes of coughing and choking associated with cyanosis, particularly if feeding is attempted. There may be copious oral secretions and abdominal distension due to gastric insufflation via the fistula. Left untreated the neonate will develop aspiration pneumonia. The diagnosis of oesophageal atresia is confirmed if it is not possible to pass a nasogastric tube and the chest Xray will show the nasogastric tube coiled in the proximal blind-ending

oesophagus (Figure 3). There may be an absent gastric bubble in isolated oesophageal atresia without a tracheoesophageal fistula.

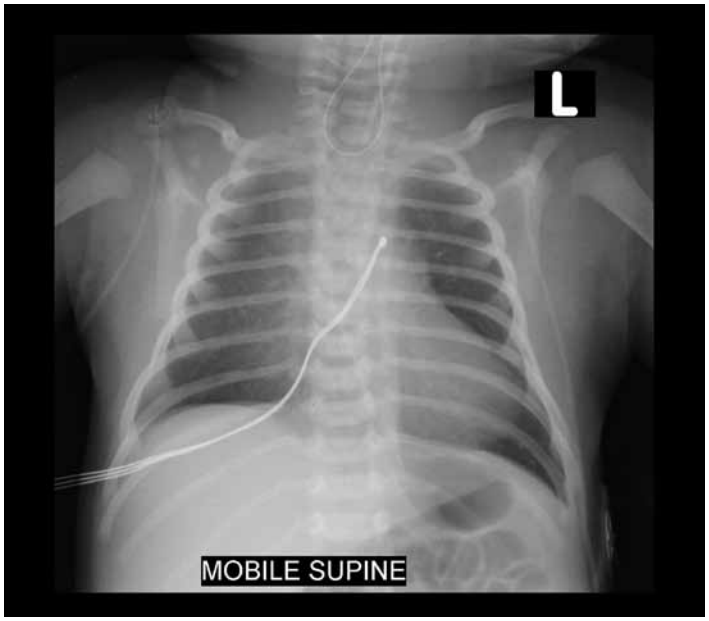

Figure 3. *Chest Xray showing nasogastric tube curled in the upper oesophageal pouch in a child with oesophageal atresia*

The goals of pre-operative management are to stabilise the child, minimise respiratory embarrassment and assess for timing of surgery. A nasogastric tube is inserted into the upper oesophageal pouch to drain secretions. The patient must be nursed head up or on the side to minimise the risk of aspiration. Intravenous fluids and prophylactic antibiotics should be commenced. This allows time for investigations such as an echocardiogram to exclude other associated congenital abnormalities.

Our preferred technique is to induce anaesthesia after pre-oxygenation and to maintain spontaneous ventilation initially with volatile or intravenous anaesthesia. Prior to repair the surgeons may perform flexible or rigid bronchoscopy to assess the level of the fistula and to see if there is a second or proximal fistula. Take note of the distance measured from the cords to the fistula to guide tracheal tube placement; the fistula is mid-tracheal in two thirds of cases, and located at level of the carina in one third of cases. Muscle relaxants and gentle mask ventilation may be given prior to intubation. If possible the tracheal tube is placed distal to the fistula, with the bevel of the tracheal tube facing anteriorly.

A right thoracotomy is performed with the patient on the side with a roll under the chest. The tube position must be checked and effective ventilation confirmed after the change of position. The lung is then retracted which often results in difficulty with ventilation, hypercapnoea and acidosis. Periods of manual ventilation may be required. If gastric distension occurs prior to ligation of the fistula, the tracheal tube should be disconnected intermittently to decompress the stomach via the airway. An arterial line is useful to facilitate arterial blood gas measurement as end tidal CO_2 measurement is unreliable. Alternatively, transcutaneous CO_2 monitoring can be used. Hypercapnoea and acidosis is of particular importance in the presence of certain cardiac anomalies as the increased pulmonary vascular resistance can lead to right-to-left shunting and severe hypoxia. Other pitfalls include ligation of the wrong structure, intubation of the fistula and endobronchial intubation.

A balanced anaesthetic should be given, with bolus fentanyl analgesia as required (1-2mcg.kg^{-1}). Blood loss should be minimal and Ringer's lactate 10-20ml.kg^{-1} is usually all that is required. The wound should be infiltrated with local anaesthetic at the end of surgery. Some term infants born in good condition and with normal preoperative pulmonary function may be extubated at the end of surgery; most are likely to require post-operative ventilation and they should be transferred to a facility able to provide this level of care for their surgery. Many patients will require serial dilatation of the oesophagus during infancy.

Congenital diaphragmatic hernia

Congenital diaphragmatic hernia (CDH) occurs in approximately 1:3000 live births. In most cases the aetiology remains unknown. A defect in the diaphragm, usually on the left side, results in herniation of midgut structures into the thoracic cavity. Pulmonary vascular structure and reactivity are abnormal and there is a varying degree of lung hypoplasia. Diagnosis is made on antenatal ultrasound or on plain Xray postnatally when the abnormal bowel loops can be seen within the thoracic cavity.

Morbidity and mortality is related to the degree of pulmonary hypertension, right ventricular dysfunction and lung hypoplasia.[21] A pre-operative echo is performed as a significant proportion of CDH have associated cardiac anomalies. Mortality still remains high in patients with significant co-existing congenital cardiac disease.[22] It is generally accepted that delaying surgery, usually for 24-48 hours, allows a period of stabilisation. The reduction in pulmonary artery pressures and improvement in right ventricular dysfunction may improve outcome.

There has been a significant improvement in survival over the past 20 years due to the introduction of 'gentle ventilation' strategies.[23] These include permissive hypercapnoea ($PaCO_2$ <70mmHg), limiting inflation pressures (avoid PIP>25cm H_2O and PEEP > 5 cm H_2O) and accepting relative hypoxaemia

(aim for pre-ductal SpO_2 90-95%). Surgery may need to be performed whilst the neonate is on high-frequency oscillation ventilation or extra-corporeal membrane oxygenation.[24]

Preoperative assessment must pay particular attention to the presence of significant pulmonary hypertension, ventilation requirements, and associated cardiac anomalies. If the infant is not already intubated, anaesthesia is induced with care to avoid gastric insufflation with bag valve mask ventilation and further lung compression. A nasogastric tube is inserted to decompress the stomach. Invasive monitoring is required to allow serial blood gas measurement. There is a risk of blood loss and a unit of packed red cells should be available. In patients with significant pulmonary hypertension, having nitric oxide available in theatre may be critical for treatment of pulmonary hypertensive crises.

A subcostal or transverse abdominal incision is made and the herniated viscera are reduced into the abdomen. The diaphragmatic defect is then either closed primarily or with a prosthetic patch if the defect is large. Thoracoscopic repair is being undertaken in some centres. Following abdominal closure, raised intra-abdominal pressures may lead to difficulty with ventilation and a risk of developing abdominal compartment syndrome, and delayed closure may be necessary. Lung compliance decreases post-operatively and post-operative ventilation is usually necessary. These patients often suffer from chronic respiratory disease, gastro-oesophageal reflux disease and neurodevelopmental delay.

Gastroschisis and exomphalos (omphalocele)

Gastroschisis and exomphalos are both ventral wall defects resulting in herniation of abdominal viscera. Diagnosis is ideally made on antenatal ultrasound scan.

Gastroschisis occurs in approximately 1:3000 live births. The herniated viscera are not covered by a sac. It is thought to occur secondary to an ischaemic insult during abdominal wall development or due to early rupture of the hernia of the umbilical cord. A relatively small percentage (10-20%) are associated with other congenital abnormalities and these predominantly involve the gastrointestinal tract.[25]

Exomphalos occurs in approximately 1:5000 live births. Failure of normal embryological development results in the bowel remaining within the umbilical cord and not returning to the abdomen. The herniated viscera is covered by a sac. There is a high incidence of associated congenital abnormalities including cardiac anomalies. Specific chromosomal associations include trisomies 13, 15, 18 and 21 and it can be associated with Beckwith-Wiedemann syndrome.

To avoid bowel injury the baby is delivered by caesarean section. The operating theatre should be warmed, the baby dried, any exposed bowel covered with plastic and a nasogastric tube is inserted to decompress the stomach. Fluid resuscitation is commenced, a urinary catheter inserted and broad spectrum antibiotics started. Co-existing congenital abnormalities, especially cardiac, should be assessed. A renal or cranial ultrasound may also be indicated.

Surgery is more urgent in gastroschisis due to the ongoing fluid losses and electrolyte and metabolic derangement. If primary closure is not possible then a 'silo' is placed over the exposed bowel, which may require a general anaesthetic if the defect needs extending to fit the device. The silo is suspended above the patient postoperatively, and the bowel is gradually reduced into the abdominal cavity under gravity over the ensuing few days in the NICU. When the patient is stable and spontaneous reduction of the bowel has reached a plateau, then surgery for reduction and closure of hernia is performed. Surgery for exomphalos is less urgent, unless the sac has ruptured. If the patient is stable and the defect is small a primary repair can usually be done. In large defects, if the sac has not ruptured, it may be treated with topical silver sulfadizine to allow epithelisation with definitive surgery at a later stage.

The neonate will require intubation and ventilation for surgery. Expect significant ongoing fluid and heat losses due to the exposed viscera. Peripheral intravenous access may be all that is required, but central venous pressure monitoring and an arterial line are useful to help guide fluid administration. Avoid the femoral vessels as there is a risk of decreased perfusion with the increased abdominal pressures. Placing the post-ductal oxygen saturation probe on either lower limb helps to give an indication if there is poor perfusion. Muscle relaxants will assist the surgeons in reducing the abdominal contents. Reduction of the bowel may cause abdominal compartment syndrome, diaphragmatic splinting and high ventilation pressures. If the intra gastric pressures are >20mmHg or the peak inspiratory pressures exceed 30cm H_2O then a staged repair is indicated.[26]

Unless there is a very small defect the infant will require post-operative ventilation and a generous opioid-based anaesthetic technique can be used (fentanyl 10-20mcg.kg^{-1}). These patients often require parenteral nutrition and a significant proportion present for further abdominal surgery.

CONCLUSION

Improving outcomes in neonatal anaesthesia is dependent on a thorough understanding of the unique anaesthetic requirements of the neonate and a detailed knowledge of the different pathologies that present during this period. Unnecessary surgery should be avoided during the neonatal period as anaesthesia and surgical stress may have detrimental effects on the very immature child.

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