Chapter 11
Neonatal Emergencies

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Key Points
- Neonatal Resuscitation Program (NRP) guidelines should be employed for management of a newborn in distress [1].
- Always consider nonaccidental trauma in newborns presenting to the emergency department (ED).
- Prognosis is often dependent on the underlying aetiology or degree of illness.
- Overall, early recognition and management will improve long-term outcome.

Section A: Common Assessment and Treatment Elements

Introduction

Newborns presenting with respiratory illness should be evaluated promptly as respiratory failure can precipitate quickly. There are many aetiologies of respiratory illness in the newborn period, of which the most common will be reviewed.

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Pathophysiology

- Respiratory distress syndrome (RDS) is caused by insufficient surfactant production, most commonly in preterm infants.
  - Insufficient surfactant causes inappropriate alveolar expansion.
  - Decreased alveolar expansion leads to respiratory distress.
- Bronchiolitis is the result of a viral infection affecting the lower respiratory tree.
  - Bronchioles become inflamed and increase secretion production.
  - Common viral aetiologies include respiratory syncytial virus, human metapneumovirus, rhinovirus, enterovirus, and coronavirus [2].
- Meconium aspiration syndrome (MAS) results from aspiration of meconium at the time of delivery.

Clinical Features

- Initial signs of respiratory distress include tachypnoea and increased work of breathing (Table 11.1)
- As distress progresses, newborns are at risk of developing respiratory failure and apnoea.
- RDS presents in the first days of life (Table 11.2)
- Bronchiolitis is a clinical diagnosis based on physical exam and history [3].
  - Newborns will have increased work of breathing with crepitations and rhonchi on exam.

Differential Diagnosis

- Consider RDS, bronchiolitis, sepsis, bronchopulmonary dysplasia (BPD), pulmonary hypertension, pneumonia, MAS, and pertussis.

Table 11.1 Signs of respiratory distress versus respiratory failure

Respiratory distress	Tachypnoea
	Retractions
	Abdominal accessory muscle use
	Nasal flaring
	Tracheal tugging
Respiratory failure	Bradypnoea
	Apnoea
	Hypoxia/hypoxemia
	Carbon dioxide retention
	Respiratory acidosis

Table 11.2 Comparison of clinical features and management of RDS and bronchiolitis

	RDS	Bronchiolitis
Age	First 2 days of life	First 2 years of life
Aetiology	Decreased surfactant	Viral infection
Signs/symptoms	Tachypnoea, increased work of breathing, hypoxia	Tachypnoea, increased work of breathing, apnoea, hypoxia
Diagnosis	CXR, clinical	Clinical
Management	Supplemental oxygen, surfactant	Suctioning, supplemental oxygen

Table 11.3 Chest x-ray findings in common newborn respiratory diseases

	Chest x-ray finding
Respiratory distress syndrome	Ground-glass opacities
	Decreased lung volumes
	Air bronchograms
Pneumonia	Focal opacities
	Diffuse infiltrates
Foreign body aspiration	Air trapping on the side of the foreign body

- History of meconium presence in amniotic fluids should raise concern for MAS.
- When considering pulmonary hypertension, conduct thorough cardiac evaluation.

Investigations

- Chest radiograph (CXR) should be obtained in patients suspected to have RDS, pneumonia, and foreign body aspiration (Table 11.3).
- CXR is not indicated in patients with bronchiolitis [3].
- Obtain nasal secretions to confirm pertussis infection.
- If suspected, infectious workup should be completed including blood, urine, and cerebrospinal fluid studies (CSF).

Treatment

- Intervention can be tailored to a degree of respiratory distress (Table 11.4).
- Surfactant should be provided to patients with RDS [4].
- Supportive care, including suctioning and supplemental oxygen, is the treatment of choice for bronchiolitis.
  - Salbutamol, normal saline, and hypertonic saline nebulisers have not been shown to improve outcome in bronchiolitis [3].
- For patients without clear viral aetiology, consider antibiotic coverage.
- If suspicious for pertussis, start prophylactic antibiotics.

Table 11.4 Recommended interventions based on level of respiratory distress

	Intervention
Apnoeic	Bag-valve-mask ventilation
	Intubation and mechanical ventilation
Severe distress	Continuous positive airway pressure (CPAP)
	Intubation and mechanical ventilation
Moderate distress	High-flow nasal cannula
	Vapotherm
	Continuous positive airway pressure (CPAP)
Minimal distress	Trial supplemental oxygen via nasal cannula

Prognosis

- Newborns in respiratory distress will require observation and treatment on an inpatient service.
- Newborns with severe RDS are at risk of requiring long-term supplemental oxygen and developing BPD.
- Bronchiolitis has an overall good prognosis with resolution of symptoms once the virus infection resolves.

Prevention

- Prevention of bronchiolitis focuses on reducing virus transmission.
- Avoidance of preterm delivery unless clinically indicated will decrease the risk of RDS.

Section B : Neonatal Cardiac Emergencies

Introduction

Congenital cardiac deformities may present in the first hours to days to weeks of life. Prompt recognition of a cardiac aetiology is imperative, as management from cardiogenic shock differs from management of other aetiologies of newborn shock.

Pathophysiology

- Congenital heart defects are a result of abnormal embryogenesis
- Defects can be classified as cyanotic (Table 11.5) and non-cyanotic (Table 11.6).
- Cyanosis is a result of right to left shunting of non-oxygenated blood into systemic circulation.

Table 11.5 Characteristics of cyanotic heart lesions

Name	Truncus arteriosus	Transposition of the great vessels	Tricuspid atresia	Tetralogy of Fallot	Total anomalous pulmonary venous return
Description	Single arterial trunk supplying pulmonary and systemic circulation	Pulmonary artery supplying systemic circulation; aorta supplying pulmonary circulation	Absence or underdevelopment of the tricuspid valve	Overriding aortic arch, pulmonary atresia, VSD, right ventricular hypertrophy	All four pulmonary veins do not connect correctly with the left atrium
Diagnosis	Echo, CT angiogram	Echo, CXR showing "egg on a string." CT angiogram	Echo	Echo, CXR showing boot-shaped heart	CT angiogram, cardiac MRI
Management	CR support, diuretic therapy, surgical intervention	CR support, PGE1, surgical intervention	CR support, PGE1, surgical intervention	Tetralogy spell: 100 % oxygen, morphine, vasopressor; CR support, surgical intervention	CR support, PGE1, supplemental oxygen, fluid support, surgical intervention

VSD ventricular septal defect, *Echo* echocardiogram, *CT* computerised tomography, *CXR* chest x-ray, *CR* cardiorespiratory, *PGE1* prostaglandin E_1

Table 11.6 Non-cyanotic heart defects

	Coarctation of the aorta	Ventricular septal defect (VSD)
Presentation	Cool BLE extremities	Time of presentation dependent on the size of VSD
	Decrease pulses in the BLE	Signs of heart failure or fluid overload of the lungs
	Decreased blood pressure in the BLE	
Diagnosis	Echocardiogram	Echocardiogram
	CT angiogram or MRA of the heart/aorta	CXR may demonstrate cardiomegaly
Management	Prostaglandin E_1	Haemodynamic support
	Haemodynamic support	Closure of the defect
	Surgical repair	

BLE bilateral lower extremities

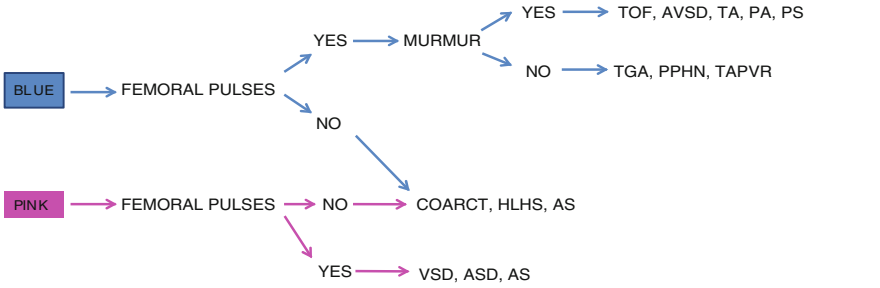
Clinical Features

- Newborns may present with a variety of findings (Table 11.7).

Table 11.7 Possible clinical features associated with congenital heart disease

History	Vital sign abnormalities	Physical exam findings
Abnormal foetal ultrasound	Tachycardia (>160 bpm)	Shock
Poor/difficult feeding	Bradycardia (<80 bpm)	Decreased central/peripheral pulses
Cyanosis	Hypoxia (<90 % on RA)	Poor perfusion
Emesis	Tachypnoea	Increased work of breathing
Poor weight gain	Bradypnoea	Pathologic murmur
Maternal diabetes	Hypertension	Lethargy
Maternal hypertension	Hypotension	Hepatomegaly
Maternal medication		Crackles on lung auscultation
Family cardiac history		

bpm beats per minute, *RA* room air



TOF: Tetralogy of Fallot AVSD: Atrioventricular Septal Defect TA: Truncus Arteriosus PA" Pulmonary Atresia
PS: Pulmonary Stenosis TGA: Transposition of the Great Arteries PPHN: Persistent Pulmonary Hypertension
TAPVR: Total Anomalous Pulmonary Venous Return Coarct: Coarctation HLHN: Hypoplastic Left Heart Syndrome
AS: Aortic Stenosis ASD: Atrial Septal Defect

Fig. 11.1 Assessment of infant colour and central pulses

Differential Diagnosis

- Serious bacterial infection (SBI) should be considered in all neonates presenting to the ED.
- Metabolic abnormalities, respiratory infections, and feeding difficulties can present similarly.

Investigations

- Assess colour of the newborn and central/peripheral pulses (Fig. 11.1).
- Measure four extremity blood pressures and pulse oximetry.
  - In the setting of coarctation, lower extremity blood pressures and pulse oximetry will be decreased compared to right upper extremity.
- CXR should be obtained to assess the cardiac silhouette.

- Obtain electrocardiogram to assess for rhythm disturbances.
- Echocardiogram is used to assess cardiac anatomy and function.
- Consider blood gas to assess pH and arterial oxygenation.
- Detailed maternal and prenatal history, including ultrasound results, should be gathered.
  - Many maternal conditions and medications can be associated with congenital heart disease [5].

Treatment

- Supplemental oxygen should be provided.
- Haemodynamic support should be provided with fluids, inotropes, and chronotropes as clinically indicated.
  - The degree of fluid resuscitation is dependent on the underlying cardiac defect.
- Prostaglandins should be initiated to delay closure of the ductus arteriosus in patients suspected of having a shunt-dependent defect.
  - Initial dose – Prostaglandin E1: 0.05 mcg/kg/min
- Initiate inhaled nitric oxide if pulmonary hypertension is suspected.
- Cardiology consult should be obtained as soon as cardiac defect is suspected.

Prevention

- Improved management of maternal diabetes and hypertension may decrease congenital heart defects.

Section C: Serious Bacterial Infections (SBI)

Introduction

Any newborn with concern for infectious process should be evaluated and treated immediately. SBIs in neonates include, but not limited to, urinary tract infections (UTIs), bacteraemia, sepsis, meningitis, pneumonia, pyelonephritis, and cellulitis. Neonates with bacterial infection are at high risk for long-term morbidity and mortality [6]. Low threshold for workup of an SBI should be maintained when evaluating children less than 29 days old.

Pathophysiology

- Newborns have reduced defence against bacterial infections secondary to an immature immune system.
- The two most common bacteria responsible for infection in newborns are group B streptococcus (GBS) and *Escherichia coli* (*E. coli*) [7].
- UTIs are the most common bacterial infection in this patient group.
- Necrotising enterocolitis (NEC) is a result of intestinal ischaemia, leading to bacterial overgrowth and gut necrosis [8].

Clinical Features

- Rectal temperature greater than 38° C is suggestive of infection.
- Infants may present with hypothermia, lethargy, increased sleeping, poor feeding, jaundice, vomiting, changes in their respiratory pattern, or abnormal vital signs.
- Detailed prenatal and antenatal history should be obtained.
  - Risk factors for infection include prolonged rupture of membranes, maternal fever, maternal infection, and lack of appropriate treatment for maternal GBS.
- Abdominal tenderness, distension, vomiting, bloody stool, and lethargy may indicate NEC.
- Redness, tenderness, or drainage from the umbilicus is concerning for omphalitis.

Differential Diagnosis

- Consider hypoglycaemia, metabolic dysfunction, hyperbilirubinemia, congenital heart conditions, and neurologic dysfunction when assessing a patient with the above features.

Investigations

- Workup should include CBC, blood culture, urinalysis, urine culture, and CSF studies including cell count, differential, glucose, protein, and culture.
- Consider obtaining herpes simplex virus (HSV) tests.
- In the setting of omphalitis, send culture of umbilical discharge.
- Obtain cultures prior to antibiotic initiation unless patient is clinically decompensating.
- For patients with dehydration or concern for metabolic abnormalities, obtain electrolytes and liver function panel.

- CXR should be considered in newborns with respiratory distress, and hypoxia.
- NEC diagnosis and management is reviewed in Table 11.11 [9].

Treatment

- Prompt antibiotic therapy is essential.
- Initiate broad-spectrum antibiotic coverage with ampicillin and gentamicin and/or cefotaxime.
  - Ampicillin, gentamicin, and cefotaxime dosing is dependent on age and weight [10].
  - Acyclovir covers for possible HSV infection: 20 mg/kg/dose every 8 h [10].
- Continue antibiotics until CSF, blood, and urine cultures have resulted.
- NEC management is reviewed in Table 11.11.

Prevention

- Appropriate treatment of maternal GBS infections reduces the likelihood of newborn infection [11].

Section D: Newborn Gastrointestinal Emergencies

Introduction

There are a variety of gastrointestinal (GI) emergencies that may present in the newborn period. The majority of diagnoses can be distinguished based on physical exam and radiologic evaluation. Omphalocele and gastroschisis are not discussed in depth, but ED physicians should be aware of these complications.

Hyperbilirubinemia

Pathophysiology

- Most newborns develop clinical hyperbilirubinemia [12].
- Most cases of hyperbilirubinemia are physiologic, or secondary to normal delayed conjugation and excretion of bilirubin in the newborn, though pathologic aetiologies must be considered.

- Factors contributing to higher bilirubin levels are listed in Table 11.8.
- Patients can be classified into low risk, medium risk, or high risk for complications of bilirubin (Table 11.9) [12].

Clinical Features

- Newborns with hyperbilirubinemia will appear jaundiced and may have decreased activity and desire to feed.
- Jaundice occurs in a cranial to caudal progression and presents within the first week of life, with peak from day 3 to 5.
- Jaundice presenting within the first 24 h of life is likely pathologic.

Differential Diagnosis

- Consider underlying genetic abnormalities, including Gilbert, Crigler-Najjar, and Dubin-Johnson, when diagnosing unconjugated hyperbilirubinemia.
- Also consider causes of elevated direct/conjugated bilirubin, including biliary atresia.
- Infection and sepsis can lead to increased bilirubin levels.

Table 11.8 Risk factors for hyperbilirubinemia

Birth trauma
Dehydration
Breast feeding jaundice
Breast milk jaundice (after 1 week of life)
Haemolysis
ABO incompatibility
Polycythaemia
Preterm infant
Infection
Ethnicity (Asian)

Table 11.9 Classifying newborns into risk category based on age and number of risk factors

	Gestational age		Number of risk factors
Low risk	38 weeks or older	with	0
Medium risk	38 weeks or older	with	1
	35 weeks to less than 38 weeks	with	0
High risk	35 weeks to less than 38 weeks	with	1 or more

Investigations

- Laboratory workup will demonstrate elevated unconjugated bilirubin levels.
- Consider blood type testing, direct bilirubin level, complete blood count, blood smear, liver function panel, and infectious workup.

Treatment

- Treatment for hyperbilirubinemia is based on age, risk factors, and level of bilirubin (Table 11.10).
- The goal of treatment is to prevent encephalopathy secondary to bilirubin deposition in the brain or kernicterus [12].
- Initial intervention includes oral hydration, intravenous (IV) hydration, and phototherapy.
- For high-risk cases or concern for kernicterus, exchange transfusion is employed [12].

Prognosis

- Overall prognosis of hyperbilirubinemia is very good [12].
- The incidence of kernicterus is declining with the advent of phototherapy and exchange transfusion, though patients with kernicterus continue to have long-term neurologic morbidities [12, 13].

Table 11.10 Trigger levels of total serum bilirubin for phototherapy and exchange transfusion in infants 35 weeks or greater [12]

Age of newborn	Risk level	Phototherapy level (μmol/L)	Exchange transfusion level (μmol/L)
24 h	Low	196	324
	Medium	162	282
	High	128	256
48 h	Low	258	376
	Medium	222	324
	High	192	290
72 h	Low	299	410
	Medium	265	360
	High	230	316
96 h	Low	336	428
	Medium	294	384
	High	247	324
5 days and older	Low	359	428
	Medium	307	384
	High	256	324

Prevention

- Prevention of hyperbilirubinemia focuses on appropriate hydration of the newborn and appropriate treatment of maternal infections at the time of delivery.
- Awareness of risk factors will assist in early recognition and intervention.
- Kernicterus is an entirely preventable disease though not reversible [12].

Duodenal Atresia

Pathophysiology

- Duodenal atresia occurs when there is an occlusion of the duodenum, restricting passage of materials through the small bowel.
- Overall incidence is reported at 1 in 6,000 births [14]
- This diagnosis is highly associated with trisomy 21.

Clinical Features

- Patients will present in the first days of life with bilious vomiting.
- Physical exam may reveal a scaphoid abdomen.
- Newborn may pass meconium but is unlikely to have any bowel movements.
- Prenatal history is often positive for polyhydramnios.

Differential Diagnosis

- Infection, oesophageal atresia, tracheaoesophageal fistula, pyloric stenosis, and reflux should be considered.
- Vomiting in the setting of duodenal atresia is bilious in nature, distinguishing this diagnosis from many others.

Investigations

- Abdominal x-ray will reveal a double bubble sign indicating trapped air in the stomach and proximal duodenum.
- Electrolytes, including glucose, should be obtained to assess hydration and nutritional status.

Treatment

- Management of duodenal atresia is reviewed in Table 11.11.

Prognosis

- Complications may include dependence of parenteral nutrition, poor growth, perforation of the duodenum, and stricture at the site of anastomosis [15].

Hirschsprung's Disease

Pathophysiology

- Hirschsprung's disease is the result of failed migration of neural crest cells leading to a lack of innervation in a section of the colon.
- The aganglionic colon is unable to relax, leading to constriction of that segment.
- Hirschsprung's disease is more common in males with a 4:1 male to female predominance [16].

Clinical Features

- Most newborns present with delayed passage of meconium and stool.
  - Ninety-eight percent of normal newborns pass meconium within 48 h.
- Newborns may present with abdominal distension, vomiting, or stringy stools
- Physical exam reveals a tight anal sphincter and explosive stooling with rectal exam.
- Patients may present with toxic megacolon – extreme dilation of the colon leading to distension, pain, perforation, and shock.

Differential Diagnosis

- Consider constipation, meconium ileus, duodenal atresia, or other intestinal obstruction.

Table 11.11 Characteristics of duodenal atresia, Hirschsprung's disease, and necrotizing enterocolitis

	Duodenal atresia	Hirschsprung's disease	NEC
Age	First days of life	First days of life	First months of life
Gender dominance	None	Male > female	None
Emesis	Bilious	Bilious, stool – coloured	Non-bilious or bilious
Abdominal x-ray findings	Double bubble sign	Dilation of the proximal colon, lack of air in the rectum	Pneumatosis, abdominal free air, portal venous gas
Diagnosis	Abdominal x-ray	Rectal suction biopsy	Clinical with x-ray findings
Management	NPO, nasogastric tube placement, IV hydration, surgical repair	NPO, nasogastric tube placement, IV hydration, surgical repair	NPO, nasogastric tube placement, IV hydration, initiate antibiotics, surgical consult

Investigations

- Rectal suction biopsy of the narrowed section of the colon is gold standard for diagnosis [17].
- Anal manometry and barium enema can assist with diagnosis.
- If toxic megacolon is suspected, obtain abdominal x-ray, electrolytes, complete blood count, and blood culture.

Treatment

- Management of Hirschsprung's Disease is reviewed in Table 11.11
- In the case of toxic megacolon, provide resuscitation as clinically indicated and IV antibiotics

Prognosis

- Long-term complications of Hirschsprung's disease include constipation, bowel dysfunction, and toxic megacolon [17].

Section E: Metabolic Emergencies

Introduction

Metabolic disorders can masquerade as many different diagnoses on initial presentation. In newborns, always consider underlying metabolic disorders such as

congenital adrenal hyperplasia (CAH), thyroid dysfunction, fatty acid disorders, amino acid disorders, urea cycle disorders, organic acid disorders, and glycogen storage disease.

Pathophysiology

- Metabolic emergencies in a newborn result from a range of disorders and deficiencies.
- Pathophysiology is entirely dependent on the underlying disorder.

Clinical Features

- Newborns may present with poor weight gain, feeding difficulties, lethargy, emesis, diarrhoea, and decreased movement.
- Female and male infants with CAH present with slight variation [18].
  - Females will likely have ambiguous genitalia with enlarged clitoris.
  - Males tend to present with salt wasting and electrolyte abnormalities including hyponatremia and hypokalaemia.

Differential Diagnosis

- Consider sepsis, genetic disorders, and cardiac abnormalities in these patients.

Investigations

- Newborn screening results should be reviewed.
- Glucose levels should be obtained immediately.
- Blood gas should be obtained to assess for signs of metabolic acidosis or alkalosis.
- Multiple metabolic disorders can present with electrolyte derangement.
- Obtain ACTH, cortisol, and 17-hydroxyprogesterone levels if concerned for CAH [18].
- Consider urine organic acids, serum amino acids, acylcarnitine profile, lactate, and pyruvate in non-emergent phase.
- Consider thyroid studies in patients with clinical signs or maternal history of antithyroid antibodies.

Treatment

- Provide dextrose for patients with hypoglycaemia defined as less than 2.6 mmol/L or symptomatic.
  - Oral feeds with dextrose (milk, formula) if patient tolerates.
  - IV dextrose 10 % bolus of 2 ml/kg.
  - If blood sugar remains low, consider an IV infusion of 10 % dextrose.
- Consider stress-dose steroids if CAH and adrenal crisis are suspected [10].
  - Hydrocortisone IV: 50–100 mg/m^2
- Correct electrolyte abnormalities as indicated.
  - CAH patients may require sodium chloride supplementation [18].
  - Consider sodium benzoate for sodium replacement after discussion with an endocrinologist.

Prognosis

- Many metabolic disorders require long-term dietary supplementation or restriction.

Prevention

- Newborn screening should be administered on all newborns in the first 3 days of life.

Section F: Haematologic Emergencies

Introduction

Bruising, bleeding, and petechiae are not common presenting issues in newborns, though when present, should raise concern. Broad differentials should be maintained for these clinical features.

Pathophysiology

- In neonatal alloimmune thrombocytopenia, maternal antibodies cross the placenta and target paternally derived antigens, causing destruction of the platelet.
- Autoimmune thrombocytopenia occurs when maternal antibodies target maternal and neonatal platelets.

Clinical Features

- Patients may present with easy bleeding, petechiae, and purpura.
  - Assess if males experienced prolonged bleeding with circumcision.
- Mental status changes could be indicative of intracranial haemorrhage.

Differential Diagnosis

- Consider vitamin K deficiency, neonatal alloimmune thrombocytopenia, and autoimmune thrombocytopenia.
- Also consider nonaccidental trauma, infection, underlying coagulopathy, necrotizing enterocolitis, haemophagocytic lymphohistiocytosis, and leukaemia.

Investigations

- Gather thorough history including maternal medications, illnesses, birth history, and vitamin K administration.
- Obtain complete blood count, coagulation panel.
- Maintain a low threshold for infectious workup
- Cranial computerised tomography (CT) scan or ultrasound should be completed if concerned for intracranial bleed.
- Consider haematology/oncology consult.

Treatment

- Immediately administer vitamin K if not previously given or coagulopathy is suspected.
  - Vitamin K: 0.5–1 mg intramuscularly, subcutaneously, or intravenously [10]
- Provide platelet transfusion if patient is thrombocytopenic and actively bleeding (Table 11.12).
- Consider fresh frozen plasma in setting of moderate to severe bleeding.
- If concerned for an antibody-mediated process, consider intravenous immunoglobulin (IVIg) and/or platelet transfusion [19, 20].
  - IVIg: 0.4–1 g/kg/day [19, 20].
  - Steroid infusion can be considered as adjunct therapy [19, 20].
- Antibiotic therapy for infants with suspected bacterial infection

Table 11.12 Potential triggers for platelet transfusion

Platelet level	Clinical condition
Normal	Platelet function disorder with bleeding
Platelet count <100	Major bleeding
	Disseminated intravascular coagulopathy
	Preoperative
	Sepsis
Platelet count <50	Minor bleeding
	Exchange transfusion
	Preterm infant
Platelet count <30	Asymptomatic term infant

Prognosis

- Intracranial haemorrhage and lower platelet levels are associated with increased risk of morbidity and mortality [21].
- Majority of newborns with thrombocytopenia have good outcomes [21].

Prevention

- Administration of vitamin K immediately following birth
- Close monitoring of infants born to moms with idiopathic thrombocytopenia

Section G: Neurologic Emergencies

Introduction

Seizures may present in the neonatal period as a secondary process or as a primary seizure disorder. The primary goal after stabilisation of the patient should be to uncover and manage the underlying aetiology of the seizure.

Pathophysiology

- Seizures are more frequent in preterm infants and infants with hypoxic ischemic encephalopathy (HIE) [22].
- Abnormal movements occur secondary to withdrawal from maternal drug exposure, commonly opioids.

Clinical Features

- Seizures may manifest with abnormal extremity or eye movements.
- Withdrawal symptoms are listed in Table 11.13 [23].
  - Neonatal abstinence scoring may be utilised to assess for risk of neonatal withdrawal.

Differential Diagnosis

- Aetiologies of seizure range from encephalopathy, stroke, HIE, metabolic disorders, infection, to genetic disorders [24].
- Electrolyte abnormality can present abnormal movements or behaviour.
- Nonaccidental trauma should always be considered.

Investigations

- Obtain glucose and electrolytes in infants with abnormal movements.
- Gather thorough history, including maternal history, maternal medications, maternal infections, and birth history [24].
- Obtain head imaging
- Complete infectious workup, including CSF studies, should be performed.
- Send urine and meconium toxicology screens to assess for withdrawal syndrome.

Table 11.13 Comparison of neonatal seizures and withdrawal syndrome

	Neonatal seizure	Withdrawal syndrome
Clinical features	– Abnormal focal or generalised movements – Decreased responsiveness	– Inconsolable, high-pitched crying – Diarrhoea, vomiting – Poor feeding – Yawning, sneezing – Sleep disturbance
Diagnosis	– Clinical/history – EEG – Infectious workup – CT or ultrasound of the head	– Neonatal abstinence scoring – Maternal drug history – Urine drug screen – Meconium drug screen
Management	– Phenobarbital 20 mg/kg IV[10] – Fosphenytoin 20 mg PE/kg IV[10] – Lorazepam 0.05 mg/kg IV[10]	– Supportive care – Methadone – Second line agents: phenobarbital, clonidine, lorazepam

Treatment

- If hypoglycaemia is present, correct with IV dextrose.
- Consider treating a seizing neonate with phenobarbital or phenytoin [24].
- If seizures are nonresponsive to the above medications, please consider lorazepam or levetiracetam.
- In cases of neonatal withdrawal, provide supportive care and tailor medical management based on maternally abused drug (Table 11.13).

Prognosis

- Prognosis depends on the underlying aetiology and duration of symptoms [24, 25].

Prevention

- Maternal education on the harms of drug use

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