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The goal of this activity is to provide background and essential, practical information for healthcare providers to aid in the recognition and management of serious neonatal emergencies.

Learning Objectives

Upon completion of this activity, participants should be able to:

1. Recognize and differentiate serious (ie, life-threatening) neonatal emergencies from routine presentations
2. Use the mnemonic "THE MISFITS" to aid in the diagnosis of neonatal emergencies
3. Effectively manage and treat serious neonatal emergencies towards a positive outcome

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Neonatal Emergencies

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Emergency department (ED) clinicians are challenged daily to recognize sick patients, diagnose life-threatening illness, and initiate treatment. Outside the everyday experience of managing sick patients in the ED are neonates -- a specific group of patients who often present anxiety-provoking diagnostic challenges. Neonates often arrive at the ED with a nonspecific chief complaint or a history of symptoms that may or may not be benign. In order to recognize which neonates will require life-saving interventions, clinicians need to remain current on these life-threatening illnesses and their management. Because neonatal emergencies are not a common problem, we must continue to educate ourselves with the available literature.

This clinical review addresses the recognition, diagnosis, and ED management of the more common neonatal emergencies. The mnemonic "THE MISFITS" is helpful to quickly recall these critical diagnoses (Table 1).

Table 1. "THE MISFITS"

T-Trauma (nonaccidental and accidental)
H-Heart disease/hypovolemia/hypoxia
E-Endocrine (congenital adrenal hyperplasia, thyrotoxicosis)
M-Metabolic (electrolyte imbalance)
I-Inborn errors of metabolism: Metabolic emergencies
S-Sepsis (meningitis, pneumonia, urinary tract infection)
F-Formula mishaps (under- or overdilution)
I-Intestinal catastrophes (volvulus, intussusception, necrotizing enterocolitis)
T-Toxins/poisons
S-Seizures

Trauma (Accidental and Nonaccidental)

Emergency evaluation of neonates with head trauma may be a difficult process. An infant with nonaccidental head trauma may only have subtle historical findings and no physical exam findings.[1] In addition, the presenting symptoms may be nonspecific. Early diagnosis of an occult head injury may prevent significant long-term morbidity.[2,3] In fact, a recent study on abusive head injuries found that an apparent life-threatening event (ALTE) was often an unrecognized presenting symptom in infants. The study went on to recommend that infants with ALTE without an immediate obvious cause should be evaluated for head trauma with neuroimaging.[4]

Evaluation of neonates with a suspected injury should always include neuroimaging, which may include a computed tomography (CT) scan, ultrasound, or magnetic resonance imaging (MRI). Skull x-rays are not always helpful because infants can have a significant intracranial injury without a skull fracture.[5] In fact, Browning and colleagues[6] found that skull radiographs in children less than 1 year of age were only likely to have positive findings if there were visible signs of injury. Neuroimaging should also be considered in any nonaccidental injury for other skeletal injuries regardless of the physical examination of the head. One study found that 37% of abused children less than 2 years of age had an occult traumatic head injury. In addition, the ophthalmologic evaluation did not demonstrate retinal hemorrhages in most of the patients in this series.[7]

ED management will depend on presenting symptoms but should include evaluation and stabilization of the ABC's (airway, breathing, circulation), a bedside blood glucose evaluation, and appropriate temperature regulation. If there is bruising or a known intracranial bleed, then the laboratory evaluation should include a complete blood count (CBC), platelet count, prothrombin time (PT), and a partial thromboplastin time (PTT). Neuroimaging should be completed after stabilization.

The patient should be admitted and the injury reported to the appropriate state department for abuse. A skeletal survey and ophthalmologic exam should be part of the hospital evaluation.

Heart Disease and Hypoxia

Cyanotic Heart Disease

Cyanosis is a pathologic process that requires immediate attention and evaluation. Although the differential diagnosis of cyanosis includes respiratory causes, infectious causes, central nervous system abnormalities and toxins, there is always the possibility of a cyanotic heart defect and so it should always be considered. Congenital heart defects that present with cyanosis are referred to as the terrible T's and are listed in Table 2:

Table 2. Terrible T's

Tetralogy of Fallot (TOF)
Tricuspid atresia (TA)
Transposition of the great vessels (TOGV)
Total anomalous pulmonary venous return (TAPVR)
Truncus arteriosus (TA)

Cyanotic heart defects may not be detected in the newborn nursery because there is still adequate oxygenated blood to the systemic circulation through a patent ductus arteriosus (PDA). Although the PDA typically functionally closes in the first 10-14 hours of life, several factors can delay its closure, including prematurity, respiratory distress, acidosis, and hypoxia.[8] The PDA is anatomically closed by 2 weeks of age, contributing to the possibility of a relatively delayed detection of cyanotic heart defects.

Providing 100% oxygen is helpful in differentiating between cardiac and noncardiac causes of cyanosis. After providing oxygen, a noncardiac disease should have at least a 10% increase in the pulse oximetry value, whereas cyanotic heart disease will have minimal change in the oxygen saturation. This can also be confirmed with a hyperoxia test, which involves an initial arterial blood gas (ABG) on room air and then a repeat ABG after 10 minutes of 100% oxygen. There should be only minimal change in PaO2 after 10 minutes of oxygen if the cause is cardiac. The ABG should include a methemoglobin percentage because congenital methemoglobinemia, severe diarrhea, and toxins can result in methemoglobin accumulation and cyanosis in a neonate. The management for methemoglobinemia would include methylene blue 1-2 mg/kg IV over 5 minutes.[9]

During stabilization the physical exam should include blood pressures in all 4 extremities and a careful cardiac exam. Although a murmur may be audible, the absence does not exclude a cardiac defect. A chest radiograph (CXR) and electrocardiogram (ECG) should be included in the evaluation, but an echocardiogram is diagnostic.

Management includes prostaglandin E1 (PGE1) as a bolus of 0.05 mcg/kg IV, followed by an infusion of 0.05-0.01 mcg/kg/min IV.[9] Intubation may not be necessary but equipment to secure the airway should be readily available, as a non-dose-dependent complication of PGE1 is profound apnea. Also, patients who require transport to a facility for pediatric subspecialty care may require definitive airway management prior to transportation.

Acyanotic Heart Disease

Acyanotic heart disease typically presents with symptoms of congestive heart failure. There is usually a more gradual clinical decompensation when compared with the cyanotic heart defects and it may not present until after the first 2-3 weeks of age. Table 3 lists the diseases that may present with heart failure.[10]

Table 3. Causes of Congestive Heart Failure in Neonates

Acyanotic heart disease (ventricular septal defect, atrial septal defect, patent ductus arteriosus, coarctation of the aorta)
Severe anemia
Trauma
Sepsis
Supraventricular tachycardia
Metabolic abnormalities
Systemic lupus erythematosus
Thyrotoxicosis

The classic symptoms for congestive heart failure include tachypnea, tachycardia, and hepatomegaly. The history may include poor or slow feeding, sweating or color change with feeding, and poor weight gain.

Initial management will include stabilization of the ABC's, a CXR, ECG, and laboratory evaluation including a CBC and serum electrolytes. An echocardiogram will be diagnostic of the heart defect and management usually includes furosemide (1.0 mg/kg IV), plus dopamine or dobutamine for cardiovascular support. Dopamine may be started at 5-15 mcg/kg/min IV and dobutamine can be initiated at 2.5-15 mcg/kg/min IV.[9] Careful attention is necessary to not fluid-overload these patients. Pediatric cardiology should be consulted. A pediatric cardiothoracic surgeon may be required for inpatient management.

Supraventricular Tachycardia

Supraventricular tachycardia (SVT) is the most common neonatal dysrhythmia. Presenting complaints may range from tachycardia to poor feeding, irritability, heart failure, and shock. This is not usually a difficult diagnosis because the heart rate is sustained at ≥ 220 beats per minute with a QRS < 0.08 seconds. ED management is dependent on the patient stability at presentation. In a stable patient, vagal maneuvers at this age include icing the face, avoiding the nares. If unsuccessful, IV access should be established, and adenosine 0.1 mg/kg IV push followed immediately by flush should be administered (maximum of 6 mg/kg). If SVT persists then a second dose of adenosine 0.2 mg/kg IV (maximum of 12 mg/kg) may be administered. An unstable patient without IV access should be treated with synchronized cardioversion (0.5-1.0 J/kg). If there is established IV access and adenosine is readily available, then the initial cardioversion may be attempted pharmacologically. If the SVT is unresponsive to adenosine or synchronized cardioversion or if a wide QRS is suspected, then amiodarone 5 mg/kg IV over 20-60 minutes may be administered. Alternatively, procainamide 15 mg/kg

IV over 30-60 minutes may be administered. Amiodarone and procainamide should not be administered together because the combination can lead to hypotension and widening of the QRS complex. Lidocaine (1 mg/kg IV) is a final option for a wide QRS and should only be used in consultation with a pediatric cardiologist. A 12-lead ECG should be obtained prior to and after conversion from SVT to normal sinus rhythm. This is a useful diagnostic tool for the cardiologists to help determine further management. A pediatric cardiologist should be consulted for further evaluation.

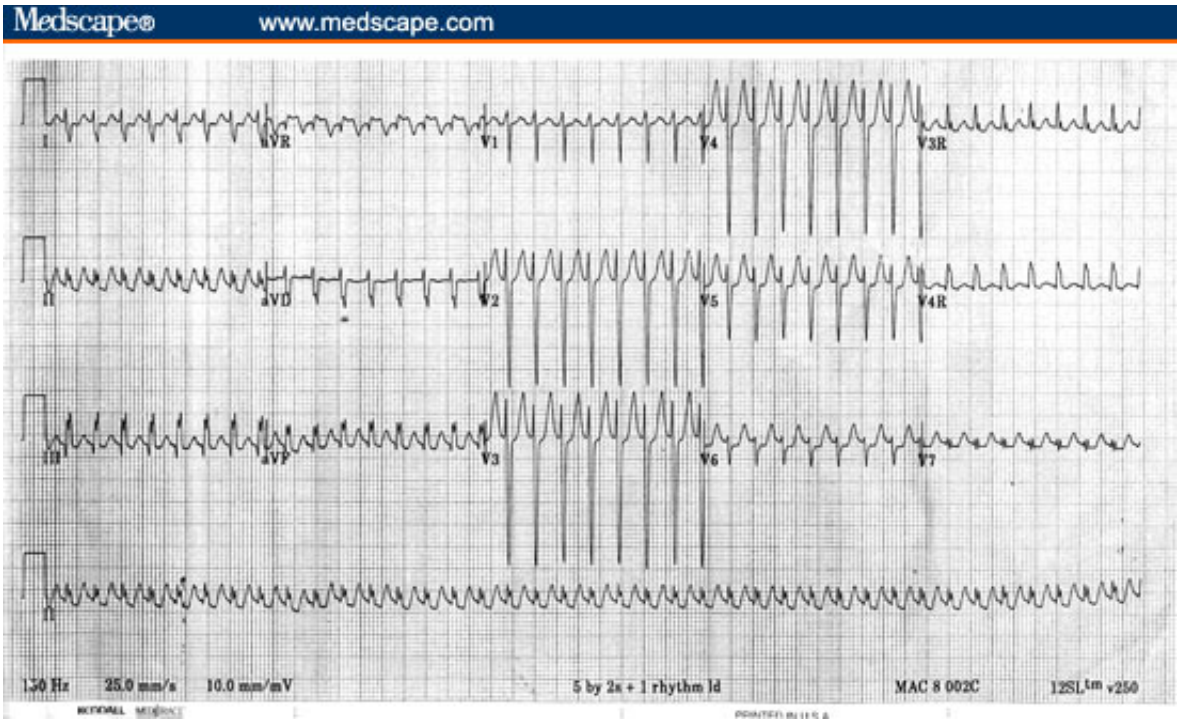

Figure 1. SVT with concomitant right ventricular hypertrophy. This 4-year-old male was postoperative from repair of congenital heart disease (Fontan repair). He was eventually converted to normal sinus rhythm after multiple doses of adenosine.
Courtesy of Stephanie Doniger, MD, Children's Hospital San Diego.

Bronchiolitis

Bronchiolitis is a viral lower-airway disease that is caused by respiratory syncytial virus (RSV) 80% of the time, but other etiologies include adenovirus, influenza, or parainfluenza.[11] In addition, RSV is responsible for 50% to 90% of bronchiolitis hospital admissions.[12] Bronchiolitis is more common in the winter and spring seasons, but may present anytime. These patients may present with more classic symptoms that include rhinorrhea, cough, congestion, or significant respiratory distress and wheezing. Apnea also may be the only initial symptom in an infant with no other respiratory symptoms.

ED management is dependent on the presenting symptoms. Infants with severe, prolonged apnea accompanied by bradycardia and who are unresponsive to oxygen therapy and stimulation may require intubation. More commonly, the decision will need to be made of whether to treat with nebulized racemic epinephrine or a beta-agonist. Nebulized racemic epinephrine has demonstrated better results on respiratory distress than a beta-agonist.[13] The adjunct use of corticosteroids has not been shown to improve the symptoms of bronchiolitis.[14] However, infants who have an underlying reactive airway component to their illness and respond to beta-agonists, or have severe bronchiolitis, are potential candidates for corticosteroid therapy. A fever or sepsis evaluation may often be part of ED management. Although there

remains controversy over the incidence of severe bacterial infections in neonates who have RSV, it has been demonstrated that the presence of a viral infection does not exclude the possibility of a concomitant urinary tract infection (UTI).[15] Hospitalization should be considered for all neonates who are RSV-positive with a strong recommendation in all premature neonates and in all neonates with other comorbid conditions.

Apnea (Apparent Life-Threatening Event, or ALTE)

Apnea is defined as a cessation of respiration for 20 seconds or more and is associated with color change (cyanosis or pallor) or bradycardia.[16] The terms apnea and ALTE are often used interchangeably in conversation but have different definitions. An ALTE is used to describe any event that is "frightening to the observer and is characterized by some combination of apnea, color change, marked change in muscle tone, choking, or gagging."[17] ED management will depend on the historical information provided by observers and by the physical examination. Hospitalization may be appropriate for observation and monitoring. Table 4 lists some of the possibilities of the extensive differential diagnosis that may present with apnea.

Table 4. Common Differential Diagnosis of Apnea

Sepsis
Pneumonia
RSV
Hypothermia
Anemia
Botulism
Dysrhythmias
Acid/base disturbance
Intracranial hemorrhage
Meningitis/encephalitis
Pertussis
Hypoglycemia
Seizures
Gastroesophageal reflux
Child abuse
Inborn errors of metabolism
Electrolyte abnormalities

Endocrine Emergencies

Congenital Adrenal Hyperplasia

Although these patients are often diagnosed at birth by routine newborn screening, occasionally the diagnosis is missed because of an inadequate blood sample, laboratory error, or inability to contact the family. In this instance, the patient may present in the first few weeks of life with symptoms of vomiting, hypoglycemia, or even shock. The most common cause of congenital adrenal hyperplasia (CAH) is a deficiency in the 21-hydroxylase enzyme.[18]

Management includes stabilization of the ABC's, a bedside blood glucose measurement, and serum electrolytes. As with any presentation of a critically ill patient, sepsis should also be included in the differential and empirical treatment with antibiotics may be warranted. The electrolyte abnormalities may include hyponatremia and hyperkalemia. Hypotension that is unresponsive to fluids or inotropes should heighten your suspicion of CAH. The patient should be treated with hydrocortisone 25-50 mg/m^2 IV.[9] It is imperative to also treat the hypoglycemia. Often hyperkalemia in these patients will respond to fluid therapy; however, if the patient is symptomatic or has ECG changes, then calcium chloride, sodium bicarbonate, insulin and glucose, and sodium polystyrene sulfonate (*Kayexalate*) may be necessary. These patients usually require pediatric critical care management and endocrine consultation.

Thyrotoxicosis

Neonatal thyrotoxicosis may develop in infants born to mothers with Graves disease. It is caused by transmission of maternal thyroid-stimulating immunoglobulin. The presentation is often delayed and may present to the ED with symptoms such as poor feeding, failure to thrive, tachycardia, irritability, hyperthermia, vomiting, diarrhea, jaundice, thrombocytopenia, respiratory distress, heart failure and shock. Initial diagnosis may be difficult without a clear history of Graves disease from the mother. Evaluation should include thyroid functions tests. Treatment after stabilization will include propranolol 0.25 mg/kg IV for the tachycardia, and propylthiouracil (PTU) 1.25 mg/kg IV followed by Lugol's solution 1-5 drops by mouth. The Lugol's solution should be given 1 hour after the PTU. This will help to control the hypermetabolic state. Endocrine consultation and admission to a pediatric hospital is recommended.

Inborn Errors of Metabolism: Metabolic Emergencies

Inborn errors of metabolism (IEM) often have a delayed diagnosis and symptoms may be unrecognized because they are uncommon and require a high level of suspicion for diagnosis. State-mandated newborn screening may be helpful for recognizing some of the IEM, but there are over 400 causes that have been identified and it is not possible to routinely screen for all of them.[19,20] Presenting symptoms may be subtle and an IEM should be considered in any neonate who does not have another obvious cause for symptoms. Nonspecific symptoms include poor feeding, vomiting, failure to thrive, tachycardia, tachypnea, or irritability. Occasionally the diagnosis may be more apparent and include symptoms of seizures, lethargy, hypoglycemia, apnea, temperature instability, and acidosis. Physical exam findings are usually normal.

Initial management should include stabilization of the ABC's and a bedside blood glucose evaluation. Laboratory evaluation should include a CBC, serum electrolytes, pH, lactate, ammonia, liver function tests, and urinalysis for reducing substances and ketones. The complete evaluation should also include blood and urine for organic and amino acids. Figure 2 outlines the diagnostic pathway for and IEM with a normal and an elevated serum ammonia. Sodium bicarbonate (starting dose of 1 meq/kg) can be life-saving for patients who are severely acidotic due to organic acidemias.

These patients usually require fluid resuscitation, IV dextrose to prevent further catabolism, and admission to a pediatric hospital with a genetics consultation.

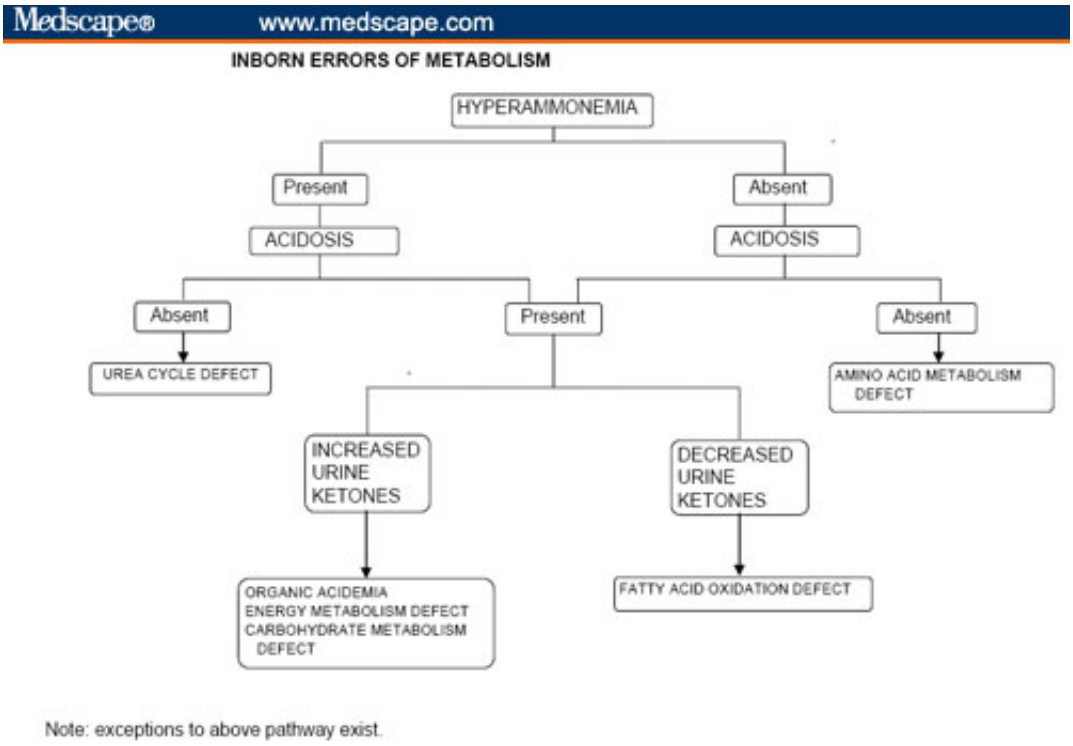

Figure 2. Diagnostic pathway for inborn errors of metabolism with normal and elevated serum ammonia levels. Courtesy of Ken Kwon, MD; University of California, Irvine.

Sepsis

It is standard of care to complete a full sepsis evaluation (CBC, blood culture, urinalysis, urine culture, cerebral spinal fluid [CSF] culture and analysis, and CXR) in any neonate with a rectal temperature of ≥ 100.4° F. Although the evaluation of a febrile neonate is well supported, it is the recognition of sepsis and the initiation of a work-up in neonates with a less specific presentation that is a more difficult challenge. The symptoms and historical facts that should prompt the consideration of a full sepsis evaluation include poor feeding, irritability, apnea, hypothermia, jaundice, rashes, increased sleeping, seizures, or vomiting. A neonate may have sepsis or other serious bacterial infection and may have only minor complaints. A thorough maternal history and physical examination may be helpful. A recent study evaluating the heart rate characteristics of neonates found that reduced heart rate variability was present before clinical signs of sepsis.[21] Initial laboratory screening is not always helpful in recognizing those neonates with an invasive bacterial infection. It has been demonstrated that the use of peripheral white blood cell count is not helpful to differentiate febrile neonates with a more serious bacterial infection from those without a serious bacterial infection.[22] One study demonstrated that a low peripheral white blood cell count increased the odds of bacterial meningitis.[23] In addition, the urinalysis may also be unremarkable in those neonates with a culture positive UTI. Approximately 14% of febrile neonates will be diagnosed with a UTI[24]

It is standard of care to administer broad-spectrum antibiotics (Table 5) to all neonates who undergo a sepsis evaluation or present with life-threatening symptoms that do not have another readily apparent cause.

Table 5. Recommended Antibiotics and Dosages for Neonatal Sepsis

Ampicillin	50-100 mg/kg IV **and**
Gentamicin	2 mg/kg IV **or**
Cefotaxime	50-100 mg/kg IV
Acyclovir	20 mg/kg IV

Neonatal herpes deserves specific mention because the symptoms may be subtle and there may be no maternal history in 60% to 80% of women with an unrecognized infection.[25] Early recognition and treatment with acyclovir 20 mg/kg IV may significantly decrease the mortality from 90% to 31%.[26] Initiation of treatment should be considered in any neonate with high fever, CSF with a lymphocytosis or numerous red blood cells in an atraumatic spinal tap, seizures, or a known maternal history of herpes infection. The CSF analysis should include a herpes polymerase chain reaction (PCR) and herpes culture. There may be an elevation on liver function tests and a CXR may demonstrate pneumonitis. These neonates typically require a higher level of care in a pediatric ICU.

Cutaneous cellulitis should broaden the antibiotic coverage to include an antistaphylococcal agent such as clindamycin 10 mg/kg IV. Omphalitis, a periumbilical infection, often requires fluid resuscitation and prompt surgical intervention because of the possible extension to the peritoneum. These patients should also undergo a full sepsis work-up.

Formula Mishaps

The inappropriate mixing of water and powdered formula or overdilution of concentrated liquid or premixed formula may result in life-threatening electrolyte disturbances or failure to thrive. Hyponatremia may present as seizures and requires recognition of an electrolyte abnormality and immediate correction to stop the seizure. (See next page: Seizures)

Intestinal Catastrophes

Vomiting in the neonatal period should always prompt the consideration of a pathologic process. It may be difficult to differentiate a life-threatening cause from a mild viral gastroenteritis or even severe gastroesophageal reflux. The initial symptoms may be nonspecific and the history may not be helpful in a neonate who has not developed a normal pattern. Bilious emesis is always concerning and should always initiate a pediatric surgery consultation.

Malrotation With Midgut Volvulus

Malrotation is caused by an abnormal rotation of bowel in utero that results in an unfixed portion of bowel that may later twist on itself, resulting in volvulus and bowel ischemia or death. Malrotation occurs in 1 out of 5000 live births and is usually diagnosed in the first month of life.[27] The presenting symptoms include bilious emesis and poor feeding, or lethargy and shock in more advanced presentations.

Initial management includes stabilization of the ABC's, fluid resuscitation, a nasogastric tube placement, and pediatric surgical consultation. Abdominal radiographs may be normal, have signs of small bowel obstruction, or the classic "double bubble" sign may be present. An upper gastrointestinal (GI) study with contrast is the gold standard for diagnosis, but an abdominal ultrasound may also be helpful in an experienced technician's hands. Confirmation radiographic studies should never delay surgical consultation or transfer to an appropriate pediatric facility.

Toxic Megacolon

Toxic megacolon or enterocolitis is a life-threatening presentation of a patient with Hirschsprung disease. Hirschsprung disease occurs in 1 out of 5000 live births and may often go unrecognized because constipation is common and usually benign.[28] The history of constipation, especially with the additional history of failure to pass meconium in the first 24 hours of life, should increase suspicion of Hirschsprung disease. Presenting symptoms may include poor feeding, vomiting, irritability, abdominal distention, and hematochezia and shock as the condition progresses to enterocolitis. Initial management should include stabilization of the ABC's, fluid resuscitation, and administration of broad-spectrum antibiotics. An abdominal radiograph may reveal an enlarged or dilated section of colon. Surgical consultation and pediatric critical care management is necessary in the presence of enterocolitis.

Necrotizing Enterocolitis

Although necrotizing enterocolitis (NEC) is classically a disease of premature neonates that is diagnosed in the neonatal intensive care unit, it may occasionally occur in the term neonate after discharge from the newborn nursery. These neonates may present with symptoms similar to those with Hirschsprung enterocolitis. Management also includes stabilization of ABC's, fluid resuscitation, and nasogastric tube placement. An abdominal radiograph that demonstrates pneumatosis intestinalis or portal air is diagnostic of NEC. Administration of broad-spectrum antibiotics, pediatric surgical consultation, and critical care management is required.

Hypertrophic Pyloric Stenosis

Infants who present with projectile vomiting should be evaluated for hypertrophic pyloric stenosis (HPS). This disease is common and occurs in 1 out of 250 live births with a male:female ratio of 4:1. This disease is more common in the firstborn male. The classic electrolyte disturbance of hypochloremic, hypokalemic metabolic alkalosis is now an uncommon finding because HPS is often diagnosed before these electrolyte abnormalities develop. The history supporting nonbilious emesis immediately after a vigorous feeding is often present. An increased incidence of HPS has been shown in neonates who have had an early exposure to oral erythromycin.[29] Increased awareness of HPS and ease of ultrasound evaluation for the definitive diagnosis usually results in an early diagnosis. The classic physical exam findings of a palpable "olive" structure in the right upper quadrant and visible peristaltic waves may be present. Diagnosis is confirmed with an ultrasound that reveals a thickened and lengthened pylorus. If an upper GI study is performed, a "string sign" will be visible.

Although surgical management is the standard, reports of pharmacologic management with IV atropine followed by oral atropine show satisfactory results.[30] ED management includes stabilization and IV access to replace fluid and electrolytes. Laboratory evaluation should include serum electrolytes.

Hyperbilirubinemia (Jaundice)

Jaundice is a physical finding in the neonate that may represent a normal process in a healthy baby or a more severe or life-threatening illness. Because jaundice is a common complaint, it may be difficult to recognize which neonates require more attention. Initial evaluation will be dependent on the clinical presentation but should include laboratory evaluation for conjugated (direct) and unconjugated (indirect) bilirubin, hematocrit, reticulocyte count, and Coombs test. Direct hyperbilirubinemia is always pathologic and the more common causes include biliary atresia, alpha-1 anti-trypsin deficiency, and hepatitis. Indirect hyperbilirubinemia is usually due to breastfeeding or normal physiologic causes, but the more concerning causes include ABO incompatibility, sepsis, glucose-6-phosphate deficiency, spherocytosis, Gilbert's disease, or Crigler-Najjar syndrome.

ED management should include stabilization of the ABC's, a bedside blood glucose level if warranted, and laboratory evaluation as described above. Initiation of phototherapy -- or in more severe cases exchange transfusion -- is dependent on the neonate's gestational age and total serum bilirubin. This management protocol is described in Table 6. Consultation

with other pediatric subspecialists, including a pediatric gastroenterologist, may be necessary depending on the suspected etiology of the hyperbilirubinemia.

Table 6. American Academy of Pediatrics Recommendations for Phototherapy and Exchange Transfusion in the Healthy Term (> 38 Weeks) Neonate

Age	Phototherapy	Exchange
24 hours	12 g/dL	19 g/dL
48 hours	15 g/dL	22 g/dL
72 hours	18 g/dL	24 g/dL
> 96 hours	20 g/dL	25 g/dL

Toxins

Toxic ingestions are uncommon in this age group, but occasionally result from a maternal ingestion in a breastfeeding mother, homeopathic remedies, or overuse of accepted medications.

Although teething does not occur in the first month of life, colic is a common concern at this point and results in lost sleep and frustration. Teething gels may be used as an attempt to relieve distress for both parents and neonates. Note that teething gels often contain benzocaine which may cause methemoglobinemia with overuse. Star anis tea is a homeopathic remedy also used in several cultures for infantile colic. A recent study in *Pediatrics* described 7 cases of neurotoxicity due to neonatal consumption of star anise tea, suggesting that this should be considered in the differential diagnosis of unexplained irritability, vomiting, or seizures.[31] ED management is primarily supportive and will depend on the clinical presentation. Hospitalization for monitoring and observation is recommended. Finally, baking soda has been used for intestinal gas and has resulted in serious toxicity.[32]

Seizures

Neonates with suspected seizure activity may be difficult to diagnose in the ED setting. The history may only include a concern by the family that their newborn is not acting right or is more somnolent. Neonates have immature cortical development, and seizure activity may not be generalized or tonic-clonic. Symptoms that should be taken seriously include lip-smacking, abnormal eye or tongue movements, pedaling, or apnea.[33,34] Table 7 describes the most common causes of neonatal seizures and reviews the causes on the basis of neonatal age.

Table 7. Common Causes of Neonatal Seizures by Age

First Day of Life	Second Day of Life	Day 4 to 6 Months of Age
Anoxia/hypoxia	Sepsis	Hypocalcemia
Trauma	Trauma	Infection
Intracranial hemorrhage	Inborn errors of metabolism	Hyponatremia/hypernatremia
Drugs	Hypoglycemia	Drug withdrawal
Infection	Hypocalcemia	Inborn errors of metabolism

Hypoglycemia/hyperglycemia	Hyponatremia/hypernatremia	Hyperphosphatemia
Pyridoxine deficiency	Hyperphosphatemia	Congenital anomalies or developmental brain disorders
	Drug withdrawal	Hypertension
	Congenital anomalies or developmental brain disorders	Benign idiopathic neonatal seizures
	Hypertension	
	Benign familial neonatal seizures	

Initial management includes stabilization of the ABC's, bedside blood glucose level, and serum electrolytes. Immediate correction of hypoglycemia (< 40 mg/dL) with 2-4 cc/kg or a 10% dextrose solution may be necessary.[9] If hypoglycemia was the cause of the seizure and the symptoms resolve with glucose replacement, then bedside blood glucose tests should be repeated and the neonate should be placed on a maintenance dextrose IV solution to prevent further hypoglycemic episodes. Other laboratory tests should include a CBC, blood culture, and liver function tests. Because 5% to 10% of all neonatal seizures are of infectious etiology, a full sepsis evaluation should be completed when patient stability permits.[35] The first-line pharmacologic management is lorazepam 0.1 mg/kg IV. This may be repeated 2 or 3 times before moving to the second-line treatment, phenobarbital. The third-line treatment would be phenytoin or fosphenytoin IV. Table 8 provides the step-wise pharmacologic treatment and doses for neonatal seizures.

Table 8. Pharmaceutical Management of Neonatal Seizures

Benzodiazepines	
Lorazepam	0.05-0.1 mg/kg IV
Diazepam	0.2-0.3 mg/kg IV or 0.5 mg/kg rectal
Midazolam	0.1 mg/kg IV or 0.2 mg/kg IM
Phenobarbital	20 mg/kg IV initially then repeat 10 mg/kg IV q 10 minutes (maximum of 50-60 mg/kg)
Phenytoin/fosphenytoin	15-20 mg/kg IV

Serum electrolyte abnormalities, if present, also should be corrected. The more common electrolyte abnormalities include hyponatremia (< 125 mg/kg) and hypocalcemia (< 7 mg/dL). Hyponatremia is corrected with 5-10 cc/kg IV of 3% saline and hypocalcemia with 100-300 mg/kg IV of calcium gluconate solution.[9]

The administration of broad-spectrum antibiotics and acyclovir should not be delayed until the completion of a sepsis evaluation. Once stabilized, neuroimaging should be completed. These patients should be admitted to a pediatric facility for completion of their evaluation and for close monitoring.

Conclusions

This review described and discussed the more common neonatal emergencies that may provoke anxiety in ED clinicians. The mnemonic "THE MISFITS" is a helpful tool that can be readily used to formulate an approach to most neonatal

emergencies that may present in the ED.

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