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To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Dietrich (editor), Dr. Skrainka (CME question reviewer), Dr. Audrey Bowen (author), Dr. Winslade Bowen (author), Dr. Haines (peer reviewer), Ms. Coplin (executive editor), and Ms. Hamlin (managing editor) report no relationships with companies related to the field of study covered by this CME activity.

AHC Media

Common Neonatal Conditions

Emergency department physicians are frequently confronted with newborn concerns from parents or caretakers. The inability of the newborn to communicate and the difficulty with discerning benign conditions from serious conditions is challenging. The authors review the spectrum from common benign concerns to serious life-threatening conditions.

— Ann M. Dietrich, MD, FAAP, FACEP, Editor

Introduction

Children in the neonatal period are very challenging to clinicians. The transition from intrauterine to extrauterine life results in a vulnerable state for the neonate. Genetic disorders, congenital anomalies, and metabolic issues may all present in the first month of life. Discerning normal from abnormal can be very difficult, and recognizing subtle abnormalities may facilitate an early diagnosis, thus improving the infant's chances for a normal life. Important areas are addressed by organ system with both benign and life-threatening diseases reviewed.

HEENT Emergencies

Parents are very aware of the appearance of the head, eyes, ears, and mouths of a neonate. The head is an area of particular concern for parents, and many have questions regarding the shape and configuration.

Caput Succedaneum

Caput succedaneum is common and occurs secondary to swelling of the scalp due to the pressure of the cervix and vaginal walls on the head during vaginal delivery. It may cross suture lines and resolves in a few days without treatment.[1]

Cephalhematoma

A cephalhematoma is a collection of blood under the periosteum of the skull. It occurs in 1-2% of newborns due to birth trauma.[1] On palpation, the swelling is fluctuant and does not cross suture lines. This requires no treatment and resolves in a few weeks. It may increase the risk for hyperbilirubinemia.

Subgaleal Hematoma

Subgaleal hematoma also can occur with birth trauma (i.e., vacuum-assisted deliveries) and is a collection of blood between the periosteum of the skull and the galea aponeurosis of the scalp. The subgaleal space extends from the orbital ridges to the nape of the neck and potentially can collect up to half of the newborn's blood volume, resulting in hemorrhagic shock. The swelling may cross suture lines and obscure the fontanelles. A subgaleal hematoma in the first 72 hours is often due to birth trauma. Treatment involves controlling the hemorrhage, and may require transfusions with packed red cells and fresh frozen plasma. (*See Table 1.*)

EXECUTIVE SUMMARY

- Bilateral choanal atresia presents shortly after birth, sometimes with intermittent cyanosis worsened with feeding and relieved by crying, and the unilateral type may not present until the infant develops an upper respiratory infection.
- Up to 20% of newborns have dacryostenosis, which is an obstruction of the nasolacrimal duct. The majority of cases are due to blockage at the membrane of Hasner, and neonates develop excessive tearing with no nasal drainage.
- Lambdoid synostosis may be confused with positional plagiocephaly. It is best distinguished by viewing the head from the vertex. The infant's ear in positional plagiocephaly is displaced anteriorly from the affected suture. In lambdoid synostosis, the ear is displaced posteriorly from the affected suture.
- Shunt infection occurs in about 10% of infants with shunts, and usually occurs within 6 months of the shunt placement. Organisms causing infections are often skin flora such as *Staphylococcus epidermidis*, *Staphylococcus aureus*, enteric bacteria such as *Escherichia coli* or diphtheroids, and *Streptococcus* species.

Craniosynostosis

The premature closure of one or more cranial sutures results in craniosynostosis. This may occur with certain syndromes, such as Apert or Crouzon, and usually involves multiple sutures, resulting in an abnormally shaped head, poor brain growth, and increased intracranial pressure. Lambdoid synostosis may be confused with positional plagiocephaly. It is best distinguished by viewing the head from the vertex. The infant's ear in positional plagiocephaly is displaced anteriorly from the affected suture. In lambdoid synostosis, the ear is displaced posteriorly from the affected suture. The infant must be examined for signs of increased intracranial pressure: sun setting eyes, prominent scalp veins, poor head control, bulging fontanelles, widened cranial sutures, and papilledema. Diagnosis is confirmed by CT scan. Neurosurgical consultation is recommended for craniosynostosis, and is urgent if there is increased intracranial pressure.

Hydrocephalus

Hydrocephalus is the accumulation of excess cerebrospinal fluid (CSF) in the ventricles of the brain, resulting in increased intracranial pressure, and is a neurosurgical emergency. Risk factors include prematurity and infection. It occurs in approximately 35% of preterm infants with intraventricular hemorrhage. Diagnosis is made with ultrasound or CT of the head. (*See Figure 1.*) Neurosurgical consultation is warranted for placement of a shunt (ventriculoperitoneal or ventriculoatrial). (*See Figure 2.*) Shunt malfunction may occur due to obstruction of the tubing, tube breakage, excess drainage, or migration of the tube. The infant may display signs and symptoms of increased intracranial pressure. Shunt series X-rays and CT of the head confirm the diagnosis. Urgent neurosurgical consultation is necessary for shunt replacement. Shunt infection occurs in about 10% of infants with shunts, and it usually occurs within 6 months of the shunt placement. Organisms causing infections are often skin flora such as *Staphylococcus epidermidis*, *Staphylococcus aureus*, enteric bacteria such as *Escherichia coli* or diphtheroids, and *Streptococcus* species. The infant may present with or without fever, irritability, and meningeal signs. Neurosurgery consultation and CSF analysis establish the diagnosis. Treatment is with intravenous antibiotics for 14 days, usually with vancomycin pending culture reports.

Ophthalmia Neonatorum

The potential etiologic agents for conjunctivitis (*see Figure 3*) in the first month of life include chlamydia, *Neisseria gonorrhea*, *S. aureus*, *Hemophilus influenza*, *Streptococcus pneumoniae*, *Streptococcus viridans*, *S. epidermidis*, and herpes simplex virus (HSV). A gram stain and culture of the conjunctival epithelium should be done. Treatment of uncomplicated bacterial conjunctivitis is with erythromycin eye ointment, except chlamydial and gonococcal infection as mentioned below. Severe infections with periorbital cellulitis or the rare infection with *Pseudomonas aeruginosa* require parenteral antibiotics.[2]

Chlamydia is the most common cause of conjunctivitis in the United States. The incidence is 6.2 per 1000 live

Table 1. Common Etiologies of Scalp Swelling

Caput succedaneum	Cephalhematoma	Subgaleal hematoma
Scalp swelling due to pressure of cervix and vaginal walls during delivery	Collection of blood under the periosteum	Collection of blood between the skull periosteum and the galea
Can cross suture lines	Does not cross suture lines	Can cross suture lines
No treatment necessary. Resolves in a few days.	No treatment necessary. Resolves in a few weeks. May increase bilirubin levels.	This may cause severe anemia from hemorrhage into the space. Blood transfusion with packed red blood cells and fresh frozen plasma may be required depending on severity of the hemorrhage.

births, with the infant often infected during vaginal delivery. The incubation period is 5-14 days after delivery. The infant presents with conjunctival injection and watery to mucopurulent exudate. Eyelid scarring and pannus formation is a late complication if untreated. Culture of the conjunctival epithelium of the everted eyelid is the gold standard for diagnosis. *Chlamydia trachomatis* is an obligate intracellular organism, and the exudate is an insufficient sample for testing. Testing for gonococcal co-infection is recommended. The treatment is oral erythromycin (50 mg/kg/day every 6 hours) for 14 days. Close follow-up is important because treatment failure may occur and a second course of erythromycin may be required. Pyloric stenosis is a possible complication of macrolide therapy in the first 2 weeks of life. Infants treated should be closely monitored for this complication.

N. gonorrhea often produces a severe, bilateral conjunctivitis in the newborn. It occurs usually within 2-5 days of life. The incidence in the United States is significantly decreased due to routine prophylaxis with erythromycin ointment. Clinically, neonates present with a copious purulent discharge, lid edema, and chemosis. Complications include corneal ulcers, perforation, and blindness. Diagnosis is made by gram stain for intracellular diplococci and culture on a modified Thayer-Martin culture medium. Cultures of the oropharynx and anus should be done on affected infants, and they should be tested for concurrent infection with *C. trachomatis*. Consideration should be given to a comprehensive evaluation for disseminated disease.

Treatment is with ceftriaxone 25-50 mg/kg, maximum 125 mg IV or IM. The infant should be hospitalized and observed for disseminated disease.

Herpetic conjunctivitis is rare and is often part of a disseminated infection in the newborn period. It often presents in the first 2 weeks of life with unilateral or bilateral conjunctival injection, nonpurulent drainage, and lid edema. Fluorescein staining shows a corneal defect; microdendrites or geographic ulcers may be present. Treatment is with intravenous acyclovir and ophthalmology consultation.[1] (*See Table 2.*)

Corneal Abrasion

A common complaint an acute care physician frequently encounters is a crying neonate. Neonates frequently scratch their own eye when they rub their eyes with their fingernails, which

Figure 1. Head CT Showing Hydrocephalus

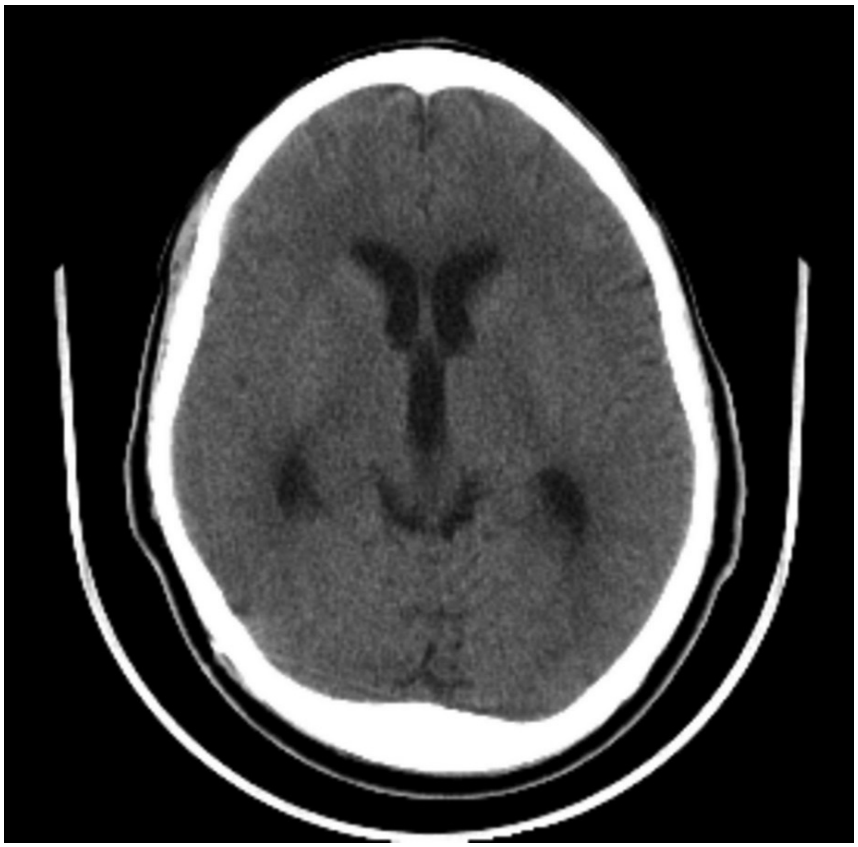


Figure 2. Ventriculoperitoneal Shunt in an Infant

Table 2. Neonatal Ophthalmologic Emergencies

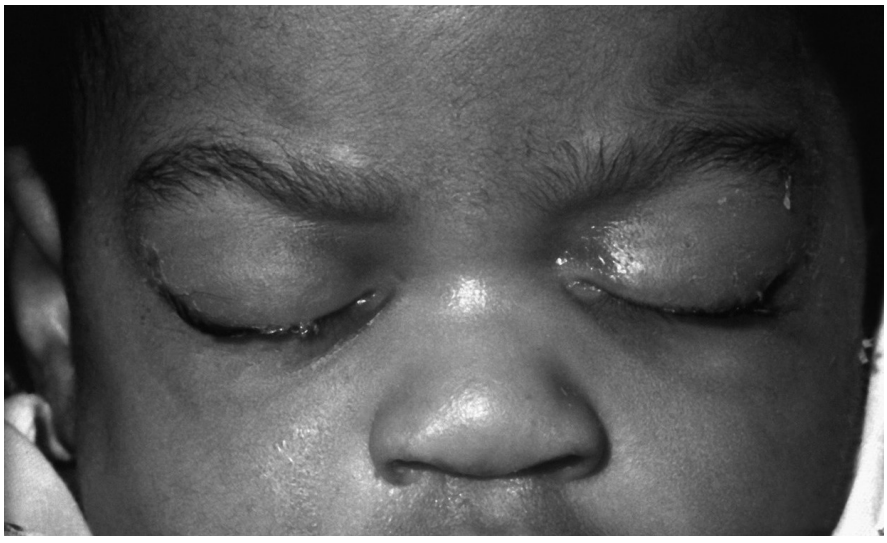
Organism	Diagnosis	Treatment	Age of Presentation
Chlamydia trachomatis	Culture of conjunctival epithelium	Erythromycin 12.5 mg/kg PO every 6 hours x 14 days Efficacy is 80% Must check for concurrent pneumonia and treatment failure Repeat treatment if necessary	5-14 days
Neisseria gonorrhea	Culture on modified Thayer-Martin culture medium	Ceftriaxone 25 to 50 mg/kg (max 125 mg) IV or IM single dose or cefotaxime 100 mg/kg IV or IM single dose at birth Aggressive saline eye irrigation Ophthalmology consult	3-5 days
Herpes simplex virus	Viral culture Fluorescein staining to detect corneal defects	Acyclovir 20 mg/kg IV every 8 hours x 14 days and a topical ophthalmic agent Trifluridine 1% Ophthalmology consult	First 2 weeks of life
Haemophilus influenza, Staphylococcus aureus, Streptococcus viridans, Staphylococcus epidermidis, Escherichia coli, Streptococcus pneumoniae, Moraxella catarrhalis	Eye culture	Erythromycin or gentamicin ophthalmic ointment Topical sulfacetamide, trimethoprim-polymyxin B, or tobramycin	First few weeks
Pseudomonas aeruginosa	Eye culture	IV and topical aminoglycosides required Ophthalmology consult	First few weeks

may result in a corneal abrasion. The infant may present with persistent crying, increased tearing, and conjunctival injection. Diagnosis with fluorescein staining of the eye is facilitated by application of tetracaine 0.5% solution, followed by fluorescein application. Inspection of the eye with a Wood's lamp will reveal the epithelial defect. The treatment is an ophthalmic ointment, such as erythromycin, with follow-up in 24 hours.

Leukocoria

Direct ophthalmoscopic examination of the infant's eye eliciting a white pupillary reflex is abnormal and is termed leukocoria. Causes of leukocoria include retinoblastoma, vitreous hemorrhage from head trauma, retinopathy of prematurity, cataracts, or intrauterine infections such as rubella and toxocariasis. Retinoblastoma is the most common intraocular childhood tumor, occurring in about 1 in 15,000 live births. It has

Figure 3. Conjunctivitis



hereditary and sporadic forms. The hereditary form (25%) is usually bilateral and presents in the first year of life. The sporadic form is more often unilateral and presents after the first year of life. Leukocoria and strabismus are common. A family history of retinoblastoma or osteogenic sarcoma increases the risk for retinoblastoma. Diagnosis is suggested by calcification in the tumor on CT or ultrasound. Urgent ophthalmologist referral is indicated.

Dacryostenosis/Dacryocystitis

Up to 20% of newborns have dacryostenosis, which is an obstruction of the nasolacrimal duct. The majority of cases are due to blockage at the membrane of Hasner, and neonates develop excessive tearing with no nasal drainage. Crusting or matting of the eyelid may occur without conjunctival injection. Treatment involves massaging the duct three times a day to help provide pressure to relieve the obstruction at the membrane of Hasner. Obstruction usually resolves by 6 months of age; if it persists after 12 months, ophthalmology referral is recommended for lacrimal duct probing. A secondary infection may occur and present with mucopurulent drainage, which is called dacryocystitis. The duct may be edematous, with warmth and tenderness to palpation. This requires admission for intravenous antibiotics and ophthalmologist consultation. Complications include periorbital and orbital cellulitis, meningitis, and sepsis.

Choanal Atresia

Choanal atresia is caused by the persistence of the bucco-nasal membrane or bony septum in the posterior nares. Infants are obligate nose breathers. Consequently, if choanal atresia is present and the nasal passage has meconium or secretions, the infant may have significant respiratory distress. The presence of choanal atresia is suspected by the inability to pass a small suction catheter 5-8 Fr through the nares into the pharynx. Bilateral choanal atresia presents shortly after birth, sometimes with intermittent cyanosis worsened with feeds and relieved by crying, and the unilateral type may not present until the infant develops an upper respiratory infection. The placement of an oral airway may alleviate the respiratory distress, and endotracheal intubation may be required if the latter is unsuccessful. A CT scan with intranasal contrast that reveals narrowing of the posterior nasal cavity confirms the diagnosis of choanal atresia.

Table 3. Causes of Neonatal Seizures

Hypoxic ischemic injury: perinatal asphyxia, focal infarction, or stroke
Infections: TORCH infections, CNS infections
Electrolyte abnormalities: hyponatremia, hypoglycemia, hypocalcemia, hypomagnesemia
Inborn errors of metabolism: amino acid disorders, organic acidurias, mitochondrial disorders, peroxisomal disorders, glucose transport (GLUT1) deficiency
Intracranial hemorrhage: intraventricular, parenchymal, subdural, subarachnoid
Congenital brain malformations: microcephaly, Sturge-Weber syndrome, tuberous sclerosis, neurofibromatosis
Pyridoxine (vitamin B6)-dependent seizure, folinic acid responsiveness syndromes
Neonatal abstinence syndrome: drug withdrawal from narcotics such as methadone, heroin, propoxyphene, or opiates
Kernicterus
Hypertension
Epilepsy syndromes: benign idiopathic neonatal seizures, benign familial neonatal seizures, early myoclonic epilepsy, Ohtahara syndrome (early infantile epileptic encephalopathy), or Coppola-Dulac syndrome (malignant migrating partial seizures in infancy)

Laryngomalacia/Tracheomalacia

Laryngomalacia is the collapse of supraglottic structures (arytenoid cartilage, epiglottis) during inspiration. Inspiratory stridor secondary to laryngomalacia occurs usually within the first few weeks and is the most common cause of congenital stridor. The stridor is increased with agitation or supine position. The stridor decreases when the infant is in the prone position because the laryngeal structures are pulled forward. The presence of an acute respiratory infection or reflux may increase the stridor. These infants may have retractions, tachypnea, poor feeding, and failure to thrive. If the latter are present, the infant should be referred to the otolaryngologist for further management. The diagnosis is made by the history and physical examination. A chest X-ray may rule out a mediastinal mass.

Tracheomalacia is due to weakness of airway cartilage with collapse of the intrathoracic trachea, resulting in expiratory wheezing. The diagnosis is made by the history and physical examination. The management is supportive. Supine positioning is recommended if respiratory distress is present. A trial of racemic epinephrine may be helpful if the infant develops significant respiratory distress.

Neurologic Emergencies

Seizures

Seizures in the newborn are often associated with significant illness or brain pathology. Seizures occur in up to 1.4% of term newborns and 10-20% of premature infants. The newborn brain is immature at birth and is still in the process of cortical organization. The brain stem and diencephalon are more developed than the cortical structures, causing distinct types of seizures. The most common seizure type in the neonate is a

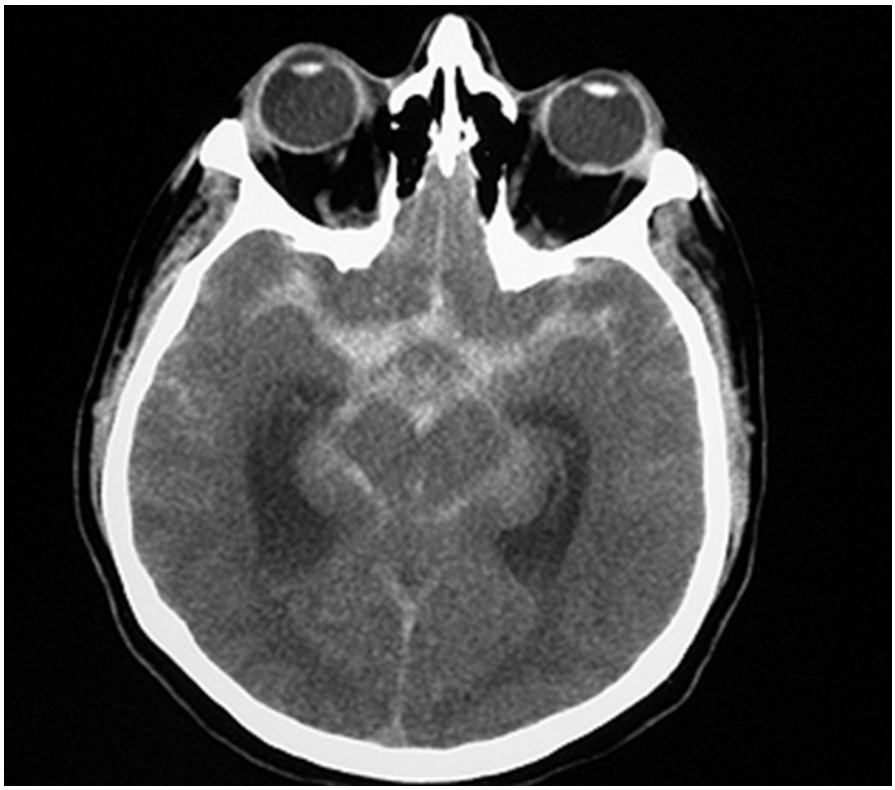
clonic seizure, which is a slow, rhythmic, jerky movement of the facial, extremity, and axial muscles. Tonic seizures are less common, and involve a single extremity or less commonly all extremities. Myoclonic seizures present with rapid twitches or jerks of the flexor muscle groups. Myoclonic jerks occurring during sleep or upon waking are regarded as benign. Seizures in the neonate may have a subtle presentation with lip smacking, chewing, eye deviation, autonomic dysfunction, and stereotypical movements such as stepping, pedaling, or swimming.[3] (*See Table 3.*)

Management of Neonatal Seizures

A careful history is very important and should include the family history, prenatal and intrapartum events, infections, substance abuse, and prenatal care. Ask questions about feeding (breastfed or formula), how formula is prepared, and the use of home remedies. The physical examination includes assessing the type of seizure activity, if present. The skin is checked for bruising in trauma, café au lait spots, cranial hemangiomas, and herpetic lesions. The presence of an unusual odor of sweat or urine may indicate an inborn error of metabolism. Listen for a cranial bruit and conduct a full neurologic exam, including newborn reflexes such as the Moro reflex.

An actively seizing or postictal patient requires immediate attention and careful monitoring of the airway, breathing, and circulation. Oxygen should be administered if the patient is hypoxic. A bedside glucose to check for hypoglycemia should be done and intravenous access rapidly established.[4] Give D10% (2-4 mL/kg) if hypoglycemia is present. Initiate anticonvulsant therapy if active seizures are present. A benzodiazepine such as lorazepam (0.05-0.1 mg/kg) is the first line of therapy for persistent seizure activity. If there is no response to the benzodiazepine, phenobarbital (15-20 mg/kg) is given.[5] It is preferred over phenytoin or fosphenytoin (15-20 mg/kg) in the neonate. (*See Table 4.*) Cardiorespiratory monitoring is mandatory in these infants, as well as monitoring for respiratory depression and the need for intubation.

Figure 4. Intracranial Hemorrhage



Perinatal events such as low APGAR scores, meconium-stained amniotic fluid, and prolonged labor pose an increased risk for perinatal asphyxia and hypoxic ischemic encephalopathy. These infants often develop seizures and developmental delay. CT of the head may reveal cerebral infarct or atrophy. Seizures in the newborn should prompt neurological consultation, radiologic imaging of the brain (CT or MRI), and EEG monitoring. The infant should be admitted to a monitored bed.

Intracranial Infections or Sepsis

Intracranial infections or sepsis may cause seizures in the newborn. Bacterial infections, TORCH infections, toxoplasmosis, congenital rubella, cytomegalovirus (CMV), and Coxsackie virus infections may cause seizures in the first week of life. HSV usually occurs after 1 week of life. Maintain a high suspicion for HSV infection in any neonate with seizures. If infection is suspected, CSF analysis for cell count and culture should be done. Antibiotics, such as ampicillin (50 mg/kg) and cefotaxime (50 mg/kg) or gentamicin (2.5 mg/kg), should be given pending culture results. Consider HSV if the infant has a vesicular rash, maternal history of HSV infection, or CSF leukocytosis with a negative gram stain. Acyclovir (20 mg/kg per dose every 8 hours) should be started until culture reports are available.

Hyponatremia

Hyponatremia may precipitate seizure activity in the newborn and may result from improper preparation of formula or inappropriate administration of free water to an infant. A careful feeding history and method of formula preparation should be obtained. Syndrome of inappropriate ADH secretion (SIADH), cystic fibrosis, malabsorption, or diarrhea may cause sodium derangements resulting in seizures. A basic metabolic panel will reveal the abnormal sodium level. The treatment is with 3% sodium chloride to increase the sodium level above the seizure threshold. Rapid correction of sodium level may result in central pontine myelinosis and should be avoided.

Hypocalcemia

Hypocalcemia may cause focal

seizures and irritability in neonates.[6] It may occur in the first few days of life in infants with diabetic mothers, intrauterine growth retardation, perinatal asphyxia, or prematurity. Late onset, after 10 days, may occur in hypoparathyroidism, DiGeorge syndrome, mitochondrial disorders, and hypomagnesemia. A chemistry panel with calcium assay demonstrates low calcium levels. Hypocalcemic seizures are treated with calcium gluconate slowly over 5-10 minutes with cardiorespiratory monitoring.

Hypomagnesemia

Hypomagnesemia occurs in transient neonatal hypomagnesemia. It can cause hypocalcemia that cannot be corrected until the hypomagnesemia is addressed. Magnesium sulfate is given intravenously to correct low magnesium levels.

Hypoglycemia

Hypoglycemia is often a complication in an infant of a diabetic mother, due to high circulating insulin levels in the mother. This is usually noted shortly after birth, and early feeding is recommended in these infants. Infants who are large or small for gestational age are also at risk for hypoglycemia. Inborn errors of metabolism or endocrinopathies may result in hypoglycemia. A bedside glucose reveals the low glucose levels. Treatment is with D10% (2-4 mL/kg).

Inborn Errors of Metabolism

Inborn errors of metabolism are genetic defects resulting in the deficiency of enzymes, which leads to an excess or deficiency of a metabolite.[7] Dysmorphic features may be evident on examination of the neonate. The parents may note a strange odor to the newborn, such as sweaty feet (isovaleric academia) or sweet-smelling urine (maple syrup urine disease). There are more than 400 inborn errors of metabolism, and all states provide newborn screening but not necessarily the same tests. The screening results are often not available when the infant first presents. The infants usually present with hypoglycemia due to an abnormality in carbohydrate metabolism or fatty acid oxidation within the first 2-3 days of life. Laboratory tests include glucose levels, complete metabolic panel, ammonia and lactate levels, serum amino acid, urine amino acids and urine ketones, CSF lactate, pyruvate, and glycine levels.

Intracranial Hemorrhage

Intracranial hemorrhage in an infant may present with seizures, lethargy, vomiting, and a bulging fontanelle.[8] (*See Figure 4.*) It occurs more frequently in premature infants. In term infants, intracranial hemorrhage may occur due to accidental or non-accidental trauma. A skeletal survey and ophthalmology consult to rule out retinal hemorrhages should be done if non-accidental trauma is suspected. There is a recent trend for parental refusal of vitamin K after delivery. These infants may develop mucosal bleeding from the nose, GI tract, and excessive bleeding after a circumcision.

Pyridoxine Deficiency

An autosomal recessive condition, pyridoxine deficiency is rare but may cause seizures in the neonate. There is decreased GABA (an inhibitory neurotransmitter) synthesis. A pregnancy history may reveal episodic staccato movements during the pregnancy. The infant presents with seizures hours after delivery. The infants are often agitated and may have vomiting, respiratory distress, and metabolic acidosis. A trial of intravenous pyridoxine is recommended for seizures not responding to conventional antiseizure medications. Close monitoring is required, and these infants may require oral pyridoxine for several weeks.

Neonatal Abstinence Syndrome

Maternal drug use, legal or illegal, during pregnancy can have effects on the fetus. The most common illegal drugs abused in the United States are cannabinoids, cocaine, heroin, and methamphetamines. The increase in narcotic abuse and methadone treatment has significant impact on the neonatal population.[9] Poor or absent prenatal care, precipitous delivery, placental rupture, and preterm labor are maternal factors associated with maternal drug use. The infant may have features such as small for gestational age, neonatal stroke, and microcephaly. The effects of withdrawal may occur shortly after birth or within 24-48 hours

Table 4. Neonatal Seizures Anticonvulsant Treatment

Drug	Dose	Maintenance
Lorazepam	0.05 to 0.1 mg/kg/dose (max 2 mg) IV over 2-5 min May repeat in 10-15 min x 1 dose	
Diazepam	0.3 to 0.75 mg/kg/dose (max 2 mg) IV May repeat in 15-30 min x 2-3 doses	0.3 mg/kg/hour continuous infusion
Midazolam	0.1 to 1.1 mg/kg/hour	0.1 to 1.4 mg/kg/hour continuous infusion
Phenobarbital	20 mg/kg/dose IV May give additional 5 mg/kg doses in 15- to 30-min intervals Max total dose 40 mg/kg.	3 to 4 mg/kg day IV daily or divided BID Check levels
Phenytoin or fosphenytoin	20 mg/kg/dose IV Administer phenytoin slowly < 1 mg/kg/min to avoid cardiac arrhythmia or hypotension	3 to 4 mg/kg/day IV divided BID to 4 times daily Check levels
Levetiracetam (Keppra)	20 mg/kg/dose IV	10 mg/kg/day IV divided BID and titrate up to 40 mg/kg/day

Table 5. Neonatal Seizures: Treatment Based on Etiology

Etiology	Risk Factors	Diagnosis	Treatment
Hypoglycemia	Infant of diabetic mother, small or large for gestational age, intrauterine growth retardation, inborn errors of metabolism	Glucose check < 45-50 mg/dL	Dextrose 10% (2 to 4 mL/kg) IV
Hyponatremia	Formula-fed infant, congenital adrenal hyperplasia, syndrome of inappropriate antidiuretic hormone due to hypoxic ischemic encephalopathy	Chemistry panel: Na (usually < 125)	Sodium chloride 3% IV 3 to 5 mL/kg over 1 hour for active seizures.
Hypocalcemia	Infant of diabetic mother, intrauterine growth restriction, perinatal asphyxia, prematurity, hypomagnesemia, hypoparathyroidism, high-phosphate formula, DiGeorge syndrome	Calcium level	Calcium gluconate 10% IV 1 to 2 mL/kg over 5-10 minutes May repeat x 1 Cardiac monitor
Hypomagnesemia	Hypocalcemia	Magnesium levels	Magnesium sulfate 12.5% IV 50 to 100 mg/kg over 1-2 hours to correct level < 1 to 6 mg/dL Cardiac monitor.
Inborn errors of metabolism Pyridoxine (B6)-responsive seizures Pyridoxal phosphate-responsive seizures	Dysmorphic features, strange odor, family history of inborn errors of metabolism.	Complete metabolic panel: decreased glucose, acidosis, increased ammonia level, increased lactate level, serum amino acids, urine amino acids, urine ketones, CSF lactate, pyruvate, glycine.	Non-ketotic hyperglycinemia is treated with sodium benzoate 250 to 750 mg/kg/day to decrease levels of glycine in combination with dextromethorphan 5 to 20 mg/kg/day Or memantine to block glycine effects on NMDA receptors
Pyridoxine (B6)-responsive seizures Pyridoxal phosphate-responsive seizures. Folinic acid-responsive seizures	Family history of inborn errors of metabolism, unexplained neonatal death in sibling, parental consanguinity	Complete metabolic panel: decreased glucose, acidosis, increased ammonia level, increased lactate level, serum amino acids, urine amino acids, urine ketones, CSF lactate, pyruvate, glycine.	Pyridoxine responsive seizures (i.e., deficiency of alpha AASA, PNPO deficiency) treated with pyridoxine 100 mg IV or 30 mg/kg PO May substitute pyridoxal phosphate 10 mg/kg IV or 30 mg/kg/day PO Some patients who don't respond to pyridoxine may respond to pyridoxal phosphate Folinic acid-responsive seizures treat with folinic acid 2.5 mg/kg/day IV
Neonatal Abstinence Syndrome (NAS)	Maternal drug use, poor prenatal care	Urine toxicology, meconium or hair assay. Symptoms in neonate such as jitteriness and seizures.	Swaddling, quiet low light environment, rocking For active seizures lorazepam 0.05 to 0.1 mg/kg/dose IV over 2-5 mins. Repeat if necessary Phenobarbital 20 mg/kg/dose IV Morphine, opium, paregoric are options utilized in different centers Avoid naloxone in infant with maternal methadone use. May precipitate seizure.

Table 6. Causes of Apnea in the Newborn

Neurologic	Seizure Hydrocephalus Meningitis/encephalitis Intracranial injury: hemorrhage, edema Prematurity
Respiratory	Bronchiolitis: respiratory syncytial virus, influenza, parainfluenza, enterovirus, rhinovirus, adenovirus Bronchopulmonary dysplasia (prematurity) Pertussis Pneumonia Congenital lung malformations: congenital diaphragmatic hernia, cystic adenomatoid malformations Spontaneous pneumothorax
Cardiac	Congenital heart disease Arrhythmias: tachyarrhythmia, bradyarrhythmia
Gastrointestinal	Gastroesophageal reflux disease aspiration
Hematologic	Anemia
Infectious	Sepsis
Metabolic	Hypoglycemia Hyponatremia

and as late as 2 weeks after delivery.[10] The latter are more likely to present in the emergency department with complaints such as irritability, jitteriness, high-pitched cry, tremors, seizures, increased or decreased tone, increased or decreased reflexes, increased or decreased suck, lethargy, fever, sweating, tachycardia, tachypnea, cyanosis, nasal congestion, vomiting, diarrhea, weight loss, or increased appetite. The severity depends on the number of drugs abused and the timing of the last dose prior to delivery. The diagnosis may be confirmed using urine toxicology screening; however, results are limited after several days. Meconium and hair analysis may be obtained for confirmation, but these are expensive and the results are not immediate. The differential diagnosis includes sepsis, meningitis, hypoglycemia, hypocalcemia, or hypomagnesemia. Symptomatic treatment is effective in some cases, and this includes swaddling, rocking, holding, and increasing caloric intake with high-calorie formula. More severe cases are treated with medications based on a neonatal abstinence syndrome scoring system. Medications include lorazepam, phenobarbital, paregoric, morphine, and methadone. The neonatal abstinence syndrome scoring system is used to track the infant's progress. Social services should be involved in these cases. (*See Table 5.*)

Botulism

Infants may be colonized with *Clostridium botulinum* spores after ingestion. The disease peaks at about 2-4 months but may present as early as 3 days. Risk factors include ingestion of honey and parents who work in agriculture and live in rural areas. Symptoms occur due to blockade of cholinergic receptors. Neonatal symptoms include constipation, hypotonia, loss of cranial nerve reflexes, flaccid paralysis (descending), and autonomic instability. A stool assay is used for the diagnosis. Treatment is supportive: primarily ventilation and nutritional support. Use of drugs that prolong cholinergic blockade (i.e., gentamicin) are avoided. Baby-BIG, human botulinum immune globulin, is available for use. It significantly decreases the length of the hospital stay by about 50%.

Respiratory Emergencies

Apnea

Apnea is the cessation of airflow for at least 20 seconds or for any duration if central cyanosis is present.[11] Risk factors include prematurity. There are three types of apnea:

1. **Central:** Inspiratory efforts are absent
2. **Obstructive:** Inspiratory efforts exist but airway obstruction is present
3. **Mixed:** Both central and obstructive

An infant presenting to the ED with a history of apnea must be placed on a cardiorespiratory monitor. Testing includes bedside glucose for hypoglycemia, electrolytes for hyponatremia, a complete blood count for anemia, EKG for arrhythmias, chest X-ray for pneumonia, congenital malformations, or pneumothorax. CT of the head is indicated if head trauma or intracranial process such as hydrocephalus is suspected. Viral testing for bronchiolitis and pertussis culture or PCR may be indicated when there is a history of a cough or coryza. If sepsis is suspected, a full sepsis workup is indicated with blood, urine, and CSF analysis and cultures. Maternal history of herpes infection, vesicular skin lesions, or seizures warrants viral cultures of lesions and CSF and treatment with acyclovir. (*See Table 6.*)

Management

The infant who is apneic in the ED requires immediate stimulation and resuscitation with bag valve mask oxygenation and possible intubation if it is prolonged or recurrent. A heart rate < 60 bpm prompts the initiation of cardiopulmonary resuscitation. Admission for monitoring and further diagnostic testing is indicated for an infant who presents with apnea.

Sudden Unexpected Infant Death and Sudden Infant Death Syndrome

Sudden unexpected infant death refers to all unexpected infant deaths and includes deaths due to sudden infant death syndrome (SIDS), the sudden death of an infant < 1 year of age that remains unexplained after a detailed investigation of the case. SIDS is the leading cause of death in infants ages 1 month to 1 year old in the United States. Potential etiologies of sudden unexplained infant death

elicited by the medical examiner include child abuse, suffocation, and metabolic diseases. Death scene investigation, clinical history, and medical examiner findings are utilized to determine the cause of death.

Acute Life Threatening Event

An apparent life-threatening event (ALTE) is an acute change in an infant's appearance, breathing, or behavior, which is frightening to the caregiver. ALTE is a term established at a consensus conference in 1986.[11,12] It includes one or all of the following features:

- **Apnea:** no respiratory effort or difficulty breathing
- **Color change:** cyanosis, pallor, or less often plethoric
- **Change in muscle tone:** often limp but sometimes rigid
- **Choking or gagging**

The incidence of ALTE is 0.05-1%. Risk factors include a history of apnea, recent respiratory illness, feeding problems, infants < 10 weeks of age, prematurity, low birth weight, maternal smoking or drug use, prone sleeping, and bed sharing. Potential etiologies includes gastroesophageal reflux, respiratory infection, seizures, non-accidental trauma, cardiac lesions or arrhythmias, overdose, inborn errors of metabolism, hypoglycemia, and sepsis.

Hospitalization for at least 24 hours for cardiorespiratory monitoring is beneficial for an infant presenting with an ALTE and any of the following:

- Event witnessed in the ED, toxic appearance, recurrent vomiting, respiratory distress
- History of prior ALTE or family history of ALTE or SIDS
- Evidence of trauma
- Presence of a syndrome or dysmorphism
- History of significant respiratory compromise, requiring resuscitation

Initial workup in the ED may include: complete blood count, electrolytes, glucose, urinalysis, calcium, magnesium, urine toxicology, ethanol levels, chest X-ray and EKG, respiratory syncytial virus (RSV), and pertussis testing (if upper respiratory infection is present). On admission, further testing, which includes workup for gastroesophageal reflux disease (GERD), child abuse, seizure, metabolic disorders, cardiac disease, or use of over-the-counter or other drugs, may be done. Recommendations prior to discharge from inpatient hospitalization include CPR training for the parents and education regarding safe home environment, such as supine sleeping position and avoiding tobacco exposure. Home apnea monitoring may be required in

Figure 5. Round Pneumonia from *S. pneumonia*

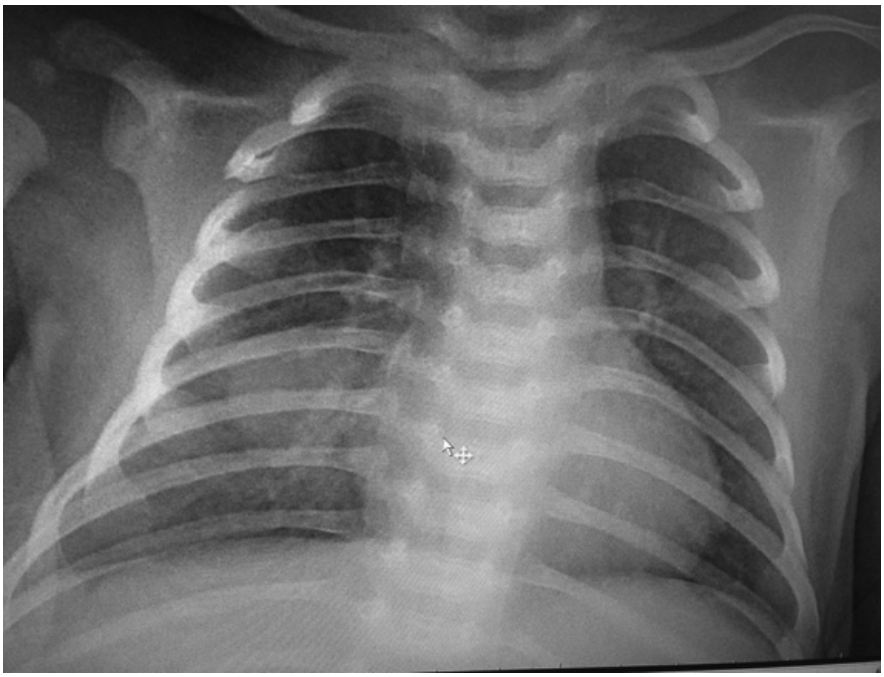
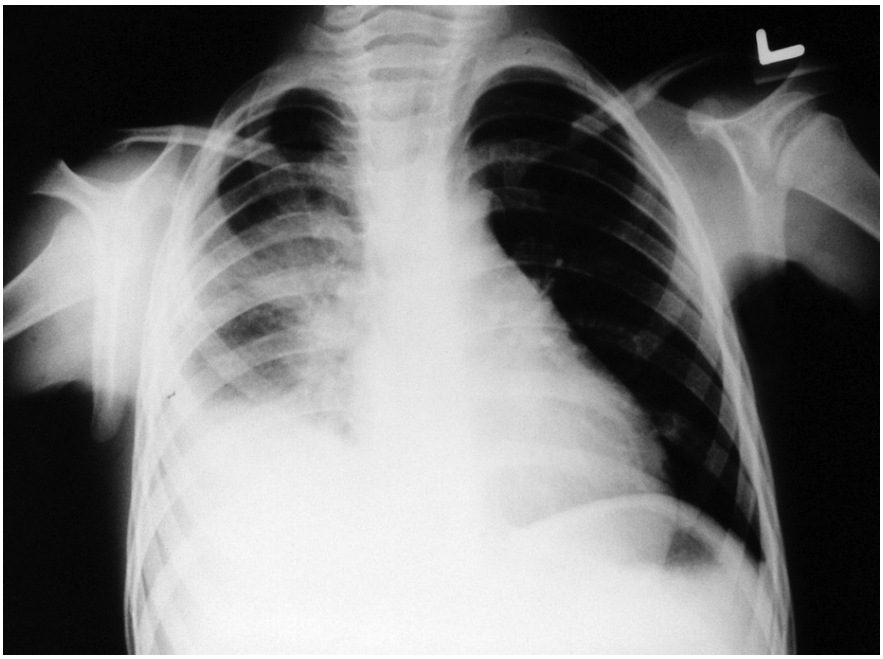


Figure 6. Pneumonia



some cases such as history of prematurity or cardiopulmonary disease. The risk of subsequent death in infants with ALTE is < 1%.

Pneumonia

Pneumonia may be due to bacterial, viral, or atypical organisms. Bacterial causes include group B strep, *S. aureus*, listeria, *E. coli,* pertussis, klebsiella, proteus, pseudomonas, group A strep, and *Neisseria meningitidis*. Viral causes include RSV, influenza, parainfluenza, human metapneumovirus, HSV, CMV, and HIV. Atypical causes include chlamydia, mycoplasma, and mycobacteria. Fungal causes include candida.

The infant may present with cough, tachypnea, respiratory distress, hypoxia, apnea, hyper- or hypothermia, and wheezing. Associated findings are poor feeding and irritability. A chest X-ray may reveal opacification, perihilar infiltrates, air bronchograms, and pneumatocoeles. *S. pneumonia* may appear as a "round pneumonia" and have associated pleural effusions and pneumatocoele.[13] (*See Figures 5 and 6.*) A complete blood count may reveal increased white blood cell count with a predominance of neutrophils in bacterial infections, or decreased white blood cell and predominance of lymphocytes in viral pneumonias. A respiratory panel PCR may reveal the offending organism. The PCR panel usually includes adenovirus, coronaviruses, influenza A and B viruses, metapneumovirus, parainfluenza virus, RSV, rhinovirus, *Bordetella pertussis*, *Chlamydia pneumonia*, and mycoplasma pneumonia.

The management of pneumonia includes oxygen supplementation for hypoxia, ampicillin, and a third-generation cephalosporin such as cefotaxime. Acyclovir is given if HSV is suspected. Erythromycin is recommended for *C. pneumonia* and zithromax for pertussis.

Admission is recommended in the neonatal group due to risk of sepsis, apnea, poor feeding, hypoxia, and respiratory deterioration.

Gastrointestinal Conditions

Newborns often lose 5-10% of their body weight in the first week of life. Weight gain then occurs at a rate of about 20-30 grams per day.

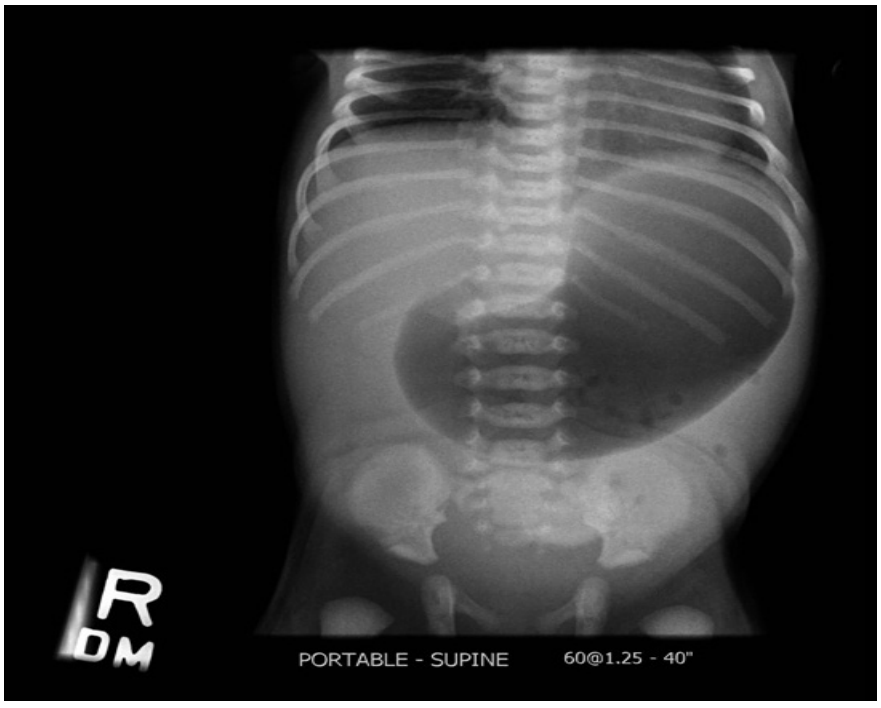
Gastroesophageal Reflux

The newborn has a relatively short esophagus, and about 50% of neonates will have benign reflux after feeding, the passing of gastric contents into the esophagus. The diagnosis may be suspected from the history or observing the infant feeding in the ED. Pathologic reflux or GERD is more common in premature infants and infants with congenital anomalies of the gastrointestinal tract and central nervous system.[14] Infants may present with failure to thrive, dysphagia, feeding aversion, hematemesis, irritability, opisthotonic posturing, coughing, apnea, or respiratory distress. Aspiration may result in pneumonitis, laryngospasm, or bronchospasm. There is some association of GERD with cow's milk allergy.[15] Parents of infants with GERD should be educated about proper feeding positions, decreasing volume and increasing frequency of feeds, and thickening feeds with rice cereal (1 tbsp for each 1-2 ounces of formula). An upright position for at least 20 minutes after feeding is recommended. Medications may be required if these measures are unsuccessful. The infants are often referred back to their pediatrician or to gastroenterology for follow up. Admission is recommended if the infant presents with severe symptoms such as apnea, respiratory distress, pneumonitis, or failure to thrive.

Pyloric Stenosis

Pyloric stenosis is a common cause of non-bilious, projectile vomiting in infants between 2-8 weeks of age. It occurs in 2-4 per 1000 births. The incidence is 2-5 times greater in males than females, and often the firstborn male. The use of macrolide antibiotics seems to be a contributing factor in some cases. Examination of the infant may be normal or may reveal a dehydrated infant. Rare cases may show visible peristalsis in the left upper quadrant and a palpable "olive" in the epigastrium, usually when the stomach is empty. Abdominal X-rays may reveal a distended stomach (*see Figure 7*), sometimes with peristaltic waves, the "caterpillar sign." Ultrasound is the diagnostic modality of choice in the ED.[16] (*See Figure 8.*) Muscle thickness > 3-4 mm and a pyloric channel length > 14-18 mm is suggestive of pyloric stenosis. A GI series, if obtained, will show delayed gastric emptying and the "string sign" of contrast passing through the narrowed

Figure 7. Distended Stomach in Pyloric Stenosis



pylorus, the "shoulder sign" — the hypertrophied pylorus imposed on the antrum, or a "double track line" created by contrast flowing through the pylorus. Baseline labs such as a CBC and electrolytes obtained in an infant with pyloric stenosis may show a hypokalemic metabolic alkalosis.

Fluid resuscitation with NS as indicated and then maintenance fluids is recommended. Surgical consult and surgical repair provide the definitive treatment.

Malrotation or Midgut Volvulus

Congenital malrotation of the midgut results in volvulus (twisting of a loop of bowel around its mesenteric attachment). The embryonic intestine rotates 270 degrees during week 5-8 of embryonic life. Failure to rotate or incomplete rotation results in incorrect mesenteric attachment of the intestine and the risk for volvulus. Infants with malrotation may have a wide variety of subtle clinical presentations including intermittent vomiting, constipation, and failure to thrive. More dramatic presentations include the sudden onset of bilious vomiting due to volvulus. Bilious vomiting in the neonate should prompt urgent evaluation for this condition. Initially, the child may appear well, but may rapidly progress to shock, hematochezia, and jaundice as necrotic bowel develops from the ischemia. Abdominal X-rays may be normal or may demonstrate a duodenal obstruction or the "double bubble sign" with a stomach and duodenal bubble with a paucity of gas in the rest of the abdomen. An upper GI series is the diagnostic test of choice. In the case of volvulus, the contrast does not cross to the left side and a "corkscrew" sign may be present due to twisting of the jejunum. Lab work should include a CBC, a complete metabolic panel, and type and screen. Dehydration and acidosis may be evident in the electrolytes. Fluid resuscitation should be initiated. A nasogastric tube should be placed and antibiotics (such as ampicillin and gentamicin) for enteric organisms should be administered. Urgent surgical consultation and admission to the hospital are necessary.

Figure 8. Pyloric Stenosis

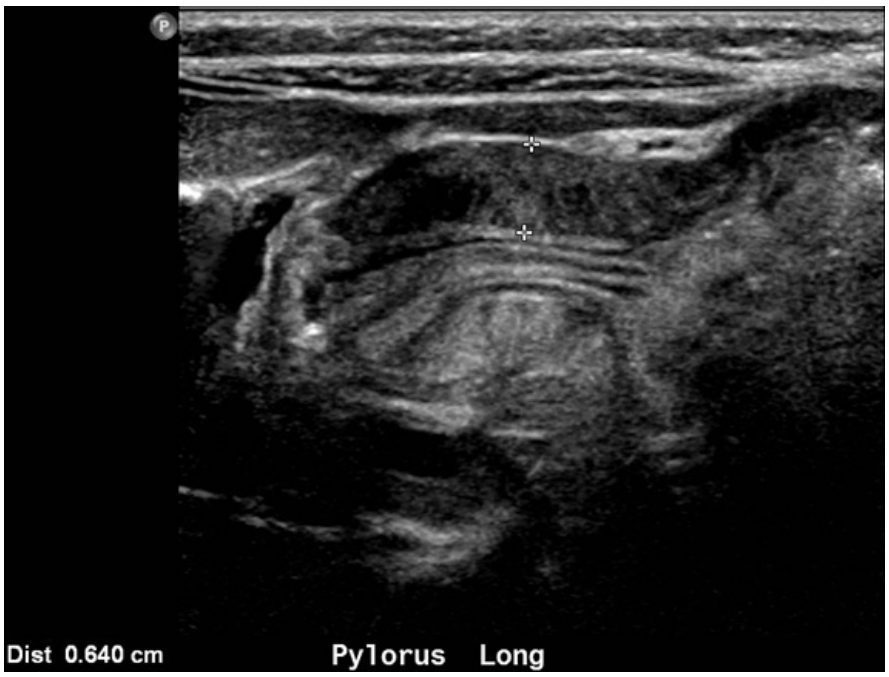
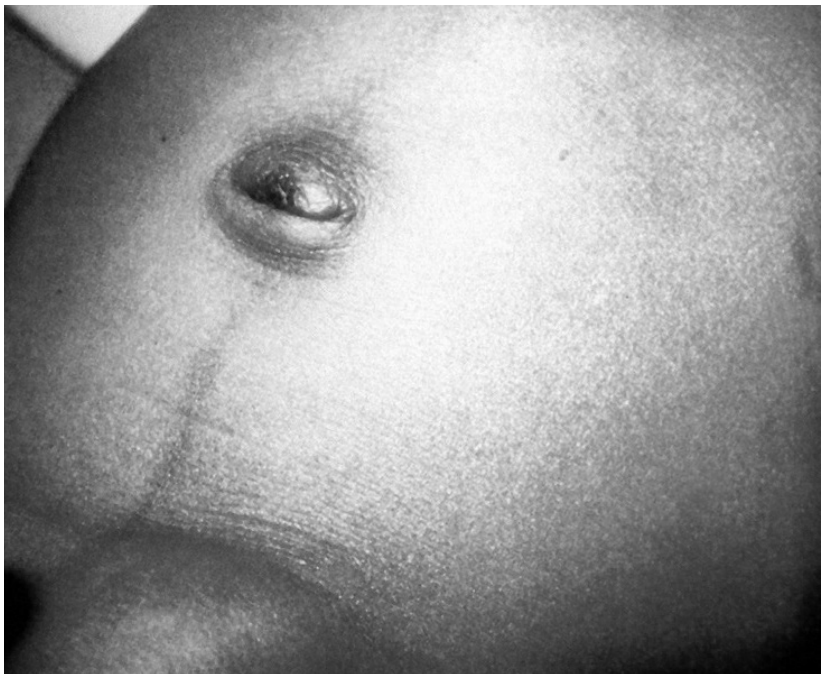


Figure 9. Hernia



Constipation/Hirschsprung's Disease

Decreased stooling or hard stools in the newborn has a wide variety of etiologies, with the majority due to the infant's diet. Formula-fed infants may stool less than breastfed infants, who may stool with each feed. Rectal stimulation or glycerin suppositories may relieve the constipation. Hirschsprung's

disease is suspected in any infant who fails to pass meconium spontaneously by 24-48 hours after birth. Often there is associated abdominal distention. The delayed passing of meconium may also be due to maternal diabetes, prematurity, or cystic fibrosis.[17]

Procedures used in the diagnosis of Hirschsprung's disease include:

- **Contrast enema:** May or may not show the characteristic transition zone in the newborn. Delayed X-rays after 24 hours may show retained contrast material.
- **Rectal biopsy:** This is used to confirm the diagnosis of Hirschsprung's. Characteristic biopsy findings include the absence of ganglion cells and hypertrophied non-myelinated axons.

Surgery consultation is required. A primary pull-through procedure or a temporary colostomy, if enterocolitis or inadequate decompression, may be performed. The definitive repair is done when the infant is stable. The infant often remains at risk for constipation, encopresis, and enterocolitis.

Incarcerated Inguinal Hernia

Inguinal hernias result from incomplete obliteration of the processus vaginalis during embryology. Bowel and peritoneal contents, including the testes or ovaries, may enter the inguinal canal and become trapped, resulting in incarceration. The prevalence is as high as 4.4% and more frequent in males, with a male to female ratio of 3:1 to 6:1. Females have a higher rate of incarceration. The rate of incarceration is 6-18% in the pediatric age group.[18] The typical infant presents with swelling in the inguinal area. (*See Figure 9.*) Parents often report a history of coughing or increased crying, which causes increased intra-abdominal pressure, followed by the development of the hernia. The infant with an incarcerated hernia may present with irritability, vomiting, abdominal distention, and decreased stools. Late signs of a strangulated hernia include bloody stools and lethargy. An infant with inguinal swelling, which is easily reduced, may be discharged with outpatient surgical follow-up. A hernia mass that is not easily reduced may require relaxation by non-pharmacological methods or by procedural sedation. The infant is placed in the Trendelenburg position and firm steady pressure applied to the hernia mass to guide it through the external inguinal ring. An urgent surgical consultation is necessary for a hernia mass that cannot be reduced or a hernia with suspected bowel necrosis. Diagnostic studies include abdominal X-rays, which may reveal air-fluid levels, or free air if perforation is present. (*See Figure 10.*) An ultrasound will help to distinguish a hernia mass from hydrocele, testicular pathology, tumor mass, or abscess. If bowel necrosis is suspected, the infant should be made NPO, aggressive fluid resuscitation initiated, and intravenous broad-spectrum antibiotics administered. Emergent surgery consultation is mandatory.

Jaundice

Jaundice is the yellowish discoloration of the skin and or sclera due to bilirubin deposition. In the newborn, jaundice is visible at bilirubin levels over 5 g/dL. Approximately 85% of term newborns and the majority of premature neonates develop clinical jaundice. The main source of bilirubin is from the breakdown of hemoglobin. Physiologic jaundice occurs in many newborns with a peak bilirubin level of about 12 mg/dL, peaking at day 3 to 5. Premature infants usually peak around day 5. Risk factors include low birth weight, prematurity, and breastfeeding. Non-physiologic jaundice is characterized by a bilirubin increase at a rate of > 5 mg/dL/24hr. The CDC uses an acronym for these risk factors (*see Table 7*).[16]

Unconjugated hyperbilirubinemia can cause bilirubin-induced neurologic dysfunction (BIND) when unconjugated bilirubin crosses the blood-brain barrier and binds to the tissue of the brain. The long-term effects of this is referred to as kernicterus. Treatment of unconjugated hyperbilirubinemia is aimed at preventing the neurologic sequelae. Several factors have contributed to the decrease in the incidence of kernicterus since the 1970s, including decreasing the incidence of neonatal Rh hemolytic disease by the administration of Rh immunoglobulin (RhoGAM) to mothers who are Rh negative and the introduction of phototherapy for the treatment of unconjugated hyperbilirubinemia. The number of exchange transfusions has also decreased.

Table 7. Risk Factors for Jaundice

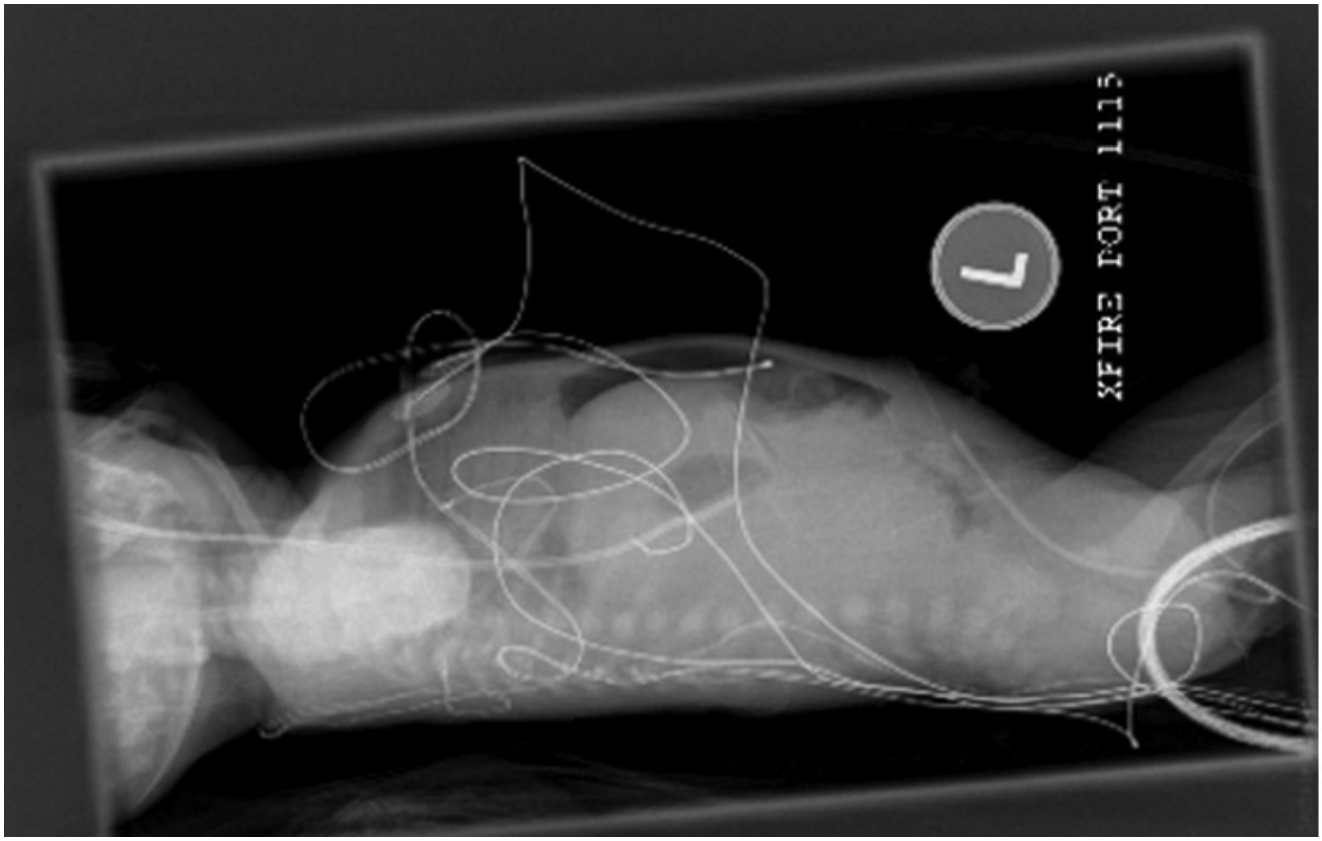
J	Jaundice in the first day of life
A	a sibling with neonatal jaundice
U	unrecognized hemolysis such as ABO or Rh incompatibility
N	non-optimal feeding
D	deficiency in G6PD or genetic disorder
I	infection
C	cephalhematoma or bruising
E	East Asian or Mediterranean descent

In 2004, the American Academy of Pediatrics' subcommittee on hyperbilirubinemia provided guidelines for the identification and treatment of infants at risk for severe hyperbilirubinemia. The guidelines pertained to infants > 35 weeks GA.[14] The recommendations include promotion of successful breastfeeding; performing a risk assessment for severe hyperbilirubinemia prior to discharge; and providing early follow-up and, when indicated, treatment of newborns with phototherapy or exchange transfusion to prevent BIND and long-term sequelae of kernicterus.

Therapeutic measures used in treating hyperbilirubinemia include phototherapy, exchange transfusion, and optimizing hydration of the neonate. The guidelines for the initiation of phototherapy are based on the hour-specific total bilirubin level, gestational age, and the presence of certain risk factors. The risk factors include the presence of isoimmune hemolytic disease, glucose-6 phosphate dehydrogenase (G6PD) deficiency, asphyxia, serum albumin levels < 3 g/dL, lethargy, temperature instability, acidosis, and sepsis. These factors increase the susceptibility of the brain to damage by bilirubin.

If total serum bilirubin is above the 95th percentile for age and approximating the level for phototherapy, lab work-up may include total and direct bilirubin; CBC to check for

Figure 10. Free Air in Abdomen



polycythemia or anemia from hemolysis; cord blood results for indirect Coombs and maternal blood type and Rh; infant's blood for blood type and Rh and direct Coombs; peripheral smear for RBC morphology (may detect hereditary spherocytosis) and reticulocyte count; G6PD screen with African, Asian or Mediterranean descent; and testing for liver disease, TORCH infections, metabolic disease, or sepsis when there is prolonged jaundice, especially increased levels of direct bilirubin. The general guidelines for initiation of phototherapy and exchange transfusion are based on the gestational age, level of bilirubin, and presence of risk factors.[19,20] Exchange transfusion is a method of removing bilirubin from the circulation when the infant has signs of neurologic dysfunction and aggressive phototherapy is inadequate. In the setting of isoimmune hemolysis, this is very helpful by removing the circulating antibodies and sensitized blood cells.

Infants requiring exchange transfusion should be admitted to the neonatal or pediatric intensive care unit. A type and crossmatch and placement of an umbilical catheter are necessary for the exchange transfusion. The blood should be irradiated and CMV safe. The circulating blood volume of an infant is about 80-90 mL/kg. A double volume exchange transfusion uses 160-180 mL/kg of cross-matched blood to replace about 85% of the infant's blood cells. This procedure reduces the total bilirubin by about 50%. In some circumstances of isoimmune hemolytic disease in which the bilirubin levels keep rising despite aggressive phototherapy, administration of IVIG (0.5-1 g) may be considered to avoid exchange transfusion. The complications of exchange transfusion include graft versus host disease, blood-borne infection, coagulopathy and thrombocytopenia, necrotizing enterocolitis, portal vein thrombosis, electrolyte abnormalities, and cardiac arrhythmias.

Conjugated Hyperbilirubinemia

Conjugated hyperbilirubinemia is the presence of direct bilirubin > 2 mg/dL and > 10% of total serum bilirubin. Causes of direct hyperbilirubinemia include hyperalimentation, biliary obstruction or atresia, choledochal cyst, hepatitis, sepsis, alpha-1 antitrypsin deficiency, hemoglobinopathies, cystic fibrosis, hypothyroidism, and inborn errors of metabolism. These infants should be admitted and investigations conducted to find the etiology. Potential treatments include phenobarbital, ursodiol, and fat-soluble vitamins.

Conclusion

An awareness of the many common neonatal issues that may present to the ED is critical to maximize the outcome for each infant. Recognizing the benign and identifying the potentially serious conditions of the neonate is important to the practice of emergency medicine.

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CME Questions

1. In infants with neonatal conjunctivitis, which of the following is true?
   A. Effective treatment for chlamydia conjunctivitis is erythromycin ophthalmic ointment.
   B. Effective treatment for chlamydia conjunctivitis is oral erythromycin 50 mg/kg/day in 4 divided doses for 14 days.
   C. Effective prophylaxis for chlamydia conjunctivitis is erythromycin ophthalmic ointment at birth.
   D. Culture of the exudate is the gold standard for chlamydia conjunctivitis.
2. All of the following are false *except*:
   A. a subgaleal hematoma can cause severe anemia and shock.
   B. a subgaleal hematoma cannot cross suture lines.
   C. a cephalhematoma can cross suture lines.
   D. a cephalhematoma is not associated with jaundice in the newborn.
3. An ALTE may include which of the following?
   A. Color change in the infant.
   B. An acute change in the infant's appearance that is frightening to the caregiver.
   C. A change in the infant's muscle tone, usually limp but can be rigid.
   D. Choking or gagging.
   E. All of the above.
4. Which of the following statements is false regarding the AAP guidelines for the identification and treatment of infants at risk for severe hyperbilirubinemia?
   A. The guidelines are for infants older than 32 weeks.
   B. Breastfeeding should be optimized.
   C. Early follow-up is recommended for at risk infants discharged from the nursery.
   D. Treatment with phototherapy and/or exchange transfusion is recommended when indicated to prevent kernicterus.
5. The radiographic features of pyloric stenosis include all of the following *except*:
   A. abdominal X-rays with a "double bubble" sign.
   B. abdominal X-rays may show a distended stomach.
   C. ultrasound of pylorus is the diagnostic method of choice and shows a thickness of > 3-4 mm and length >14-18 mm.
   D. upper GI series may show the "string sign."

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PEDIATRIC EMERGENCY MEDICINE **REPORTS**

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Common Neonatal Conditions

Neonatal Ophthalmologic Emergencies

Organism	Diagnosis	Treatment	Age of Presentation
Chlamydia trachomatis	Culture of conjunctival epithelium	Erythromycin 12.5 mg/kg PO every 6 hours x 14 days Efficacy is 80% Must check for concurrent pneumonia and treatment failure Repeat treatment if necessary	5-14 days
Neisseria gonorrhea	Culture on modified Thayer-Martin culture medium	Ceftriaxone 25 to 50 mg/kg (max 125 mg) IV or IM single dose or cefotaxime 100 mg/kg IV or IM single dose at birth Aggressive saline eye irrigation Ophthalmology consult	3-5 days
Herpes simplex virus	Viral culture Fluorescein staining to detect corneal defects	Acyclovir 20 mg/kg IV every 8 hours x 14 days and a topical ophthalmic agent Trifluridine 1% Ophthalmology consult	First 2 weeks of life
Haemophilus influenza, Staphylococcus aureus, Streptococcus viridans, Staphylococcus epidermidis, Escherichia coli, Streptococcus pneumoniae, Moraxella catarrhalis	Eye culture	Erythromycin or gentamicin ophthalmic ointment Topical sulfacetamide, trimethoprim-polymyxin B, or tobramycin	First few weeks
Pseudomonas aeruginosa	Eye culture	IV and topical aminoglycosides required Ophthalmology consult	First few weeks

Neonatal Seizures: Treatment Based on Etiology

Etiology	Risk Factors	Diagnosis	Treatment
Hypoglycemia	Infant of diabetic mother, small or large for gestational age, intrauterine growth retardation, inborn errors of metabolism	Glucose check < 45-50 mg/dL	Dextrose 10% (2 to 4 mL/kg) IV
Hyponatremia	Formula-fed infant, congenital adrenal hyperplasia, syndrome of inappropriate antidiuretic hormone due to hypoxic ischemic encephalopathy	Chemistry panel: Na (usually < 125)	Sodium chloride 3% IV 3 to 5 mL/kg over 1 hour for active seizures.
Hypocalcemia	Infant of diabetic mother, intrauterine growth restriction, perinatal asphyxia, prematurity, hypomagnesemia, hypoparathyroidism, high-phosphate formula, DiGeorge syndrome	Calcium level	Calcium gluconate 10% IV 1 to 2 mL/kg over 5-10 minutes May repeat x 1 Cardiac monitor
Hypomagnesemia	Hypocalcemia	Magnesium levels	Magnesium sulfate 12.5% IV 50 to 100 mg/kg over 1-2 hours to correct level < 1 to 6 mg/dL Cardiac monitor.
Inborn errors of metabolism Pyridoxine (B6)-responsive seizures Pyridoxal phosphate-responsive seizures	Dysmorphic features, strange odor, family history of inborn errors of metabolism.	Complete metabolic panel: decreased glucose, acidosis, increased ammonia level, increased lactate level, serum amino acids, urine amino acids, urine ketones, CSF lactate, pyruvate, glycine.	Non-ketotic hyperglycinemia is treated with sodium benzoate 250 to 750 mg/kg/day to decrease levels of glycine in combination with dextromethorphan 5 to 20 mg/kg/day Or memantine to block glycine effects on NMDA receptors
Pyridoxine (B6)-responsive seizures Pyridoxal phosphate-responsive seizures. Folinic acid-responsive seizures	Family history of inborn errors of metabolism, unexplained neonatal death in sibling, parental consanguinity	Complete metabolic panel: decreased glucose, acidosis, increased ammonia level, increased lactate level, serum amino acids, urine amino acids, urine ketones, CSF lactate, pyruvate, glycine.	Pyridoxine responsive seizures (i.e., deficiency of alpha AASA, PNPO deficiency) treated with pyridoxine 100 mg IV or 30 mg/kg PO May substitute pyridoxal phosphate 10 mg/kg IV or 30 mg/kg/day PO Some patients who don't respond to pyridoxine may respond to pyridoxal phosphate Folinic acid-responsive seizures treat with folinic acid 2.5 mg/kg/day IV
Neonatal Abstinence Syndrome (NAS)	Maternal drug use, poor prenatal care	Urine toxicology, meconium or hair assay. Symptoms in neonate such as jitteriness and seizures.	Swaddling, quiet low light environment, rocking For active seizures lorazepam 0.05 to 0.1 mg/kg/dose IV over 2-5 mins. Repeat if necessary Phenobarbital 20 mg/kg/dose IV Morphine, opium, paregoric are options utilized in different centers Avoid naloxone in infant with maternal methadone use. May precipitate seizure.

Causes of Neonatal Seizures

Hypoxic ischemic injury: perinatal asphyxia, focal infarction, or stroke
Infections: TORCH infections, CNS infections
Electrolyte abnormalities: hyponatremia, hypoglycemia, hypocalcemia, hypomagnesemia
Inborn errors of metabolism: amino acid disorders, organic acidurias, mitochondrial disorders, peroxisomal disorders, glucose transport (GLUT1) deficiency
Intracranial hemorrhage: intraventricular, parenchymal, subdural, subarachnoid
Congenital brain malformations: microcephaly, Sturge-Weber syndrome, tuberous sclerosis, neurofibromatosis
Pyridoxine (vitamin B6)-dependent seizure, folinic acid responsiveness syndromes
Neonatal abstinence syndrome: drug withdrawal from narcotics such as methadone, heroin, propoxyphene, or opiates
Kernicterus
Hypertension
Epilepsy syndromes: benign idiopathic neonatal seizures, benign familial neonatal seizures, early myoclonic epilepsy, Ohtahara syndrome (early infantile epileptic encephalopathy), or Coppola-Dulac syndrome (malignant migrating partial seizures in infancy)

Neonatal Seizures Anticonvulsant Treatment

Drug	Dose	Maintenance
Lorazepam	0.05 to 0.1 mg/kg/dose (max 2 mg) IV over 2-5 min May repeat in 10-15 min x 1 dose	
Diazepam	0.3 to 0.75 mg/kg/dose (max 2 mg) IV May repeat in 15-30 min x 2-3 doses	0.3 mg/kg/hour continuous infusion
Midazolam	0.1 to 1.1 mg/kg/hour	0.1 to 1.4 mg/kg/hour continuous infusion
Phenobarbital	20 mg/kg/dose IV May give additional 5 mg/kg doses in 15- to 30-min intervals Max total dose 40 mg/kg.	3 to 4 mg/kg day IV daily or divided BID Check levels
Phenytoin or fosphenytoin	20 mg/kg/dose IV Administer phenytoin slowly < 1 mg/kg/min to avoid cardiac arrhythmia or hypotension	3 to 4 mg/kg/day IV divided BID to 4 times daily Check levels
Levetiracetam (Keppra)	20 mg/kg/dose IV	10 mg/kg/day IV divided BID and titrate up to 40 mg/kg/day

Risk Factors for Jaundice

- **J** Jaundice in the first day of life
- **A** a sibling with neonatal jaundice
- **U** unrecognized hemolysis such as ABO or Rh incompatibility
- **N** non-optimal feeding
- **D** deficiency in G6PD or genetic disorder
- **I** infection
- **C** cephalhematoma or bruising
- **E** East Asian or Mediterranean descent

Supplement to *Pediatric Emergency Medicine Reports*, December 2014: "Common Neonatal Conditions." Authors: Audrey Bowen, MD, Florida Hospital, Orlando, FL; and Winslade Bowen, MD, FAAP, Florida Hospital Memorial Medical Center, Daytona, Beach, FL.

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