Contents lists available at ScienceDirect



American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Diagnostic

Is my baby normal? A review of seemingly worrisome but normal newborn signs, symptoms and behaviors



Zachary Drapkin, MD^{a,b,*}, Kathleen Franchek-Roa, MD^c, Ganga L. Srinivas, MBBS^c, Karen F. Buchi, MD^c, Michael J. Miescier, MD, MPH^a

^a Department of Pediatrics, Division of Pediatric Emergency Medicine, University of Utah, Salt Lake City, UT, USA

^b Department of Surgery, Division of Emergency Medicine, University of Utah, Salt Lake City, UT, USA

^c Department of Pediatrics, University of Utah, Salt Lake City, UT, USA

ARTICLE INFO

Article history: Received 8 November 2018 Received in revised form 18 March 2019 Accepted 20 March 2019

Keywords: Newborn Infant Neonate Infant behavior

ABSTRACT

Infant patients are a unique challenge to emergency department (ED) physicians as the spectrum of normal infant signs, symptoms and behaviors are often difficult to differentiate from abnormal and potentially lifethreatening conditions. In this article, we address some common chief complaints of neonates and young infants presenting to the ED, and contrast reassuring neonatal and young infant signs and symptoms against those that need further workup and intervention.

© 2019 Published by Elsevier Inc.

1. Introduction

Caring for newborns and young infants in the Emergency Department (ED) can present several unique challenges. Young infants present with pathology that is not encountered in other age groups and require unique diagnostic considerations and evaluations since severe pathology can often present with vague findings. Young infants may exhibit signs and symptoms that are worrisome to parents, but are actually normal for a child of that age. We present a review of several worrisome but normal signs and symptoms, and address how to distinguish these from potentially dangerous conditions.

2. Discussion

This review will focus on several worrisome but normal signs, symptoms and behaviors that may prompt parents to bring their child to the ED. We will address how to distinguish these normal behaviors and exam findings from potentially dangerous pathology.

2.1. My baby has a goopy eye

Causes of neonatal conjunctivitis, otherwise known as ophthalmia neonatorum, may be categorized as infectious or non-infectious. Infectious causes in the newborn require a full sepsis evaluation (blood, urine, and cerebrospinal fluid for analysis and culture), whereas most non-infectious causes can be managed as an outpatient (Table 1). Non-infectious causes

When silver nitrate was routinely used on all newborns, chemical conjunctivitis immediately after birth was common and self-limiting, lasting 48–72 h. However, newer eye ointments and drops are far less likely to cause a chemical conjunctivitis [1]. The scant yellow crusting caused by congenital nasolacrimal duct obstruction, also known as dacryostenosis, is distinguished from conjunctivitis by the absence of significant conjunctival injection (Fig. 1). Dacryostenosis is managed as an outpatient with warm compresses and gentle tear duct massage, [2] with referral to an ophthalmologist if it persists beyond 6 months of age.

A dacryocystocele is a fluid collection, often with a blue hue, in the inferomedial canthus resulting from an obstruction of the lacrimal duct (Fig. 2). This requires consultation with an ophthalmologist as it is estimated that 60% of these will become infected [3-5]. Nasolacrimal duct obstruction and/or dacryocystocele can progress to an acute dacryocysitis, which is an infected blocked tear duct characterized by warmth, redness, and the ability to express purulence from the punctum. Dacryocystitis in an infant requires a full sepsis evaluation,

[☆] We have no grants or funding to report.

^{☆☆} Meetings: This work has not been presented at any meetings to date.

^{*} Corresponding author at: University of Utah, Department of Pediatrics, Division of Emergency Medicine, 295 Chipeta Way, Salt Lake City, UT 84108, USA. *E-mail address:* zachary.drapkin@hsc.utah.edu (Z. Drapkin).

Table 1

Common causes of eye discharge in neonates

Cause of eye discharge	Description/presentation	Age of onset	Need for full sepsis evaluation	Management	Notes
Chemical Conjunctivitis	Non-purulent conjunctivitis	Hours after birth	No	None	Much less of an issue now that silver nitrate is no longer routinely used
Dacryostenosis	Scant yellow discharge without conjunctival injection	Within days to months after birth	No	Warm compresses and gentle tear duct massage	Referral to ophthalmologist if persists beyond 6 months of age
Dacryocystocele	Fluid collection with bluish hue in the inferomedial canthus	Variable	No unless progression to acute dacryocystitis	Referral to ophthalmologist	Estimated that up to 60% of these will become infected
Acute Dacryocystitis	Categorized by warmth, redness and purulent discharge from punctum	Variable	Full sepsis evaluation and systemic antibiotics	Admission to hospital with ophthalmology consultation	Often requires surgical intervention
Gonococcal Conjunctivitis	Copious purulent discharge	First 5 days of life	Full sepsis evaluation	Admission to hospital and systemic antibiotics	Can rapidly progress to corneal ulceration and globe rupture so begin saline eye washes in the ED
Chlamydial Conjunctivitis	Purulent discharge with conjunctival injection	5–14 days of life	Evaluate for Chlamydial pneumonia	Systemic macrolide	Chlamydial pneumonia may present up to the first 8 weeks of life
HSV conjunctivitis	Tearing with conjunctival injection. Often associated with vesicular skin lesions, dendritic corneal lesions, abnormal liver function tests or fever	Variable	Full sepsis evaluation	Admit to hospital, parenteral antibiotics until cultures negative, parenteral Acyclovir, anti-HSV topical drops	Potentially sight-threatening and can be associated with skin lesions, viremia, hepatitis and meningoencephalitis

systemic antibiotics, and hospital admission and ophthalmology consultation [5,6].

The usually benign, non-infectious causes of neonatal conjunctivitis must be distinguished from the infectious causes which can be vision and/or life threatening. Some of the most common infectious agents for neonatal conjunctivitis include gonorrhea, chlamydia, herpes simplex virus (HSV), staphylococcal species, streptococcal species and common viruses. Compared to the mild crusting seen in non-infectious neonatal conjunctivitis, infectious conjunctivitis can present with copious and persistent discharge and conjunctival injection [1].

All forms of infectious neonatal conjunctivitis should prompt an urgent ophthalmologic consultation because of the risk to vision, and an evaluation for systemic disease. Gonorrheal conjunctivitis typically presents within the first 5 days of life and is often associated with an impressive purulent discharge. Treatment is with topical and systemic antibiotics, usually after a full sepsis evaluation. As gonococcal conjunctivitis can rapidly progress to full thickness corneal ulceration and globe perforation, frequent eye irrigation with normal saline should be initiated in the ED. [1,6] In addition to bacteremia, gonococcal meningitis is a possibility. Topical and systemic antibiotics at meningitic doses are indicated. Chlamydial conjunctivitis usually presents in the first 5–14 days of life. Perinatally acquired chlamydial conjunctivitis may be associated with chlamydia pneumonia, which may also present in isolation up to the first 8 weeks of life. Treatment is with systemic macrolides [1,6]. HSV conjunctivitis is a potentially sight-threatening infection and can be associated with skin lesions, viremia and hepatitis, and meningoencephalitis. Clinicians should suspect this especially if there is an exposure to maternal HSV in the birth canal, neonatal fever, abnormal hepatic function, dendritic lesions on fluorescein staining of the cornea, or if the characteristic vesicles are present [1,6,7]. Appropriate gram-stain, cultures, and PCR can help distinguish between the different infectious causes of neonatal conjunctivitis [1].

2.2. My baby's belly button is not normal

Normally, the umbilical stump will turn a dark brown/black color and dry out prior to separating, typically within 1 week [8]. While dry cord care is appropriate for most hospital deliveries, the use of a topical antiseptic such as chlorhexidine, is recommended in resource poor settings [9]. Delayed cord separation does not have a formal definition, but most authors consider >3 weeks to be delayed which can be concerning for leukocyte adhesion deficiency [10-12].

With normal umbilical stump separation, there may be a foul "rotting fruit" smell. There may be a scant amount of yellowish discharge and a tinge of blood when separating from the non-erythematous umbilicus [13]. Parents who are concerned about discomfort with separation



Fig. 1. Crusting of the eyes due to dacryostenosis. Photo used with permission courtesy of Janelle Aby, MD (newborns.stanford.edu).



Fig. 2. Dacroyocystocele with bluish hue. Photo used with permission courtesy of Janelle Aby, MD (newborns.stanford.edu).

of the umbilical stump may also need reassurance that the umbilical cord is insensate.

The healing umbilical base can have a persistent moist raw fleshcolored tissue with clear discharge: this may represent an umbilical granuloma (Fig. 3). This can be treated with silver nitrate cauterization if not epithelialized several weeks after the cord has separated from the umbilicus [14,15]. If drainage of fluid persists after 1–2 months, evaluation with ultrasound for a patent urachus can be considered.

Reassurance to parents about umbilical stump concerns should be offered only if there are no signs of omphalitis, which is a medical and potentially surgical emergency. Any erythema around the umbilicus can be concerning for omphalitis, especially if there is streaking, warmth, swelling, purulent discharge and/or fever [16]. A dry umbilical stump can cause some erythema where it contacts the abdominal skin but this erythema should be without other signs of infection and limited to a distribution where the stump makes contact with the skin, and resolves rapidly with application of a barrier (Fig. 4). Omphalitis requires admission for systemic antibiotic therapy due to the risk of systemic infection given the proximity to the central circulation and the potential for progression to necrotizing fasciitis [16].

Umbilical hernias are common in childhood, as is diastasis recti. They rarely cause problems as long as they are soft and reducible. They tend to resolve in the first few years of life. Surgery is rarely performed before 3–5 years of age [17].

2.3. My baby is vomiting

While spitting-up is a normal infant behavior, it can be distressing to parents and must be distinguished from potentially dangerous causes of vomiting. Every infant will spit-up due to the small size of the stomach and the poor tone of the gastro-esophageal sphincter. As long as the infant is urinating normally, feeding, and growing, no intervention is needed. Spit-ups tend to peak at 4 months and resolve over the first year of life [18,19]. Overfeeding can present with increased spit-ups due to the small stomach size of infants. Obtaining a careful history can distinguish spitting-up, which tends to be a small amount and effortless, from forceful vomiting.

Unlike spitting-up, vomiting that is forceful or projectile typically has an underlying etiology. Projectile non-bilious emesis after feeds in an infant with a voracious appetite in the first 12 weeks of life suggests pyloric stenosis, and is diagnosed by ultrasound [20,21]. This is often associated with a hypochloremic, hypokalemic metabolic alkalosis [20,21]. Inborn errors of metabolism can present with emesis and are often associated with an abnormal newborn screen and/or abnormal metabolic laboratory studies such as hypoglycemia, hyperammonemia, and lactic acidosis [22]. Necrotizing enterocolitis (NEC) can also present with emesis and often abdominal distention, or bloody stools. Pneumatosis can be seen on abdominal radiograph [22]. While 90% of infants with NEC have a history of prematurity, term infants can also



Fig. 3. Umbilical granuloma. Photo used with permission courtesy of Daniel Stulberg, MD.



Fig. 4. Erythema due to dry cord care without evidence of omphalitis. The brown/black color of the umbilical stump is also normal appearing. Photo used with permission courtesy of Janelle Aby, MD (newborns.stanford.edu).

present with NEC. These term infants often present within the first few days of life and often have other risk factors such as sepsis, polycythemia, low birth weight, or congenital heart disease [23-25]. Presentation of NEC can be delayed beyond the first month of life, especially when there are other abnormalities such as congenital heart disease [23]. Bilious emesis, or emesis with a green or bright yellow color, may occur with malrotation and/or midgut volvulus, which is a surgical emergency and can be evaluated with an upper GI series and surgical consultation [26]. Bilious emesis can also be seen with intestinal atresia, intestinal webs, or meconium ileus [22]. While swallowed maternal blood from cracked nipples is a common cause of hematemesis and warrants a breast exam of breastfeeding mothers, hematemesis may also have a more serious etiology such as submucosal mass, vascular malformation, hemobilia, vitamin K deficiency, variceal bleeding, or trauma [27]. Abusive head and/or abusive abdominal trauma remains a consideration in every neonate with vomiting [22].

2.4. My baby is not stooling normally

Parents are often concerned about the color, texture, frequency, and pain associated with stooling. Similar to adults, neonates may grunt or strain with stooling, and this by itself does not necessarily indicate constipation. Infant dyschezia, or straining and crying for 10 or more minutes before passage of soft stool in a healthy infant, is benign and thought to be caused by poor coordination of the muscles that are used to defecate. Infant dyschezia usually resolves by 6 months of age [28,29].

Meconium stools are very dark green, tarry-looking stools and should transition to normal stools by day of life 3–5. If meconium stools persist after 5 days of life, then maternal milk production and infant milk intake should be assessed. After the first 5 days, an infant's stool is often a seedy, mustard-color for breastfed infants, but tends to have more of a greenish color in formula-fed infants [30]. Infants with acholic stools should be evaluated for obstructive processes such as biliary atresia [31] as well as causes of neonatal hepatitis, inborn errors of metabolism and congenital infections [32,33]. Bloody stools or black stools after transition from meconium should include an evaluation for gastrointestinal bleeding, which may include swallowed maternal blood, milk protein allergy, and anorectal fissure. There are however potentially more serious causes of lower gastrointestinal bleeding such as obstruction, necrotizing enterocolitis, Hirschprung disease and associated enterocolitis, volvulus, and vitamin K deficiency. These diseases can be diagnosed by targeted laboratory and imaging studies [27,34]. Obstruction and necrotizing enterocolitis can be evaluated with abdominal radiographs; [27] Hirschprung disease can be evaluated with a barium enema but the gold standard is a biopsy showing absence of ganglion cells in the colonic submucosa; [35] and volvulus can be evaluated with an upper gastrointestinal series [36]. For vitamin K deficiency bleeding, parents should be asked if the infant received a vitamin K injection at birth.

An infant's frequency of stooling is a common cause for parental distress. In the first few days of life, after the breastmilk supply is established, infants usually stool with each feeding. Infrequent stools in the first 10 days of life and/or failure to pass meconium in the first 48 h are concerning for cystic fibrosis, Hirschprung disease and other neurogenic causes of delayed colonic transit. Over days to weeks, there can be a dramatic change in frequency: formula fed infants may stool several times a day or may go more than a day without stooling; breastfed infants may stool several times a day or may stool as infrequently as once every few days and all these patterns may still be considered normal if the stools are still soft [30,37-41]. Constipation in infants, therefore, is defined by texture (hard stools) rather than frequency. While an infant does not need to stool every day, constipation is a heralding sign of infantile botulism and often precedes other signs of infantile botulism such as weakness, poor feeding, weak suck, ptosis and eventual respiratory failure and paralysis [42].

Another concerning genitourinary finding for parents is the orange tinged urine that can be found with urate crystals. Urate crystals appear as a pink to orange color in the urine and may be mistaken for blood [43]. Urate crystals are often seen in breastfed infants where the mother's milk is delayed and can be normal in the first 5 days of life, after which evaluation for adequate nutritional intake is warranted [40].

2.5. What's going on under my baby's diaper?

Hormonal fluctuations in the neonatal period can result in changes in the genitourinary area that are concerning to parents. Female infants may experience scant vaginal bleeding due to hormonal withdrawal between the third and seventh day of life [44]. Likewise, a prominent hymenal tag may be present at birth, and will regress as estrogen levels decrease [45].

Fluctuations in hormones can lead to other changes in infants as well. Notably, higher prolactin levels may lead to the appearance of breast buds in the first week in both male and female infants. These breast buds are normal and may even be associated with a scant discharge known as witch's milk, and usually resolve by 4 weeks. Infants with an increase in size of breast buds or new onset of breast buds after 2 months should be evaluated for endocrine abnormalities [46].

The appearance of the circumcision site can be a concern for parents of male infants. Despite the erythema and yellow film that commonly accompanies normal healing, infection is rare, ranging from 0.01 to 0.06% [47]. Mild inflammation limited to the glans can be treated with topical antibiotic as most of these represent swelling and fibrinous exudates that represent normal healing. If the glans and/or shaft of the penis has increasing edema, erythema and warmth, an infection should be considered and a full sepsis evaluation, systemic antibiotics, and consultation with a pediatric urologist should be initiated [48]. The foreskin of a neonatal uncircumcised penis does not need to be retracted for cleaning [49].

It is important to distinguish undescended testes from retractile testes: retractile testes can be brought into the scrotum by the examiner and will stay in the scrotum until a cremasteric reflex occurs, whereas an undescended testes will immediately retract out of the scrotum when released [50]. While hydroceles are a common finding in newborn males and the majority will resolve during the first year of life, [51] a tense hydrocele warrants ultrasound as it may need surgical management and may represent in-utero torsion with absorption of the testis [52]. Unlike umbilical hernias, all infants with either an inguinal hernia on exam or a history consistent with a reduced inguinal hernia need prompt surgical follow-up due to the potential for incarceration [53,54].

2.6. My baby is not moving normally

Parents are often concerned about the way that their newborn appears to move. Distinguishing normal newborn movements from neurologic problems can be difficult given the subtle differences between normal newborn movements and abnormal movements caused by seizures, infantile spasms, drug withdrawal/intoxication, or non-accidental trauma (Table 2).

Normal newborn jitteriness tends to occur with state variation (sleep to arousal) or external stimulus. It is a "fine tremor" that is generally <3 cm and >6 Hz and is not associated with altered level of consciousness or abnormal gaze/eye movements. Reassuring features of jitteriness include resolution of movement when the limb is held or the infant is sucking. Episodes become rarer and require more stimulation over time as the condition resolves in the first few months of life [55-59].

Benign neonatal sleep myoclonus can be confused with seizures. Benign neonatal myoclonus presents as repetitive myoclonic jerks that cease when the baby is awakened. They can be exacerbated by sound and usually appear in the early phases of sleep. Onset is typically in the first 2–4 weeks of life with resolution by 6 months of age. While persistent myoclonus during sleep warrants investigation, occasional myoclonic jerks is considered normal [59,60]. Bilateral ankle clonus can be normal up until 8 months as long as it is a few beats, not sustained, and the child is developing normally [61].

While many children have benign causes of jitteriness and myoclonic jerks, these must be distinguished from movement disorders that can affect newborns and infants. Abnormal jitteriness can present at rest without either internal or external stimulus and the amplitude does not diminish with time. Arms and legs may also be involved rather than just the hands and feet. Important diagnostic considerations for abnormal jitteriness and exaggerated neonatal reflexes include hypoglycemia, hypocalcemia, hypomagnesemia, tetany, sepsis, drug withdrawal, and central neurologic processes [55,56]. The onset of withdrawal from neuroactive drugs used during pregnancy varies according to the class of drug used. Symptoms can be delayed for days to weeks for long-acting opiates such as methadone and for selective serotonin reuptake inhibitors. As such, the first signs of drug withdrawal such as irritability, increasing tremors, vomiting, diarrhea, rhinorrhea, poor feeding, temperature instability, hypertonia, diaphoresis or seizures may be in the ED rather than the newborn nursery [62].

Seizures can have a variety of presentations in newborns. These can include abnormal repetitive movements that cannot be suppressed and abnormal eye movements, with or without altered mental status. All abnormal neonatal movements that are not explained by neonatal jitteriness or neonatal sleep myoclonus warrant a further evaluation. Generalized tonic-clonic seizures in neonates are rare because the neuronal connections are not sufficiently developed [63]. In contrast to older children, infants <3 months old with new onset seizures often have underlying causes that can be detected by laboratory and/or neuroimaging studies that would change acute management in the ED. [64-66] American Academy of Neurology guidelines allow for non-urgent outpatient magnetic resonance imaging study (MRI) for a child <1 year old with new onset seizure and allow for labs/lumbar puncture at physician discretion as long as the child does not have a postictal focal deficit and has returned to baseline within several hours [67]. However, other studies focusing on children <6 months have found high rates of actionable findings on laboratory studies and neuroimaging, and MRI, the preferred imaging modality, is often not available in the ED. [66,68] Therefore, for infants <3 months presenting with a new seizure in the ED, a non-contrast head CT should be strongly considered to evaluate for congenital abnormalities evidence of abusive head trauma. Labs should include electrolytes, glucose, ammonia, acid-base studies, and lactate to evaluate for inborn errors of metabolism; a full sepsis evaluation should be strongly considered to evaluate for bacterial and viral meningitis, especially in infants who are febrile, have a concerning birth history, or who have not returned to baseline.

Table 2

Benign infant movements that may be mistaken for pathology

Movement	Description/presentation	Age of onset/resolution	Dangerous mimics
Jitteriness	Fine tremor, <3 cm, >6 Hz, suppressible, normal level of consciousness, tend to occur with sleep-wake arousal or external stimulus	Onset typically at birth, resolves by first few months	-Seizures -Drug withdrawal -Electrolyte abnormalities -Tetany -Sepsis
Benign neonatal sleep myoclonus	Few repetitive myoclonic jerks while sleeping that resolve when awake, usually in early phase of sleep	Onset typically at 2–4 weeks, resolves by 6 months	-Seizures -Drug withdrawal -Electrolyte abnormalities -Tetany -Sepsis
Saccadic intrusions	Small amplitude conjugate interruption of smooth eye pursuit while trying to track without slow/fast phase	Onset typically at birth, resolves by first few months	-Nystagmus -Blindness -Seizures -Neurologic dysfunction -Opsoclonus
Gaze asymmetry	Disconjugate gaze at initiation of conjugate focus	Onset typically at birth, resolves by 4 months	-Cranial nerve palsy -Orbital/intracranial tumor -Neuromuscular disorder -Increased intracranial pressure

One type of seizure that is unique to infants and commonly has a delayed diagnosis is infantile spasms. Infantile spasms often occur in a cluster of spasms, usually just following arousal. They consist of sudden brief contractions of multiple muscle groups followed by a longer tonic phase. The child often cries after these spasms occur. The peak age for onset is 4–8 months but they can present earlier. Infantile spasms are often associated with developmental arrest and regression, both of which are concerning features that should prompt further evaluation. Infantile spasms can be associated with long term problems such as developmental delays, and persistent seizures [69,70]. Early diagnosis and intervention is associated with improved outcomes [71-73].

Eye movements can appear abnormal in infants as well. As children learn to stay fixed on a moving object, they may demonstrate saccadic intrusions that interrupt smooth pursuit eye movements. These saccadic movements can be normal as long as the infant is still trying to track and there does not appear to be a slow and fast phase as in true nystagmus. These interruptions of smooth pursuit tend to diminish over the first few months of life [74,75]. Eye movements that do not fit this description, such as abnormal eye movements at rest, nystagmus with a fast and slow phase, or roving eye movements (slow aimless movements of the eyes which can be an early presentation of blindness) should be referred to a pediatric ophthalmologist [74]. Gaze asymmetry at initiation of conjugate focus can be normal up until 4 months. Over time, infants should be able to focus on a face, and by 2 months, track across 180 degrees without auditory cues. An infant with strabismus that persists beyond 4 months should be evaluated by a pediatric ophthalmologist [76].

2.7. My baby is breathing funny

Periodic breathing may be concerning to parents and must be distinguished from apnea. Periodic breathing is seen in a comfortable appearing neonate without tachypnea or cyanosis, with pauses in breathing that last <10 s [73]. Periodic breathing is normal and is the reason that practitioners listen to a child breathe for 60 s when checking a respiratory rate as pauses in periodic breathing may be followed by a transient rapid succession of breaths. Apnea, which is defined as a pause in respirations of >20 s [77] is concerning. Causes of apnea unique to infants include bronchiolitis and viral respiratory infections (particularly in children <44 weeks post gestational age) [78], pertussis, meningitis, non-accidental trauma, obstructive apnea, seizure, and neuromuscular weakness. Detailed guidelines for brief resolved unexplained events (BRUE) have been published [79].

Noisy breathing is also common in infants as well. Laryngomalacia is the most common congenital laryngeal abnormality and is a common cause of stridor in infants. Symptoms typically develop over the first 2 weeks of life and tend to peak at 2 months and resolve by 12–-24 months. As long as symptoms are mild and intermittent and the infant is feeding and growing well, this condition can be monitored by a pediatrician. Stridor at rest, when the infant is supine, and that seems to worsen with age instead of improving should be investigated [80]. Factors on history than can be worrisome include history of neck or mediastinal surgery which is concerning for paretic vocal cord; history of prolonged intubation which may suggest subglottic stenosis; stridor along with a cutaneous hemangioma which may suggest subglottic hemangioma; acute stridor along with infectious symptoms which may represent croup, epiglottitis, and/or bacterial tracheitis [80].

2.8. My baby's skin is a funny color

All babies have some degree of cyanosis at birth and this cyanosis should resolve within first 10 min of life [81]. Acrocyanosis, or a bluish discoloration of hands and/or feet, can be normal within the first few days of life as long as there is no central cyanosis or signs of respiratory distress [40,82]. Other causes of peripheral cyanosis include cold exposure and increased oxygen extraction from shock or sepsis [83]. Central cyanosis is defined as a dusky appearance of the body and the mucous membranes and is always concerning beyond the first 10 min of life. The main causes of central cyanosis can be categorized as problems with respiration, problems with ventilation, congenital heart abnormalities, and hemoglobinopathies. A chest radiograph, four extremities blood pressure and oxygen saturations, the hyperoxia test, or obtaining an arterial blood gas after the baby is on 100% oxygen can help distinguish between these causes [83,84].

Another skin discoloration that can be concerning is neonatal jaundice. It is most commonly an unconjugated hyperbilirubinemia caused by a combination of increased bilirubin production and decreased bilirubin clearance due to immature conjugation. Current American Academy of Pediatrics recommendations are that after discharge from the hospital, measurement of bilirubin should be performed if the jaundice appears excessive for the child's age with the caveat that visual estimation of bilirubin levels can be inaccurate [85]. While all infants have some level of physiologic jaundice, proper identification and management of severe cases is crucial as infants may develop bilirubininduced neurological dysfunction or progress to acute bilirubin encephalopathy (kernicterus). Physicians need to combine knowledge of the patient's risk factors, the infant's age in hours, and a bilirubin level in order to determine if the infant needs follow-up, phototherapy, or an exchange transfusion [85]. It is important to note that while neonatal jaundice is common and most often requires no intervention, there are other causes of jaundice that must be further evaluated. Jaundice beyond the first three weeks of life should be evaluated with a measurement of total and fractionated bilirubin as well as a review of the newborn metabolic screen. While prolonged jaundice can be a manifestation of breast milk jaundice, this is a diagnosis of exclusion. Prolonged high elevations of unconjugated bilirubin may be the result of glucuronyl transferase deficiency such as in Crigler-Najjar syndrome or Gilbert's syndrome, or due to excess hemolysis from blood group incompatibility or G6PD deficiency. Conjugated hyperbilirubinemia in infants should be concerning for metabolic abnormalities, hypothyroidism, galactosemia, sepsis, hepatitis, and obstructive problems such as biliary atresia, choledochal duct cysts, duodenal webs [86].

2.9. My baby has a bruise

Distinguishing between potentially dangerous and benign skin lesions can be challenging. While a young infant with bruising or burn marks warrants an evaluation for child abuse since young infants are too young to roll and injure themselves, several skin lesions can be mistaken for bruising or burn marks. Congenital dermal melanocytosis ("Mongolian spots") are flat, purple to blue colored lesions that are most frequently located in the sacral area and do not fade or rapidly progress through color changes like bruising [87-89]. An infant who is lying down may develop sudden erythema of the dependent side of the body known as a harlequin color change that may last for several minutes afterwards and may continue up to 3 weeks of life [90]. Capillary hemangiomas can be mistaken for bruising as they often have a violaceous base-these lesions usually appear during the first few weeks of life and rapidly grow before eventual involution. Unlike bruises they blanch with pressure [88]. Bullous impetigo is an infectious disorder caused by Staphylococcus aureus which begins as vesicles that coalesce into large bullae that eventually rupture and form a well demarcated annular area with marked erythema that may develop secondary scaling and crusting. A pathognomonic finding for bullous impetigo is a "collarette sign," or a ring of scale around the ruptured lesions [91,92]. These lesions may be mistaken for burn marks [89,91]. Unlike burns, these lesions follow a distinct progression of vesicles coalescing into bullae and then rupturing and crusting-parental photographs may help demonstrate this progression. Bullous impetigo lesions should be cultured; mild lesions can be treated with topical antibiotics while more severe or generalized lesions or children with fever or other systemic signs are treated with topical and systemic antibiotics after a complete sepsis evaluation [91-93].

2.10. My baby has spots

While there are many skin findings that are unique to young infants [87,90], one particular challenge for ED physicians is distinguishing potentially dangerous pustules and vesicles from benign lesions. In addition to recognizing potentially dangerous infectious causes of pustular lesions, ED physicians should be familiar with common benign causes of pustular and popular lesions. Erythema toxicum neonatorum is common and tends to present as crops of small papules and pustules surrounded by a large inflammatory wheal that appears on the first few days of life and resolves spontaneously by two weeks of life [90,93]. Transient neonatal pustular melanosis is present at birth and is more common among black infants; these lesions presents as pustules without surrounding erythema that rupture easily leaving behind a hyperpigmented macule that fades within several weeks [90]. Neonatal acne presents as erythematous papules and pustules and may be associated with open and closed comedones; these lesions tend to be isolated to the face and resolve within the first four months of life [90]. Milia and miliaria are common small white to yellow colored papules without an erythematous base and are caused by retention of keratin and sweat respectively [90].

These benign pustular lesions must be distinguished from potentially dangerous infectious causes of pustules. Staphylococcal pustulosis can present as pustular lesions over an erythematous base [94,95] and requires treatment with systemic antibiotics with methicillin resistant *Staphylococcus aureus* coverage. Culture and gram stain of these lesions can be helpful for confirming diagnosis. If fever or systemic illness is present, a full sepsis evaluation should be performed prior to initiation of antibiotics [94,96]. HSV can manifest as groups of vesicles on an erythematous base; neonates suspected of having HSV need a full sepsis evaluation and need to be started on antiviral therapy while awaiting confirmatory testing [93].

3. Conclusion

Newborns can have a variety of signs, symptoms, and behaviors that may appear worrisome to parents, but are within the range of normal for their age. EM physicians need to be familiar with these normal variations of newborn findings in order to distinguish them from potentially dangerous pathology and appropriately reassure parents.

References

- Teoh DL, Reynolds S. Diagnosis and management of pediatric conjunctivitis. Pediatri Emerg Care 2003. https://doi.org/10.1097/00006565-200302000-00014.
- [2] Takahashi Y, Kakizaki H, Chan WO, Selva D. Management of congenital nasolacrimal duct obstruction. Acta Ophthalmol 2010. https://doi.org/10.1111/j.1755-3768.2009. 01592.x.
- [3] Wong RK, VanderVeen DK. Presentation and management of congenital dacryocystocele. Pediatrics 2008. https://doi.org/10.1542/peds.2008-0934.
- [4] Paysse EA, Coats DK, Bernstein JM, Go C, de Jong AL. Management and complications of congenital dacryocele with concurrent intranasal mucocele. J AAPOS 2000. https://doi.org/10.1016/S1091-8531(00)90011-9.
- [5] S. P-S, M. S, T.-X. L, A. G, B. B, B. V, et al. Dacryocystitis: systematic approach to diagnosis and therapy. Curr Infect Dis Rep 2012. doi:https://doi.org/10.1007/s11908-012-0238-8
- [6] Wong MM, Anninger W. The pediatric red eye. Pediatr Clin North Am 2014. https:// doi.org/10.1016/j.pcl.2014.03.011.
- [7] Kimberlin DW, Brady MT, Jackson MA, Long S. Report of the committee on infectious diseases. American Academy of Pediatrics 2018;2018 (Herpes Simplex).
- [8] Bhalla JN, Nafis N, Rohatgi P, Singh J. Some observations on separation of the umbilical stump in the newborn. Indian J Pediatr 1975;42:329–34.
- [9] Stewart D, Benitz W. Umbilical cord care in the newborn infant. Pediatrics 2016. https://doi.org/10.1542/peds.2016-2149.
- [10] Oudesluys-Murphy AM, Eilers GAM, de Groot CJ. The time of separation of the umbilical cord. Eur J Pediatr 1987. https://doi.org/10.1007/BF00444944.
- [11] Hayward AR, Leonard J, Wood CBS, Harvey BAM, Greenwood MC, Soothill JF. Delayed separation of the umbilical cord, widespread infections, and defective neutrophil mobility. Lancet 1979;313:1099–101.
- [12] Wilson CB, Ochs HD, Almquist J, Dassel S, Mauseth R, Ochs UH. When is umbilical cord separation delayed? J Pediatr 1985;107:292–4.
- [13] Janssen PA, Selwood BL, Dobson SR, Peacock D, Thiessen PN. To dye or not to dye: a randomized, clinical trial of a triple dye/alcohol regime versus dry cord care. Pediatrics 2003;111:15–20.
- [14] SS SHETH, MALPANI A. The management of umbilical granulomas with cryocautery. Am J Dis Child 1990;144:146–7.
- [15] Chamberlain JM, Gorman RL, Young GM. Silver nitrate burns following treatment for umbilical granuloma. Pediatr Emerg Care 1992;8:29–30.
- [16] Fraser N, Davies BW, Cusack J. Neonatal omphalitis: a review of its serious complications. Acta Paediatr 2006;95:519–22.
- [17] Kelly KB, Ponsky TA. Pediatric abdominal wall defects. Surg Clin 2013;93:1255–67.
 [18] Nelson SP, Chen EH, Syniar GM, Christoffel KK, Group PPR. One-year follow-up of symptoms of gastroesophageal reflux during infancy. Pediatrics 1998;102(6):e67.
- [19] Nelson SP, Chen EH, Syniar GM, Christoffel KK. Prevalence of symptoms of gastroesophageal reflux during infancy: a pediatric practice-based survey. Arch Pediatr Adolesc Med 1997;151:569–72.
- [20] MacMahon B. The continuing enigma of pyloric stenosis of infancy a review. Epidemiology 2006;195–201.
- [21] Hernanz-Schulman M. Infantile hypertrophic pyloric stenosis. Radiology 2003;227: 319–31.
- [22] Chandran L. Vomiting in children. Pediatr Rev 2008;29:183.
- [23] Maayan-Metzger A, Itzchak A, Mazkereth R, Kuint J. Necrotizing enterocolitis in fullterm infants: case-control study and review of the literature. J Perinatol 2004;24: 494.
- [24] Ostlie DJ, Spilde TL, St Peter SD, Sexton N, Miller KA, Sharp RJ, et al. Necrotizing enterocolitis in full-term infants. J Pediatr Surg 2003;38:1039–42.

- [25] Lambert DK, Christensen RD, Henry E, Besner GE, Baer VL, Wiedmeier SE, et al. Necrotizing enterocolitis in term neonates: data from a multihospital health-care system. J Perinatol 2007;27:437.
- [26] Millar AJW, Rode H, Cywes S. Malrotation and volvulus in infancy and childhood. Semin Pediatr Surg, vol. 12, Elsevier; 2003, p. 229–36.
- [27] Boyle JT. Gastrointestinal bleeding in infants and children. Pediatr Rev 2008;29:39.
 [28] Hyman PE, Milla PJ, Benninga MA, Davidson GP, Fleisher DF, Taminiau J. Childhood functional gastrointestinal disorders: neonate/toddler. Gastroenterology 2006;130:
- 1519–26. [29] Hyman PE, Cocjin J, Oller M. Infant dyschezia. Clin Pediatr (Phila) 2009;48:438–9.
- [30] den Hertog J, van Leengoed E, Kolk F, van den Broek L, Kramer E, Bakker E-J, et al. The defecation pattern of healthy term infants up to the age of 3 months. Arch Dis Childhood-Fetal Neonatal Ed 2012;97:F465–70.
- [31] McKiernan PJ. Neonatal cholestasis Semin Neonatol, vol. 7, Elsevier; 2002, p. 153-65.
- [32] Lai M-W, Chang M-H, Hsu S-C, Hsu H-C, Su C-T, Kao C-L, et al. Differential diagnosis of extrahepatic biliary atresia from neonatal hepatitis: a prospective study. J Pediatr Gastroenterol Nutr 1994;18:121–7.
- [33] Chang M-H, Hsu H-C, Lee C-Y, Wang T-R, Kao C-L. Neonatal hepatitis: a follow-up study. J Pediatr Gastroenterol Nutr 1987;6:203–7.
- [34] Fox VL. Gastrointestinal bleeding in infancy and childhood. Gastroenterol Clin North Am 2000;29:37–66.
- [35] de Lorijn F, Kremer LCM, Reitsma JB, Benninga MA. Diagnostic tests in Hirschsprung disease: a systematic review. J Pediatr Gastroenterol Nutr 2006;42:496–505.
- [36] Applegate KE. Evidence-based diagnosis of malrotation and volvulus. Pediatr Radiol 2009;39:161–3.
- [37] WL NYHAN. Stool frequency of normal infants in the first week of life. Pediatrics 1952;10:414–25.
- [38] Pediatrics AA of. Caring for your baby and young child: Birth to age five. Bantam; 2004.
- [39] Hagan JF, American Academy of Pediatrics, Shaw JS, Duncan PM. Bright Futures : Guidelines for Health Supervision of Infants, Children, and Adolescents. Vol Fourth editionElk Grove Village, IL: American Academy of Pediatrics; 2017. http://search. ebscohost.com/login.aspx?direct=true&db=nlebk&AN=1471774&site=ehostlive, Accessed date: 28 March 2019.
- [40] McInerny TK, Adam HM, Campbell DE, Foy JM, Kamat DM. AAP textbook of pediatric care. Am Acad Pediatr 2016:757.
- [41] Biggs WS, Dery WH. Evaluation and treatment of constipation in infants and children. Am Fam Physician 2006;73.
- [42] M.A. C-M. Botulism. Pediatr Rev 2016. https://doi.org/10.1542/pir.2015-0018.
 [43] Jeng JY, Franz WB. Orange stains in a healthy neonate's diaper. Clin Pediatr (Phila)
- 2014. https://doi.org/10.1177/0009922814536777.
 [44] Huber A. The frequency of physiologic vaginal bleeding of newborn infants. Zentralbl Gynakol 1976;98:1017–20.
- [45] Sugar NF, Graham EA. Common gynecologic problems in prepubertal girls. Pediatr Rev 2006:27:213–23.
- [46] Madlon-Kay DJ. "Witch's milk". Galactorrhea in the newborn. Am J Dis Child 1986; 140:252–3.
- [47] Circumcision TFO. Male circumcision. Pediatrics 2012;130(3):756-85 (peds-2012).
- [48] Baskin LS, Canning DA, Snyder HM, Duckett JW. Treating complications of circumcision. Pediatr Emerg Care 1996. https://doi.org/10.1097/00006565-199602000-00018.
- [49] Osborn LM, Metcalf TJ, Mariani EM. Hygienic care in uncircumcised infants. Pediatrics 1981;67:365–7.
- [50] Docimo SG, Silver RI, Cromie W. The undescended testicle: diagnosis and management. Am Fam Physician 2000;62(9):2037–44.
- [51] Cooper CS, Kirsch AJ, Snyder III HM. Early diagnosis and management of neonatal abdominoscrotal hydroceles. Urol Int 1999. https://doi.org/10.1159/000030343.
- [52] Gross BR, Cohen HL, Schlessel JS. Perinatal diagnosis of bilateral testicular torsion: beware of torsions simulating hydroceles. J Ultrasound Med 1993. https://doi.org/ 10.7863/jum.1993.12.8.479.
- [53] Wang KS. Assessment and management of inguinal Hernia in infants. Pediatrics 2012. https://doi.org/10.1542/peds.2012-2008.
- [54] Maisonet L. Inguinal hernia. Pediatr Rev 2003;24:34-5.
- [55] Huntsman RJ, Lowry NJ, Sankaran K. Nonepileptic motor phenomena in the neonate. Paediatr Child Health (Oxford) 2008. https://doi.org/10.1093/pch/13.8.680.
- [56] Rosman NP, Donnelly JH, Braun MA. The jittery newborn and infant: a review. J Dev Behav Pediatr: JDBP 1984;5(5):263–73.
- [57] Futagi Y, Suzuki Y, Toribe Y, Kato T. Neurologic outcomes of infants with tremor within the first year of life. Pediatr Neurol 1999. https://doi.org/10.1016/S0887-8994(99)00037-5.
- [58] Kramer U, Nevo Y, Harel S. Jittery babies: a short-term follow-up. Brain Dev 1994. https://doi.org/10.1016/0387-7604(94)90045-0.
- [59] Coulter DL, Allen RJ. Benign neonatal sleep myoclonus. Arch Neurol 1982;39:191–2.
- [60] Marx C, Masruha MR, Garzon E, Vilanova LCP. Benign neonatal sleep myoclonus. Epileptic Disord 2008. https://doi.org/10.1684/epd.2008.0196.
 [61] Futagi Y, Otani K, Goto M. Prognosis of infants with ankle clonus within the first year
- of life. Brain Dev 1997. https://doi.org/10.1016/S0387-7604(96)00069-1.
- [62] Hudak ML, Tan RC. Committee on drugs, committee on fetus and newborn, American academy of pediatrics. Neonatal drug withdrawal Pediatrics 2012. https://doi.org/10.1542/peds.2011-3212.
- [63] Korff C, Nordli DR. Do generalized tonic-clonic seizures in infancy exist? Neurology 2005. https://doi.org/10.1212/01.wnl.0000187125.87414.f3.

- [64] Hsieh DT, Chang T, Tsuchida TN, Vezina LG, Vanderver A, Siedel J, et al. New-onset afebrile seizures in infants: role of neuroimaging. Neurology 2010. https://doi.org/ 10.1212/WNL0b013e3181c91847.
- [65] Hsieh DT, Walker JM, Pearl PL. Infantile seizures: Infants are not just little children. Curr Neurol Neurosci Rep 2008. https://doi.org/10.1007/s11910-008-0022-1.
- [66] Bui TT, Delgado CA, Simon HK. Infant seizures not so infantile: first-time seizures in children under six months of age presenting to the ED. Am J Emerg Med 2002. https://doi.org/10.1053/ajem.2002.34796.
- [67] Hirtz D, Ashwal S, Berg A, Bettis D, Camfield C, Camfield P, et al. Practice parameter: evaluating a first nonfebrile seizure in children. Neurology 2000. https://doi.org/10. 1212/WNL55.5.616.
- [68] Scarfone RJ, Pond K, Thompson K, Fall I. Utility of laboratory testing for infants with seizures. Pediatr Emerg Care 2000. https://doi.org/10.1097/00006565-200010000-00001.
- [69] Mackay MT, Weiss SK, Adams-Webber T, Ashwal S, Stephens D, Ballaban-Gill K, et al. Practice parameter: medical treatment of infantile spasms: report of the American Academy of Neurology and the Child Neurology Society. Neurology 2004. https:// doi.org/10.1212/01.WNL0000127773.72699.C8.
- [70] Shields WD. Infantile spasms: little seizures, BIG consequences. Epilepsy Curr 2006. https://doi.org/10.1111/j.1535-7511.2006.00100.x.
- [71] Riikonen R. Long-term outcome of patients with West syndrome. Brain Dev 2001. https://doi.org/10.1016/S0387-7604(01)00307-2.
- [72] Riikonen R. The latest on infantile spasms. Curr Opin Neurol 2005. https://doi.org/10. 1097/01.wco.0000162847.91531.9c.
- [73] Kelly DH, Stellwagen LM, Kaitz E, Shannon DC. Apnea and periodic breathing in normal full-term infants during the first twelve months. Pediatr Pulmonol 1985. https:// doi.org/10.1002/ppul.1950010409.
- [74] Dinkin MJ, Rizzo JF. Abnormal eye movements in children. Int Ophthalmol Clin 2008. https://doi.org/10.1097/IIO.0b013e31816924b2.
- [75] Roucoux A, Culee C, Roucoux M. Development of fixation and pursuit eye movements in human infants. Behav Brain Res 1983. https://doi.org/10.1016/0166-4328 (83)90159-6.
- [76] Donahue SP, Baker CN. Procedures for the evaluation of the visual system by pediatricians. Pediatrics 2016. https://doi.org/10.1542/peds.2015-3597.
- [77] Lemons JA, Blackmon LR, Batton DG, Bell EF, Engle WA, Kanto WP, et al. Apnea, sudden infant death syndrome, and home monitoring. Pediatrics 2003. https://doi.org/ 10.1542/peds.2005-1877.
- [78] Ralston S, Lieberthal A, Meissner H, American Academy of Pediatrics. Clinical practice guideline: the diagnosis, management, and prevention of bronchiolitis. Pediatrics 2014. https://doi.org/10.1542/peds.2006-2223.
- [79] Tieder JS, Bonkowsky JL, Etzel RA, Franklin WH, Gremse DA, Herman B, et al. Brief resolved unexplained events (formerly apparent life-threatening events) and evaluation of lower-risk infants. Pediatrics 2016. https://doi.org/10.1542/peds.2016-0590.
- [80] Virbalas J, Smith L. Upper airway obstruction. Pediatr Rev 2015. https://doi.org/10. 1001/archinte.1977.03630150005004.
- [81] Dawson JA, Kamlin COF, Vento M, Wong C, Cole TJ, Donath SM, et al. Defining the reference range for oxygen saturation for infants after birth. Pediatrics 2010. https://doi.org/10.1542/peds.2009-1510.
- [82] Chang AC. Pediatric cardiac intensive care. Lippincott Williams & Wilkins; 1998
- [83] Sasidharan P. An approach to diagnosis and management of cyanosis and tachypnea in term infants. Pediatr Clin North Am 2004. https://doi.org/10.1016/j.pcl.2004.03. 010.
- [84] Steinhorn RH. Evaluation and Management of the Cyanotic Neonate. Clin Pediatr Emerg Med 2008. https://doi.org/10.1016/j.cpem.2008.06.006.
- [85] Pediatrics AA of. Management of hyperbilirubinemia in the newborn infant or more weeks of gestation. Pediatrics 2004. https://doi.org/10.1542/peds.114.1.297.
- [86] Tiker F, Tarcan A, Kilicdag H, Gurakan B. Early onset conjugated hyperbilirubinemia in newborn infants. Indian J Pediatr 2006. https://doi.org/10.1007/BF02758562.
- [87] McLaughlin MR, O'Connor NR, Ham P. Newborn skin: part II. Birthmarks. Am Fam Physician 2008;77.
- [88] Harris TS. Bruises in children: normal or child abuse? J Pediatr Heal Care 2010;24: 216–21.
- [89] Schwartz KA, Metz J, Feldman K, Sidbury R, Lindberg DM, Investigators E. Cutaneous findings mistaken for physical abuse: present but not pervasive. Pediatr Dermatol 2014;31:146–55.
- [90] O'Connor NR, McLaughlin MR, Ham P. Newborn skin: part I. Common rashes. Am Fam Physician 2008;77.
- [91] Cole C, Gazewood J. Diagnosis and treatment of impetigo. Am Fam Physician 2007; 75.
- [92] Johnston GA. Treatment of bullous impetigo and the staphylococcal scalded skin syndrome in infants. Expert Rev Anti Infect Ther 2004;2:439–46.
- [93] Van Praag MCG, Van Rooij RWG, Folkers E, Spritzer R, Menke HE, Oranje AP. Diagnosis and treatment of pustular disorders in the neonate. Pediatr Dermatol 1997;14: 131–43.
- [94] Sharp WW, Davis C, McQueen A. Neonate with pustular rash. Ann Emerg Med 2015; 65:464.
- [95] Mogre DA. Generalised staphylococcal pustulosis in a neonate: a case report. Australas Med J 2013;6:532.
- [96] Kharazmi SA, Hirsh DA, Simon HK, Jain S. Management of afebrile neonates with skin and soft tissue infections in the pediatric emergency department. Pediatr Emerg Care 2012;28:1013–6.