An Evidence-Based Review Of Neonatal Emergencies

A 5-day-old boy is brought into the emergency department for poor feeding and lethargy. The patient is the full-term product of a vaginal delivery to a healthy mother who received routine prenatal care. He had been eating well—2 oz of formula every 2 hours—until today, when he began sucking poorly and taking less than half an ounce with each feeding. He has been afebrile, and the review of his systems is otherwise negative. On examination, the baby is notably difficult to arouse. He appears slightly jaundiced and mottled, which the mother believes are new findings. His temperature is low at 35.5°C (95.9°F), his heart rate is 190 beats per minute, his respiratory rate is 50 breaths per minute, and his blood pressure reading is 66/38 mm Hg. His anterior fontanel is open and flat, his lungs are clear, the cardiac examination reveals significant tachycardia, the liver is palpable 1 cm below the costal margin, results of the abdominal examination are unremarkable, and the capillary refill time is poor at 5 seconds. It has been some time since you reviewed the differential diagnosis of the ill neonate, but you recall the mnemonic THE MISFITS and generate an extensive list: trauma, heart disease, electrolyte disturbances, metabolic, inborn errors, sepsis, formula mishaps, intestinal catastrophes, toxins, and seizures. You have pediatric colleagues available, but this newborn looks like he needs some intervention before they are likely to return your page. Where should you start with the resuscitation? If little blood is available, what are the high-yield laboratory tests? What if the nurses can’t obtain access in this critical patient? And what illnesses are most likely (ie, to help you establish a diagnosis and start disease-specific treatment as quickly as possible)?

The ill neonate is a frightening entity for most emergency clinicians. Neonates are a rare entity at many nonpediatric emergency departments (EDs), and when they are brought in, it is frequently

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CME Objectives
1. Recognize and understand basic principles of resuscitation in the ill neonate.
2. Properly interpret test results and definitively diagnose emergent conditions.
4. Avoid pitfalls in neonatal diagnosis and management.

Date of most recent review: July 10, 2010
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Medium: Print and Online
Method of participation: Print or online answer form and evaluation
Prior to beginning this activity, see “Physician CME Information” on the back page.

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E B MED I C I N E
P E D I A T R I C
E M E R G E N C Y M E D I C I N E
P R A C T I C E

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for minor complaints. When critically ill infants do present, appropriate newborn resuscitation equipment and consultations are often unavailable. Even when a general pediatrics consultation is readily available, the experience with ill children may be limited. One study of academic pediatric training programs indicated that only 36% of graduating residents had a pediatric resuscitation, and a handful had no pediatric advanced life support (PALS) training. It is easy to understand why the resuscitation of a neonate can be an intimidating and lonely experience for an emergency clinician.

This issue of Pediatric Emergency Medicine Practice will discuss recognition of the causes as well as general and disease-specific means of stabilizing the critically ill neonatal patient. There are many rare diseases that can cause shock in a neonate. This article will concentrate on some of the most common: sepsis/serious bacterial infections (SBIs), including meningitis, bacteremia, and urinary tract infection; malrotation; necrotizing enterocolitis (NEC); ductal-dependent cardiac lesions, including cyanotic congenital heart disease and ductal-dependent obstructive lesions; inborn errors of metabolism (IEMs) that present with significant metabolic derangements in the neonatal period (specifically, urea cycle defects and organic acidemias); salt-wasting types of congenital adrenal hyperplasia (CAH); and non-accidental trauma (NAT).

The Treatment of The Critically Ill Neonate — visit Available Online At No Charge To Subscribers

Critical Appraisal Of The Literature

Population-Based Data

Neonatal mortality information was derived from the National Vital Statistic System maintained by the Centers for Disease Control and Prevention. This is a national reporting database. Small prospective studies on the incidence and test characteristics of neonatal sepsis were also available.

Diagnosis, Treatment, And Outcome Data

Each of the diseases discussed in this article is an uncommon entity, and the diagnosis and treatment is generally based on extrapolation from pathophysiology, expert opinion, and small retrospective data sets. A large prospective study was available on treatment of IEMs and optimal antibiotic therapy for the infected neonate. A well-researched literature review resulting in national guidelines was available regarding screening for congenital heart disease and treatment of neonatal sepsis. Small prospective studies were done on the association between SBIs and apparent life-threatening events (ALTEs). The remainder of the studies used in this paper consisted of retrospective case series.

Literature Search

PubMed® and Ovid MEDLINE® were searched for literature on neonatal emergencies published from 1950 to the present. Multiple search terms were used because of the variety of conditions discussed. Pertinent abstracts were used for non-English language studies.

Pathophysiology

Malrotation represents an arrest of the normal rotation of the intestine during early fetal development. Volvulus occurs in the setting of an underlying malrotation and refers to the twisting of the small bowel around the superior mesenteric artery, causing intestinal ischemia. Malrotation alone is generally asymptomatic; however, when volvulus occurs, the bowel ischemia and resultant perforation and translocation of bacteria can become life threatening.

Necrotizing enterocolitis is due to ischemic necrosis of the intestinal mucosa, which can lead to invasion of enteric organisms. The causes are multifactorial, but the immature mucosa in premature infants renders them particularly susceptible.

The IEMs discussed here involve defects in the breakdown of protein, either via enzyme deficiencies of the urea cycle that result in failure to metabolize nitrogen or via impaired breakdown of amino acids, causing abnormal urinary excretion of organic acids. In utero, the fetus’s metabolic requirements are supported by the mother, masking metabolic defects. After birth, this maternal reservoir no longer exists; after 3 to 5 days, toxic metabolites have had the
opportunity to build up to toxic levels in the neonate with a significant IEM. Similarly, the maternal glucose that fed the fetus is no longer available, and the lack of glycogen stores and the immature ability to mobilize glucose predispose the ill or underfed infant to uncompensated hypoglycemia.

In the case of CAH, maternal circulation is also protective against a salt-wasting crisis, and the effects of sodium loss and steroid insufficiency begin to accumulate only after birth. Although 5 different enzyme deficiencies can cause CAH by impairing the conversion of cholesterol to cortisol,6 90% to 95% of neonates suffering from a salt-wasting crisis have a 21-hydroxylase deficiency.7,8

Ductal-dependant cardiac lesions include both cyanotic lesions (tetralogy of Fallot, truncus arteriosus, total anomalous pulmonary venous return [TAPVR], transposition of the great arteries [TGA], pulmonary atresia, and tricuspid atresia) and lesions that obstruct the systemic outflow tract (hypoplastic left heart syndrome, coarctation of the aorta, interrupted aortic arch, and critical aortic stenosis). Fetal circulation is dependent on the ductus arteriosus, which connects the pulmonary and systemic circulations, initially masking cardiac abnormalities.9 At birth, there is a drastic reduction in pulmonary vascular resistance and closure of the ductus arteriosus, with functional closure by the third day of life8 and anatomic closure completed by 2 to 3 weeks. This change establishes distinct pulmonary and systemic circulations with differing pressure gradients, allowing cardiac anomalies to become clinically apparent.

Infections and child abuse are less a function of fetal development or the physiologic changes occurring from fetus to neonate. Instead, infections are often acquired at the time of birth. Premature rupture of membranes is the main risk factor for “early-onset” sepsis in the first week of life, whereas postnatal complications contribute more heavily to “late-onset” sepsis, or infections that occur beyond day 6 of life.11,12 Neonatal sepsis refers to the presence of bacteremia in the context of systemic signs of an infection such as temperature instability or lethargy. Early-onset sepsis is frequently due to group B streptococci or Escherichia coli, whereas late-onset sepsis often involves coagulase-negative staphylococci and Enterobacteriaceae.13 Premature and low-birth-weight infants are particularly susceptible to late-onset sepsis, and consideration should be given to the possibility of multidrug-resistant organisms or Candida in patients with a prior neonatal intensive care unit (NICU) hospitalization.

With a thin, pliable skull and relatively large head, infants are particularly susceptible to child abuse and the effects of rigorous shaking on the part of an abusive caregiver. Sufficient angular deceleration, particularly from shaking followed by an impact, can cause a subdural hematoma or diffuse axonal injury.

Epidemiology

Approximately 18,275 neonates (defined as younger than 28 days of age) die annually in the United States alone. The majority, more than 13,000, die of perinatal complications, many with sequelae of prematurity.14 These children are unlikely to leave the NICU and are thus beyond the purview of this article. The remainder die of a myriad of causes, the most prevalent being congenital malformations (97 per 100,000), sudden infant death syndrome or SIDS (4.6 per 100,000), cardiac disease (2.8 per 100,000), trauma (2.5 per 100,000), and endocrine/metabolic causes (1.6 per 100,000).14 A separate report of 55 consecutive autopsies on neonates who died unexpectedly within the first week found a cause in 23 (42%). Of these, 9 were due to congenital anomalies (mainly cardiac), 9 to infection, and 3 to metabolic disease.15 Although CAH is included, it has become a rare phenomenon in the ED. The baseline rate in North America is 1 case per 15,000 live births, not all of which present in the neonatal period.12 The newborn screen is widely used, and although it is low in specificity,16 it effectively identifies severe neonatal forms,17 preventing most salt-wasting crises.18 However, as the results of the screen may not be available or the caregiver not informed of the results until after a crisis occurs, CAH is discussed in this article.

Prehospital Treatment

In many ways, the priorities for prehospital management of neonates are similar to those used for adults. Known trauma requires immobilization, and emphasis is placed on the basics of airway, breathing, and circulation. Most pediatric and neonatal codes are respiratory, and assessment of the airway is of paramount importance. Neonatal intubation can be difficult; many pediatric trainees fail to meet accepted standards.19 Prehospital intubation without medication tends to be difficult in patients of any age,19 with success rates as low as 48% in children with impending respiratory failure.20 Fortunately, most infants are easy to ventilate by bag-valve-mask, and survival and neurologic outcomes are comparable to intubation in children ventilated with bag-valve-mask in the prehospital setting.21 Insufflation of air into the stomach will cause difficulty in achieving adequate chest rise, and consideration should be given to a nasogastric tube (NGT) or orogastric tube (OGT) in situations where prolonged bagging is anticipated. Glucose levels should also be checked, and access established if possible.
**Emergency Department Evaluation**

**Important History Questions**

Depending on the urgency of the resuscitation effort, the emergency clinician’s ability to complete a thorough history and physical examination of the neonate may be limited. A brief history may unmask the likely cause of symptoms and guide further questioning. In the case of an infant with nonspecific findings, however, the possibility of sepsis/SBI, cardiac anomalies, metabolic disease, gastrointestinal (GI) tract issues, and child abuse (including poisoning) must be explored.

Prenatal and perinatal histories may provide an answer to what is wrong with the patient. Formal structural ultrasounds may give an indication of cardiac disease, although this finding is missed in more than half of cases. Prolonged membrane rupture or perinatal maternal fevers should heighten suspicion of bacteremia. Neonatal herpes can be a devastating illness, with a history of active maternal herpetic lesions ascertained in about half of these cases. Any NICU admission immediately after birth is important. Prematurity is a significant risk factor for NEC and late-onset sepsis. A known cardiac lesion is also a risk factor for NEC. Consanguinity or a history of suspicious results on the neonatal screen may point to CAH.

In assessing the symptoms of an ill infant, the emergency clinician should be familiar with the spectrum of activities expected in a healthy neonate. After the first day of life, 6 to 8 breastfeedings of approximately 15 minutes per side are normal. Bottle-fed infants require about 5 oz/kg/d, generally taken as a 2- to 3-oz feeding over 15 to 20 minutes. Infants should regain their birth weight by day 10 of life and subsequently gain 15 to 30 gm/d. The average infant produces yellow seedy stools between 3 and 6 times per day (this can vary widely) and about 8 urinations per day after the first 48 hours.

Deviations from these patterns may be indicative of significant disease. Slow feeding, particularly in the context of poor weight gain or vomiting, can be a symptom of significant pathology but is classically associated with heart disease. Gastroesophageal reflux disease is common in neonates, but bilious emesis or significant emesis with poor weight gain can indicate a GI tract disorder. One single-center prospective study of neonates with bilious emesis found a surgical cause in 38%, including (in descending order of frequency) Hirschsprung disease, bowel atresia, malrotation, meconium ileus, meconium plug, and other inspissation. Notably, this included newborns not yet discharged from the hospital, representing a slightly different population than the older neonates who present to an ED. It is important to avoid the pitfall of a cavalier diagnosis of gastroenteritis. In one study of patients with CAH, while many had vomiting and failure to thrive, 30% of caregivers also reported diarrhea, in another study, 20% of patients were initially misdiagnosed as having gastroenteritis.

Subtle changes in mental status can be difficult to determine. Healthy infants sleep a median of 16.2 hours per day. An infant who seems lethargic to parents should be evaluated for sepsis, metabolic disease, or abusive head trauma. Colic or frequent crying, particularly in the late afternoon and early evening, often occurs in the first month of life, but a change in pattern, inconsolability, or crying that progresses to lethargy should not be attributed to colic.

Specific symptoms may also be attributable to serious neonatal diseases. Fever, of course, is worrisome, though not ubiquitous in sepsis. Poor urine output can occur in any condition associated with dehydration or shock, whereas polyuria is reported in CAH. An ALTE such as an episode of apnea, change in skin color or tone, or choking/gagging that is frightening to the caregiver can occur in any of the diseases discussed in this article. Specifically, the rate of SBI in infants 0 to 60 days of age presenting with an ALTE was 2.7% in one study, and rates of child abuse in all patients with an ALTE have been reported to be between 3% and 32%.

Finally, several risk factors for abuse can be assessed. Personality disorders or stressed marital situations are not uncommon among abusers. Inquiries into the social support structure available to a neonate are mandatory if NAT is being considered. Also, a careful interrogation regarding the use of prescription and over-the-counter medications by both the mother while pregnant and the child, or their availability, may unmask the cause of illness. Parents can unintentionally poison children while trying to provide relief for respiratory symptoms or fussiness or intentionally attempt to pharmacologically quiet a fussy infant.

Table 1 lists features of neonatal emergencies commonly revealed by the history and physical examination.

**Physical Examination**

Immediate assessment of airway and breathing is mandatory. Neonates are often initially polycythemic and then reach a hemoglobin nadir around 6 weeks of life. Because of the polycythemia, overt cyanosis is much easier to appreciate in the first few days of life, but less obvious in the relatively anemic child nearing 28 days of age. Additionally, healthy neonates can experience either acrocyanosis (cyanosis limited to the hands and feet) due to vasomotor instability or perioral cyanosis due to a prominent superficial venous plexus. Neither represents true hypoxemia. However, these potential pitfalls underscore the need for objective determination of the patient’s oxygen saturation level, usually by pulse oximetry.

Assessing the work of breathing and respiratory rate is also important. Healthy neonates experience
periodic breathing, with brief respiratory pauses alternating with periods of rapid breathing. Therefore, a full minute of observation or auscultation is required to accurately assess the respiratory rate. The work of breathing is often helpful in diagnosing illness in a neonate. Children with respiratory disease or pulmonary edema tend to have retraction and obvious increased work of breathing. Patients with cyanotic congenital heart disease without pulmonary edema tend to have a quiet tachypnea, with minimal work of breathing in spite of the rapid respiratory rate. Of the cyanotic congenital heart diseases, TGA, truncus arteriosus, and TAPVR are associated with an increase in pulmonary blood flow and often pulmonary edema, whereas pulmonary or tricuspid atresia and tetralogy of Fallot are associated with restriction of pulmonary blood flow and clear lungs. Neonates with acidosis, including many with metabolic disease, sepsis, or hypoplastic left heart syndrome, may be tachypneic in an attempt to compensate for the metabolic acidosis.

Circulation and hydration can be difficult to assess in neonates. Capillary refill time is helpful and should be less than 3 seconds, but the test may have to be performed more centrally because of acrocyanosis. Tear production may not occur until 2 weeks of age. Hypotension tends to be a very late finding in infants. Therefore, tachycardia must be taken seriously in the ill child and not attributed merely to agitation or fever. Similarly, bradycardia can be an ominous finding and should trigger a reassessment of the patient’s respiratory status and temperature. As many ill neonates come in vomiting or with poor intake, most will have some degree of poor perfusion. Specifically, infants with obstructive cardiac disease such as hypoplastic left heart syndrome or coarctation of the aorta may have hypoperfusion out of proportion with other indicators of dehydration or lower extremity perfusion that is significantly worse than that noted in the upper extremities.

Abnormalities in respiratory rate, heart rate, or blood pressure can be found in any critically ill neonate. Vital signs are of particular importance in neonates, as they may not appear as ill at first glance as an older child with significant pathology. The normal neonatal resting heart rate is between 120 and 160 beats per minute; the respiratory rate, 40 to 60 breaths per minute; and the systolic blood pressure reading, 60 to 90 mm Hg. A fever in an infant less than 1 month of age is defined as a temperature

Table 1. Common Signs And Symptoms Of Life-Threatening Neonatal Illnesses

<table>
<thead>
<tr>
<th>Disease</th>
<th>Systemic Signs and Symptoms</th>
<th>Gastrointestinal Signs and Symptoms</th>
<th>Neurologic Signs and Symptoms</th>
<th>Other Signs and Symptoms</th>
<th>Typical Age at Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sepsis</td>
<td>Fever or hypothermia</td>
<td>Poor feeding, abdominal distension, jaundice</td>
<td>Lethargy</td>
<td>Respiratory distress</td>
<td>Early onset: &lt; 1 week; late onset: 7-28 days</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>May present with concurrent infection</td>
<td>Poor feeding, vomiting, hepatomegaly</td>
<td>Murmur (44%); hypoxia (81%)</td>
<td></td>
<td>&lt; 10 days</td>
</tr>
<tr>
<td>Malrotation</td>
<td>May present with shock if accompanied by bowel ischemia</td>
<td>Bilious emesis (97%); bloody stools, constipation (89%); normal examination results (60%-76%)</td>
<td>Often associated with other syndromes or anomalies</td>
<td></td>
<td>3-7 days</td>
</tr>
<tr>
<td>Necrotizing enterocolitis</td>
<td>Vital sign abnormalities (59%); lethargy, apnea</td>
<td>Abdominal distension (100%); gastric retention (64%); heme-positive stool or emesis (36%)</td>
<td>Often associated with prematurity (88%); heart disease</td>
<td></td>
<td>10-12 days</td>
</tr>
<tr>
<td>Inborn errors of metabolism</td>
<td>Concurrent infection (34%)</td>
<td>Vomiting (30%); poor feeding (19%)</td>
<td>Any (76%); altered level of consciousness (63%); seizure (9.6%)</td>
<td></td>
<td>3-5 days</td>
</tr>
<tr>
<td>Congenital adrenal hyperplasia</td>
<td>May present with concurrent infection or shock</td>
<td>Vomiting (28%-63%); poor feeding, failure to thrive (60%)</td>
<td>Lethargy, irritability, seizure (12%)</td>
<td>Ambiguous genitalia in girls (92%-100%); hyperpigmentation of genitalia or axilla (23%-40%)</td>
<td>Begins 2nd week of life; median time of diagnosis is 1 month</td>
</tr>
<tr>
<td>Child abuse</td>
<td></td>
<td>Apnea, seizure, altered mental status</td>
<td>Retinal hemorrhages (53%-80%); bruising, history of abuse (24%)</td>
<td></td>
<td></td>
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</tbody>
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of 38°C (100.4°F) or above. Temperature instability is characteristic of neonatal sepsis, with fever predicting an SBI in about 7.7% of febrile neonates (meningitis, 0.5%; bacteremia, 1.8%; urinary tract infection, 5.4%). Hypothermia is not an uncommon finding in SBI—approaching 31% to 40% in gram-negative neonatal sepsis—and portends a poorer prognosis. Many of the other conditions discussed here can be precipitated or complicated by infection, and temperature instability in a neonate with a suspected cardiac, GI, or metabolic disorder is likely to represent an associated infection.

Assessment of the neurologic status of newborns can be difficult, as the majority of their day is spent sleeping with many episodes of crying in between. A social smile has yet to develop. They are generally arousable, however, and are usually consolable by a parent, particularly with feeding. Stranger anxiety has not yet developed, so attributing fussiness to fear of the medical provider is folly. Occasional myoclonic jerks, a startle response (Moro reflex), an upgoing Babinski reflex, and several beats of clonus are all normal findings. Persistent tremor and odd stereotyped movements (eg, moving the legs as if pedaling a bicycle) are abnormal findings and should heighten concern about a metabolic disorder with hyperammonemia. Seizures in this age group can present either overtly or subtly with apnea or changes in tone alone. Seizures may indicate abnormalities involving glucose or electrolyte levels, central nervous system (CNS) infections, metabolic disease, or child abuse. Finally, assessment of tone is appropriate even in the newborn. A healthy neonate prefers to maintain his arms and legs in a flexed position and can maintain his head in line with his body when suspended prone with his abdomen supported. Hypertonia may represent seizure or metabolic disease, while decreased tone can be present in any critically ill infant. It is important to note that substantial nonaccidental head injury can exist without neurologic findings; however, the presence of neurologic findings should raise this concern as well.

The abdominal examination is of variable utility and may prove falsely reassuring. One small study of neonates with a diagnosis of malrotation reported that 95% appeared well, including 92% of those with volvulus. No abnormalities were noted on abdominal examination in 76% of the neonates, including 60% of those with volvulus. Infants with NEC will have abdominal distension, often with gastric retention and occasionally bloody emesis or stools. Ill infants with gram-negative sepsis may display abdominal distension as well. Palpable, distended loops of bowel and abdominal wall erythema and edema can be appreciated in advanced cases of NEC. Additionally, hepatomegaly can sometimes be seen in patients with cardiac disease or certain IEMs.

Reliance on heart murmurs for the identification of ductal-dependent cardiac lesions has its pitfalls. Between 0.6% and 4.2% of all newborns exhibit heart murmurs, and the yield after further workup of these neonates is about 15%. The yield increases to 33% when the murmur is still present at days 7 through 10. Conversely, heart disease may exist despite normal findings on cardiac examination. One study found that only 44% of infants with structural heart disease had a murmur. Another study showed a difference in the sensitivity of the clinical examination based on the experience of the physician, with pediatric house staff detecting 39% of structural heart disease and cardiologists 94%. Notably, the studies done on this topic have involved all neonates, not a critically ill population presenting to the ED, meaning the yield from physical examinations in the ED setting may be higher than the figures quoted here.

Pulse oximetry, which should be uniformly measured in ill neonates, is expected to be normal by 24 hours of life, with a median rate of 97.6% at days 2 to 7 of life. Literature on neonates with known critical congenital heart disease suggests that 85% will have a pulse oximetry reading below 95%. However, hypoxemia may also be present in a critically ill neonate with sepsis or hypoglycemia. As certain lesions demonstrate a gradient in perfusion between the upper and lower extremities, assessing both for a difference of more than 3% to 4% in oxygen saturation may increase the sensitivity of pulse oximetry to 92.4% for the detection of critical congenital heart disease. Similarly, obstructive cardiac lesions can cause perfusion to be poorer and blood pressure readings to be lower in the lower extremities than in the upper extremities.

In addition to cyanotic skin, other important manifestations of infectious diseases may be noted during the physical examination. Erythema around the umbilicus can be indicative of omphalitis, a serious infection requiring admission for intravenous (IV) antibiotics. Diffuse pustulosis may indicate a staphylococcal infection, and methicillin-resistant Staphylococcus aureus (MRSA) outbreaks, which have occurred in NICUs and well-baby nurseries, are also a possibility. In the neonate, vesicles upon an erythematous base that appear similar to herpetic lesions in older individuals can indicate herpes infection. As the mortality of this infection is high upon progression to systemic or CNS herpetic disease, early recognition and inpatient treatment of isolated skin or mouth lesions is of paramount importance.

Unfortunately, a number of benign newborn rashes can be confused or occur concurrently with more ominous pathology. The most common of the benign rashes by far is erythema toxicum, occurring in 11% to 33% of newborns. Erythema toxicum is characterized by pinpoint whitish-yellow papules and pustules surrounded by larger blotchy erythematous macules. It generally appears on the trunk within the first 48
hours of life, often progressing outward and lasting for 2 weeks. The skin of infants with conditions such as erythema toxicum must be studied carefully to ensure that pathologic lesions are not hiding among the more benign rashes. Jaundice can be appreciated on skin examination and by the presence of scleral icterus. It can be difficult to identify in a pale or darker-skinned infant, however, and briefly blanching the skin with pressure may facilitate identification. Often, jaundice is a benign finding associated with breastfeeding or blood cell breakdown in the setting of an immature liver. But new-onset jaundice later in the neonatal period can be seen with sepsis, metabolic disease (classically at 3 weeks of age), and urinary tract infections.

Bruising in the neonate without a clear and well-witnessed trauma should raise the concern for abuse; however, many neonates with inflicted head injury have no cutaneous signs of abuse. Hyperpigmentation in the genital or axillary regions may be caused by CAH.

Although assessment for retinal hemorrhages can be difficult without dilation and ophthalmology consultation, it should still be attempted if abuse is on the differential. Although lack of hemorrhages does not rule out this occurrence, the finding has a 93% positive predictive value for abuse.

Ambiguous genitalia occurs in nearly all female neonates with CAH and can produce genitals so convincingly male that the presence of an apparent penis with bilateral cryptorchidism should prompt suspicion of the condition.

### Laboratory Testing

Ideally, testing should be guided to some degree by clinical suspicion. For many critically ill neonates, however, the underlying diagnosis remains a mystery after the history and physical examination, and a wide net must be cast when evaluating these patients further. For any significantly ill neonate, electrolyte, glucose, and calcium levels; a complete blood cell count (CBC); prothrombin time (PT); partial thromboplastin time (PTT); and a blood culture should be considered once IV access is obtained.

In the remainder of this section, laboratory tests and their characteristics are considered by diagnosis. Clearly, some of the laboratory studies discussed are more accessible in the ED than others; however, a discussion of less commonly used studies that may become mainstream in the near future is included. Table 2 (see page 8) contains a list of suggested laboratory and other tests by diagnosis.

### Sepsis/SBI

Standard tests for the ill neonate with suspected sepsis include CBC; urinalysis; cerebrospinal fluid (CSF) parameters; blood, urine, and CSF cultures; Chem-7; calcium level; and PT/PTT. White blood cell count is the most widely used marker of neonatal infection. Unfortunately, it is difficult to find a white cell count with adequate sensitivity and specificity, and there is substantial overlap (area under the receiver operating characteristic curve, 0.723) between the white blood cell count of neonates with and without SBIs. Low white blood cell counts are frequent in ill infants, and at least one study has correlated a lower white blood cell count with an increased risk of meningitis relative to bacteremia in neonates.

Immature neutrophil (band cell) to total neutrophil ratio may improve the sensitivity of the CBC.

Recently, C-reactive protein (CRP) has been extensively studied for its ability to predict infection. The greatest limitation to use of CRP is that it requires 8 to 10 hours for synthesis, giving it a sensitivity range of 14% to 100% in the first 24 hours. Additionally, it is a nonspecific marker of inflammation, and its elevation is not limited to SBIs. A cutoff of 70 g/L has been proposed.

Although not a standard ED test, procalcitonin level has also been receiving significant attention in the research. A prehormone of calcitonin produced mainly by hepatocytes and monocytes, it increases within 2 hours of onset of infection. Although physiologic elevations within the first 48 hours of life can make interpretation difficult, the test characteristics are quite good in early-onset sepsis: a sensitivity of 92.6% and a specificity of 97.5%. Procalcitonin level is less impressive in identifying SBIs that occur later in the first month of life (sensitivity, 69%, and specificity, 89%).

Many biochemical markers with limited availability, particularly in ED, have been studied as predictors of neonatal sepsis, including a variety of cytokines. Interleukin 6 is probably the most widely studied; it has better sensitivity (89%) and better negative predictive value (91%) in the early phase of sepsis than CRP does.

Cultures of blood, urine, and CSF are required in all neonates with a suspected SBI. None of the tests discussed previously provide adequate risk-stratification to obviate consideration of meningitis. The CSF should be tested in all febrile neonates for herpes simplex virus polymerase chain reaction (PCR), especially if CSF pleocytosis exists without an obvious alternative etiology. Although consideration of meningitis is imperative in an ill neonate regardless of other laboratory test results, lumbar puncture is not. Although it is extremely helpful to have the results of a lumbar puncture before the initiation of antibiotics, if a neonate is too ill to safely tolerate the procedure, it should be deferred pending stabilization.

Serum PCR has been tested as an alternative to a culture as well, and it has demonstrated good sensitivity (96.2%) and specificity (96.3%) when compared with a culture drawn before the use of antibiotics. Unfortunately, the sensitivity falls after the initiation of antibiotics, rendering it unreliable in the case of an infant who has been pretreated with the drugs. The prevalence of bacteremia in the setting of a urinary tract infection is...
much higher in infants under 2 months (22.7%)\textsuperscript{58} than in older children and adults, and the finding of one SBI in a neonate does not rule out the possibility of another.

Once sepsis has been identified, neutropenia, acidosis, and increased PT are laboratory markers of poor prognosis.\textsuperscript{59}

**Cardiac Disease**

Laboratory tests are less helpful in the diagnosis of congenital heart disease, though a CBC and Chem-7 should be drawn. B-type natriuretic peptide (BNP) may or may not be helpful in establishing the diagnosis of congenital heart disease in the neonate. Typically, established normal levels for neonates are higher than those routinely found in adults (mean triage BNP at 4-6 days of age, 48.4 pg/mL).\textsuperscript{60} Several studies have established increased BNP levels in patients with congenital heart disease later in childhood and in adulthood. A sensitivity of 93.1% and a specificity of 100% were found in neonates with left to right shunts, but reliability was lower for those with cyanotic heart disease.\textsuperscript{61} A recent review found little evidence to suggest that BNP is a reliable marker for identifying congenital heart disease in neonates without shunting or heart failure.\textsuperscript{62} Nevertheless, it may be a useful adjunct to further exclude congenital heart disease in an ill neonate in whom the pretest probability is low.

A CBC may be helpful in determining the need for a transfusion in patients with cyanotic congenital heart disease, and a Chem-7 may guide electrolyte replacement or the correction of acidosis. In patients with poor systemic perfusion, such as those with hypoplastic left heart syndrome, acidosis can be profound and may be a clue to diagnosis.

**GI Disease**

Although unlikely to be helpful diagnostically, Chem-7, CBC, calcium level, and PT/PTT should be checked in ill-appearing neonates with a suspected GI etiology.

**Malrotation**

Infants with significant intestinal necrosis from volvulus can become acidic, thrombocytopenic, and anemic, with alterations in white blood cell count in either direction. No laboratory test has been extensively studied to identify the neonate with malrotation, however.

**Necrotizing Enterocolitis**

A recent systematic literature review analyzed several potential markers for NEC. C-reactive protein had a likelihood ratio (LR) of 1.78 for a positive test and 0.32 for a negative test, and neutropenia had a LR of 0.69. More experimental and less commonly available tests such as cytokine levels have also been studied. Among these, endotoxin performed well, with a sensitivity of 8.9% and a specificity of 97.6%, as did intestinal fatty acid binding protein, with a LR+ of 6.58.\textsuperscript{63}

**Metabolic Disease**

Infants with a suspected IEM should have blood sent for electrolytes, ammonia, liver function tests, and CBC testing. The hallmark laboratory finding in IEMs that cause significant deterioration in the neonatal period is an elevated ammonia level. Ideally, ammonia levels should be sent on ice from free-flowing blood (venous or arterial) and run

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### Table 2. Suggested Testing By Suspected Diagnosis

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Recommended Emergency Department Laboratory Studies</th>
<th>Other Recommended Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infections</td>
<td>CBC, urinalysis, blood/urine/CSF cultures, CSF herpes simplex virus PCR, Chem-7, calcium, PT/PTT</td>
<td>Chest radiograph</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>CBC, Chem-7</td>
<td>ECG, chest radiograph, hyperoxia test, echocardiogram</td>
</tr>
<tr>
<td>Gastrointestinal disease</td>
<td>CBC, Chem-7, calcium, PT/PTT</td>
<td>Abdominal radiograph, UGI study, ultrasound</td>
</tr>
<tr>
<td>Inborn errors of metabolism</td>
<td>Chem-7, venous blood gas, CBC, ammonia, lactate, urinalysis, urine organic acids, plasma amino acids\textsuperscript{a}</td>
<td></td>
</tr>
<tr>
<td>Congenital adrenal hyperplasia</td>
<td>Chem-7, 17-hydroxyprogesterone, dehydroepiandrosterone, androstenedione, testosterone</td>
<td></td>
</tr>
<tr>
<td>Child abuse</td>
<td>CBC, PT/PTT, Chem-7, AST/ALT</td>
<td>Noncontrast head CT, skeletal survey if patient is stable</td>
</tr>
</tbody>
</table>

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CBC, complete blood cell count; CSF, cerebrospinal fluid; CT, computed tomography; ECG, electrocardiogram; PCR, polymerase chain reaction; PT, prothrombin time; PTT, partial thromboplastin time; UGI, upper gastrointestinal.

\(\textsuperscript{a}\) Can be frozen and held for ordering by specialist.
within 90 minutes. Inappropriate collection (eg, long tourniquet time) or dehydration can cause elevations, but an elevation 3 to 5 times the normal level (ie, above 150-200 µmol/dL) is unusual outside the context of frank decompensated shock or metabolic disease. Large ketones on urinalysis are unusual in healthy neonates and in all children after less than 12 hours of fasting. Their presence is highly suggestive of a metabolic disorder. Most metabolic patients are not hypoglycemic on presentation; however, hypoglycemia is a potential feature of metabolic disease and should be promptly checked.64

Several metabolic disorders can result in neutropenia or thrombocytopenia, particularly in the setting of an acute concurrent infection. Elevated transaminase levels and muscle breakdown causing release of creatine kinase or aldolase are characteristics of specific metabolic diseases and may assist the geneticist in further diagnosis or increase the emergency clinician’s suspicion of metabolic disease, though they are unlikely to alter care in the ED.

With many of the early presenting IEMs, the definitive diagnosis is made via urine organic acids and/or plasma amino acids, which require 48 to 72 hours for results. Lactate and pyruvate levels may be helpful as well. At some hospitals, these tests are easily sent. At others, ordering may be difficult; in this case, urine and blood can be frozen (in a heparinized plasma tube) for ordering later. There are 3 specific reasons for setting these body fluids aside. Firstly, it may expedite the patient’s final diagnosis. Secondly, some disorders are more easily recognized on testing when a patient is in an acute crisis. Finally, the mortality associated with the fulminant version of these diseases in the neonatal period approaches 50%. If the child dies, establishment of a definitive postmortem diagnosis is not always possible upon autopsy, and the results of these laboratory tests will be invaluable for future genetic counseling of the parents.

Patients with salt-wasting forms of CAH typically have hyponatremia, hyperkalemia (in the 6-12 mEq/L range), and low bicarbonate levels. The acidosis is due to hydrogen ion reclamation in the kidney and is not associated with an anion gap unless the dehydration and shock have caused the additional production of lactic acid. The lack of steroids renders these patients more susceptible to hypoglycemia, and the glucose level should be checked not only initially, but throughout the course of resuscitation. Definitive diagnosis is made with 17-hydroxyprogesterone, dehydroepiandrosterone, androstenedione, and testosterone. These samples must be collected prior to steroid therapy, so extra blood should be held/frozen for use by the endocrinologist before administration of medications.

Child Abuse

Any neonate with an intracranial injury should have CBC, Chem-7, PT/PTT, and liver function tests performed. For legal purposes in suspected cases of abuse, documentation of cutaneous bruising or intracranial bleeding should also include a CBC, PT, and PTT. Hyponatremia from syndrome of inappropriate antidiuretic hormone or cerebral salt wasting is a concern in this situation as well. Abuse can be associated with a number of other morbidities, including intra-abdominal injury. In a large prospective study in infants under 3 years of age who were suspected abuse victims, 3.2% had elevated transaminase levels (above 80 IU/L), and this elevation had a sensitivity of 77% and a specificity of 82% for abdominal injury. In 26% of children with an abdominal injury, no other abdominal findings were noted on physical examination.65 Poisoning can be the sole manifestation of child maltreatment or one seen in combination with other forms of abuse; thus, urine toxicology is suggested in the ill infant for which no etiology has been uncovered or in whom abuse seems a reasonable possibility.

Other Diagnostic Studies

Sepsis/SBI

Although a chest radiograph is not required in all febrile neonates because of the low incidence of pneumonia, it may be useful in the septic-appearing neonate. Not only is pneumonia a potential cause of the illness, but it is also known that 67% of older children with MRSA have lung findings such as abscesses or septic emboli.66 Neonates can contract this condition in the NICU or well-baby nursery, and the lung findings may aid in the diagnosis.

Cardiac Disease

An electrocardiogram (ECG) and chest radiography are relatively easy to obtain in the ED. Although helpful if abnormal, these test results may be equivocal or even normal in the face of significant disease. On radiograph, some neonates with congenital heart disease have the characteristic appearance of a boot (tetralogy of Fallot), an egg on a string (TGA or TAPVR), or a snowman (total anomalous pulmonary venous return). The upper limit of normal size for a neonatal heart on the posteroanterior chest radiograph is 60% of the transthoracic distance. In one study of children up to age 12 years, radiologists were able to distinguish normal radiograph results from those of patients with congenital heart disease 78% of the time.67
As congenital heart disease represents a spectrum of different anomalies, possible findings on ECG range from normal to heart failure, left ventricular hypertrophy, right ventricular hypertrophy, biventricular hypertrophy, and even ischemia. The normal neonatal ECG result differs from that of an adult in the prominence of right-sided forces. Pediatric ECG interpretation in the ED is good, at best, with a sensitivity of 75% overall for the identification of congenital heart disease when read by pediatric emergency clinicians. Even when interpreted by specialists, ECG results for as many as 54% of neonates with heart disease were normal. Therefore, while potentially helpful, a normal radiograph or ECG finding does not exclude heart disease.

A well-described method for distinguishing cyanotic congenital heart disease from other forms of cardiac and noncardiac disease is the hyperoxia test. In centers with limited capacity for pediatric echocardiogram, this test may prove valuable in guiding the emergency clinician’s course of treatment. The child is placed on 10 minutes of 100% oxygen; then an arterial blood gas measurement is taken. A PaO₂ below 150 mm Hg after such oxygenation is highly suspicious for cyanotic congenital heart disease. If a blood gas measurement is impossible to obtain, an increase of 10% or more in oxygen saturation indicates a very low likelihood of a cardiac etiology. Although limited literature exists on the accuracy of this test, it is easy to perform in the ED, and one textbook suggests that an abnormal hyperoxia test result indicates a diagnosis of cyanotic congenital heart disease with 90% certainty. There are 2 notable exceptions. In TAPVR, a very rare type of cyanotic congenital heart disease, the PaO₂ is frequently above 150 mm Hg. Also, obstructive noncyanotic lesions do not fall under this rule, although they are usually suspected with shock and poor lower extremity perfusion.

The echocardiogram is the best diagnostic test in the ED; however, its sensitivity may prove unacceptable when practitioners use an adult echocardiography laboratory for diagnosis of pediatric congenital heart disease. In one study, adult laboratories made major errors in 44% of cases, moderate errors in 28%, and minor errors in 12%, compared with rates of 0%, 4%, and 4%, respectively, in pediatric laboratories. The majority of these errors were interpretive and could thus be overcome if pediatric cardiologists remotely read the echocardiograms in settings where telemedicine is available.

GI Disease
For malrotation with or without volvulus, plain radiographs are often nondiagnostic or produce normal findings. When results are abnormal, classic findings of malrotation with volvulus include a distended bubble of air in the stomach and proximal duodenum with an otherwise gasless abdomen. Occasionally, free air from a perforated segment of ischemic bowel may be appreciated. The diagnostic test of choice is an upper gastrointestinal (UGI) study, which has a sensitivity of 96% for malrotation. Sensitivity for identifying volvulus, however, is lower at 54%. False-positives can also occur. Few studies have examined use of ultrasound in diagnosis of malrotation, but it is likely to have efficacy as a screening tool in equivocal cases. One small review showed ultrasound had a 21% false-positive rate for identification of malrotation, with only a 2% false-negative rate when compared with UGI.

For NEC, plain radiograph is the current diagnostic standard, and signs include intraluminal gas (appearing as curvilinear lucencies in the bowel wall), portal venous gas, free intraperitoneal gas, dilated loops of bowel, bowel wall thickening, and perforation. Perforation occurs in 12% to 31% of cases. Significant radiologic evidence is seen in 90% of ill neonates with NEC, with bowel dilatation being the most common and sensitive sign. Both flat plate and cross-table lateral views are recommended.

Ultrasonography has the potential advantage of also identifying intra-abdominal fluid and assessing perfusion and thickness of the bowel wall. It may be more sensitive than plain radiograph in the detection of intramural and portal venous gas. Echogenic dots or granular densities can be seen on ultrasound, with good sensitivity in patients in the early stages of NEC.

Child Abuse
Any suspicion of NAT to the head requires a noncontrast head computed tomography (CT) scan. A skeletal survey can be a helpful adjunctive test to support the diagnosis of child abuse. It is unlikely to radically change management in an unstable infant with an intracranial finding, and the stability of the patient must be considered before he or she is sent to radiology for this extensive series. When deemed safe, however, this survey should be performed from either the ED or the inpatient unit. A 4-year retrospective review of suspected child abuse cases found fractures in 24% of radiographs, with a mean of 2.5 fractures per child. Other studies have shown similar results at 26% to 35% positive rates, with the higher percentage coming from a study looking specifically at abused infants with intracranial findings.

Although magnetic resonance imaging (MRI) is not traditionally obtained in the ED, it is an option in the stable patient in whom subdural collections are suspected; however, it is unlikely to be an option in the critically ill infant. Even when the diagnosis is substantiated on CT scanning, additional pathology may be uncovered on MRI once the child is more stable. Infants who die must be made coroner’s cases for full forensic autopsy.

Any suspicion of child abuse on the part of the emergency clinician requires prompt reporting to the Department of Child and Family Services (DCFS). In the setting of a critically injured child, cross-reporting to the police in the jurisdiction where the abuse is likely to
Treatment

Neonatal Advanced Life Support And Resuscitation

In patients who present in extremis and who require full resuscitation, the latest neonatal advanced life support guidelines suggest a 3:1 ratio of compressions to breaths, at an overall rate of 120 events per minute (90 compressions and 30 breaths). Compressions are indicated if the neonate is pulseless or has a pulse less than 60 beats per minute. Intubation in the term neonate, if indicated, is usually accomplished with a 3.5 or 4.0 uncuffed endotracheal tube (ETT). If epinephrine is administered IV, the dose should be 0.01 mg/kg of the 1:10000 solution, as high-dose epinephrine is no longer recommended. Termination of resuscitative efforts should be considered after the child has been without signs of life for 10 minutes of resuscitative efforts, or earlier (in conjunction with parents) in a child with a known poor prognosis due to extreme prematurity or significant congenital malformations.

Intravenous access deserves special mention in the neonate population, as it can be very difficult to obtain in those who are clamped down because of extreme illness. Scalp veins, external jugulars, and saphenous veins are alternative sites for peripheral access, especially if ultrasound-assisted placement can be performed in a timely fashion. If access is unattainable and the situation is urgent, an intraosseous (IO) line is a reasonable choice. If the patient is semistable, umbilical lines can often be attempted up to 7 to 10 days of life by cutting down the hardened part of the umbilical stump. More traditional types of central lines can be very difficult to achieve in this age group and are likely to take more time than is reasonable in the critical infant.

An overall treatment algorithm is presented on page 12.

Infectious Disease

For the neonate in shock, 10- to 20-mL/kg boluses of normal saline should be administered rapidly until shock reversal is achieved or 60 mL/kg. Reversal is defined as achievement of a capillary refill time of 2 seconds or less, normal pulses, normal blood pressure (67-94 mm Hg systolic/35-56 mm Hg diastolic), normal mental status, and urine output less than 1 mL/kg/h. Packed red blood cell transfusion should be considered in infants with hemoglobin levels below 12 g/dL if they are in shock. The American Heart Association’s PALS course recommends 10 mL/kg of packed red blood cells; however, if the patient has severe anemia or the possibility of cardiac dysfunction exists, 5 mL/kg can be given to start.

If the neonate is resistant to these measures, epinephrine (0.1-0.3 mcg/kg/min) or dopamine (2-20 mcg/kg/min) can be started. The PALS guidelines indicate a preference for epinephrine in the hemodynamically unstable neonate. Hydrocortisone should be considered in neonates with adrenal insufficiency, and extracorporeal membrane oxygenation should be considered in refractory cases.

Traditionally, antibiotic therapy for neonatal sepsis has combined ampicillin (50 mg/kg) with either cefotaxime (50 mg/kg) or gentamicin (2.5 mg/kg). A large prospective study involving NICU patients in the first 3 days of life showed a lower mortality in neonates given ampicillin and cefotaxime (odds ratio, 1.5; 95% confidence interval, 1.4-1.7). Ceftriaxone is not recommended for use in the first month of life. A recent Cochrane database Review found no evidence to support the superiority of one antibiotic regimen over another for late-onset sepsis; however, most regimens cited included a β-lactam antibiotic with or without an aminoglycoside. In infants with CSF pleocytosis or with skin findings of herpes, acyclovir should be started. The risk of herpetic disease is 1% in neonates with CSF pleocytosis and fever, and at some centers, the risk in all febrile neonates is comparable to that of bacterial meningitis. Mortality is high (60%), and treatment is with acyclovir (20 mg/kg).

Cardiac Disease

Initial management of the ill neonate often involves administration of oxygen for cyanosis and IV fluid for poor perfusion. For the acutely ill infant without a clear diagnosis, both interventions are acceptable in the ED and can be discontinued if the patient appears to be worsening. In general, oxygen will encourage closure of the ductus arteriosus and pulmonary flow; therefore, in cases of suspected ductal-dependent lesions, the patient may ultimately be better managed if oxygen saturations in the mid to high 80s and low 90s are accepted. Glucose levels should be checked and corrected as necessary. Many of these patients are quite acidotic, especially those with poor systemic perfusion. Acidosis can be corrected with sodium bicarbonate at a dose of 1 mEq/kg using the 4.2% solution.

Prostaglandin E1 (PGE1) is critical in the treatment of ductal-dependent lesions, both cyanotic and obstructive, as it improves blood flow through the closing ductus arteriosus. The treatment should be initiated as quickly as possible and should not be delayed pending definitive diagnosis by echocardiogram or transfer. The initial dose is 0.05 µg/kg/min via any access, titrated as needed. Side effects of hypotension, seizure, and apnea should be anticipated and usually occur within 1 hour of therapy initiation. This fact becomes important when
Clinical Pathway: Management Of The Critically Ill Neonate

Does the neonate require emergent resuscitation?

NO

Perform history and physical examination; check laboratory test and radiograph results; conduct further testing as needed.

What is the suspected diagnosis?

Cardiac disease

SBI

GI disease

NAT

Metabolic disease

Start PGE1 at 0.05 µg/kg/min (Class 2); correct acidosis (Class 3), if indicated, consider:

- furosemide 1 mg/kg,
- dobutamine 2 to 20 µg/kg/min;
- packed red blood cells 10 mL/kg.

Start ampicillin/gentamicin (Class 1); start IV acyclovir if WBCs in CSF (Class 2). For sepsis, start normal saline with 10- to 20-mL/kg bolus until patient is stable or 60 mL/kg is reached (Class 1).

Insert NGT or OGT; arrange for surgical consult; IVF.

Correct coagulopathy; consult neurosurgery; contact Department of Child and Family Services.

Start D10 ¼ normal saline at 1.5 times maintenance (Class 1); initiate sodium benzoate and sodium phenylacetate at 0.25 g/kg (Class 1); consider L-carnitine (Class 3); correct hypoglycemia.

Schedule surgery.

NEC

Malrotation

Yes

Secure the airway; perform chest compressions if heart rate < 60 bpm; check glucose level (Class 1); initiate appropriate PALS algorithm.

Abbreviations: BPM, beats per minute; CSF, cerebrospinal fluid; D10, dextrose 10%; GI, gastrointestinal; IV, intravenous; IVF, intravascular fluids; NAT, nonaccidental trauma; NEC, necrotizing enterocolitis; NGT, nasogastric tube; OGT, orogastric tube; PALS, pediatric advanced life support; PGE1, prostaglandin E1; WBC, white blood cells; SBI, serious bacterial infection.

Class Of Evidence Definitions

Each action in the clinical pathways section of Pediatric Emergency Medicine Practice receives a score based on the following definitions.

Class I
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case control studies
- Less robust RCTs
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

deciding if prophylactic intubation is required for transfer. Side effects must be managed symptomatically, but they are not an indication for discontinuation of PGE1. Hyperpyrexia is another side effect; however, since infection can occur as a result of decreased GI perfusion in heart disease, a low threshold for starting antibiotics should be maintained, even while recognizing that the fever might be a medication effect.

The need for intubation should be carefully considered, not a reflex reaction to suspicion of congenital heart disease or treatment with PGE1. If these patients require advanced airway management, the emergency clinician should remember that mechanical ventilation, especially with high positive end-expiratory pressure, will increase resistance to pulmonary perfusion. This can be helpful in cases of obstructive lesions but deleterious in patients with certain cyanotic lesions. If the neonate becomes more deeply cyanotic after intubation, consideration should be given to decreasing the positive end-expiratory pressure and tidal volume in addition to troubleshooting the airway.

Adjacent agents administered to neonates are similar to those used in adult cardiology. If a hemodynamically stable infant develops pulmonary edema, a dose of furosemide (1 mg/kg) can be given. For additional treatment of heart failure or to augment systemic perfusion, dobutamine (2-20 µg/kg/min), amrinone (0.5 µg/kg/h slow IV push), or milrinone (10-50 µg/kg over 10 minutes) can be used. A recent study of adults in cardiogenic shock demonstrated better outcomes with norepinephrine than dopamine for additional blood pressure support; presently, no literature indicates that this superiority applies to neonates. In neonates with cyanosis and congenital heart disease, transfusion of 10 mL/kg of packed red blood cells should be considered if hemoglobin is below 12 g/dL.

For many patients with congenital cardiac disease, surgery is promptly performed and often takes the form of a Blalock-Taussig shunt or a Sano shunt. The more common Blalock-Taussig shunt connects the subclavian artery to the pulmonary artery, thus supporting pulmonary circulation via the subclavian. As a result, the patient remains cyanotic, with oxygen saturation around 80%. The patient should have a significant murmur due to the movement of blood through the shunt; lack of a murmur is an ominous sign that the shunt is not patent. These procedures are done so early that a neonate may return to the ED later, often for worsening cyanosis. Fevers causing shunting of blood away from the pulmonary circuit or cloting of the shunt should be considered in this event. If clotting of the shunt is a possibility, aggressive hydration and cardiology consultation for removal of the clot are appropriate emergently.

**GI Disease**

For malrotation with or without volvulus, the hallmark of treatment in neonates is surgery. Because of the poor sensitivity of the UGI study for volvulus, surgical consultation should be considered urgent in all symptomatic neonates in the ED with suspected malrotation and volvulus. Surgery may consist of a single stage, or it may require a second look to determine if the bowel is viable. In the ED, infants should have an NGT placed and should receive fluid resuscitation with correction of electrolyte levels. If necrosis or perforation is suspected, broad spectrum antibiotics should be promptly initiated. Mortality is 3% to 5%.

Treatment of NEC includes placement of an NGT or OGT, initiation of antibiotics with anaerobic and gram-negative coverage (eg, ampicillin 50 mg/kg, gentamicin 2.5 mg/kg, and metronidazole 7.5-15 mg/kg), and aggressive hydration. Patients recently discharged from a NICU may need additional gram-positive coverage with vancomycin (15 mg/kg). Pneumoperitoneum is classically considered an indication for surgical intervention, though promising results have been found in small studies looking at medical management of these patients. For patients without pneumoperitoneum, repeated radiographs are recommended every 6 to 8 hours to assess for progression of NEC. Mortality is high (between 20% and 40%).

**Metabolic Disease**

The main focus of therapy for neonates with an IEM is to stop the exposure to proteins that may be converted to toxic metabolites and clear the body of toxic byproducts. The first goal is accomplished by discontinuing oral feeding of formula or breast milk and interrupting the body’s catabolism of endogenous proteins by providing adequate glucose substrate. This is most easily accomplished by starting dextrose 10% (D10) 1/4 normal saline at 1.5 times maintenance (neonatal maintenance is 4 mL/kg/h). The temptation exists to start with saline boluses in the dehydrated patient before initiating the dextrose-containing fluid, but it is important to note that catabolism will continue until the infant is provided with dextrose. Therefore, every attempt should be made to give the dextrose-containing fluid concurrently with the bolus, either by using a second line or a Y-connector to infuse them through the same line. If the newborn is hypoglycemic, the glucose level can be corrected more rapidly with use of a D10 5-mL/kg bolus.

Hyperammonemia of 3 to 5 times the normal limit generally necessitates treatment with a combination of sodium benzoate and sodium phenylacetate at an IV dose of 0.25 g/kg over 90 to 120 minutes, followed by infusion of 0.25 g/kg over 24 hours. This regimen was established in a large, prospective trial of infants with urea cycle disorders. At levels of 10 times normal, hemodialysis may be required. Cofactor L-carnitine (100 mg) and arginine hydrochloride (6 mL/kg of a 10% solution over 90 minutes) may be recommended by a geneticist if certain disorders are suspected.
For particularly ill patients without a prompt response to therapy, a potential benefit exists with the use of simultaneous insulin (0.05 U/kg/h) and dextrose (10 mg/kg/min) drips. These drugs can be titrated upward. Careful monitoring is required to keep the serum glucose levels at 120 to 170 mg/dL.\textsuperscript{86}

Patients with organic acidemias typically present with extreme acidosis. Administration of sodium bicarbonate is recommended by many experts in the field for pH < 7.0-7.2, with recommended doses ranging from 0.25–2 mEq/kg.\textsuperscript{97,98} In addition to the well-known complications of bicarbonate in patients of any age (ie, intracellular acidosis, shifting of the oxygen dissociation curve), the emergency clinician must recognize that treatment of acidosis with bicarbonate does not correct the underlying disorder and should not replace standard therapy. Some experts have also expressed concern that large doses of sodium bicarbonate may contribute to cerebral edema in these patients. In summary, bicarbonate is a reasonable treatment option for the ill neonate with IEM and acidosis, but it should be given thoughtfully, in conjunction with the other treatments discussed previously and, if possible, after discussion with a geneticist.

Mortality can be as high as 27% to 50% in these disorders. Regardless of whether the patient survives the ED stay, a tube of urine and heparinized plasma should be frozen for definitive diagnosis.

For patients with suspected CAH, hydrocortisone (25 mg) should be given IV or intramuscularly (IM) as quickly as possible, followed by 50 mg/m\textsuperscript{2}/d (roughly 2-3 mg/kg/d) as a continuous drip or divided every 6 hours.\textsuperscript{7,8} Hydrocortisone is the preferred steroid because of its mineralocorticoid effects, but if it is not available, the initial dexamethasone dose is 0.1-0.2 mg/kg IV or IM. Patients will require fluid and sodium resuscitation, which can be initiated with 20-mL/kg boluses of normal saline in the ED until stabilization, followed by initiation of maintenance fluids, often initially with dextrose 5% normal saline. The hyperkalemia may be impressive, but it is generally well-tolerated. In the absence of ECG changes, it will typically resolve with fluids and hydrocortisone.\textsuperscript{7} Hyperkalemia associated with ECG changes can be treated in the standard fashion, with care taken not to further dehydrate the patient or cause hypoglycemia. Calcium gluconate (100 mg/kg) is preferred over calcium chloride in neonates. Mineralocorticoid replacement with fludrocortisone (0.1 mg/d) should be delayed until the patient resumes an oral diet. Disease-specific mortality in the era of screening is 0.7%.\textsuperscript{6}

**Child Abuse**

In the setting of an acute subdural hematoma, any identified coagulopathy should be urgently corrected with either 10 mL/kg of fresh frozen plasma or platelets. Intubation for airway protection is based on the infant’s mental status. There are no clear recommendations on seizure prophylaxis in neonates, and neurosurgical input should be elicited. Urgent neurosurgical consultation should be obtained; smaller and more chronic bleeds are likely to be observed, whereas larger clots or those with active bleeding will likely require craniotomy and evacuation. Neurologically intact survival occurs in approximately 62% of patients with subdural hematomas and is dependent on mental status at the time of presentation.\textsuperscript{99}

The other role of the emergency clinician is reporting the potential of abuse. Objective findings should be clearly documented in the medical record, and DCFS alerted. Cross-reporting to the police is helpful as well, both in catching the perpetrator before he or she flees and in obtaining assistance should the family attempt to flee with the infant.

**Transfer Issues**

Not infrequently, these patients will require timely transfer to a tertiary care center for definitive testing and treatment. Although contact should be made with appropriate facilities and transport teams early in the ED course, life-saving treatment should not be withheld. Laboratory studies, including lumbar puncture when indicated and radiographs, should be obtained quickly. Reporting potential abuse cases to DCFS should be done by the hospital of first contact. Indicated treatments such as fluids, antibiotics, PGE1, dextrose, or hydrocortisone should not be delayed pending definitive diagnosis or transfer.

**Disposition**

The critically ill neonates discussed in this article warrant admission to a neonatal or pediatric intensive care unit with appropriate subspecialist consultation. This treatment may require transfer using a physician or pediatric critical care transport team.

**Summary**

Very different types of illnesses can culminate in the neonate as oral intolerance, vomiting, and lethargy. It is the responsibility of the emergency clinician to treat the unstable neonate, narrow the differential to the most likely diagnoses, begin life-sustaining treatment, and ensure a safe disposition. Laboratory tests and ancillary studies can be helpful, but empiric treatments such as antibiotics and PGE1 must often be started on the basis of suspicion, rather than a definitive diagnosis.

**Case Conclusion**

Access was obtained rapidly via a scalp vein, and the initial electrolyte panel was normal. A CBC showed a
1. “The child did not appear blue, so I didn’t investigate the possibility of a cyanotic cardiac anomaly.”
   Apparent cyanosis requires 5 g/dL of deoxygenated hemoglobin. Early in the neonatal period, most infants are polycythemic and can manifest cyanosis overtly at an oxygen saturation of 85%. However, later in the neonatal period or in the anemic neonate, the oxygen saturation required to produce a blue appearance is much lower.

2. “The neonate had some bilious emesis, but the plain radiographs of the abdomen were normal.”
   A surgical cause of bilious emesis in the neonate is found in 30% to 40% of cases. In malrotation specifically, findings from plain radiographs are often normal. A UGI study should still be considered, but if it is not possible, the patient should be observed or a pediatric surgeon consulted.

3. “The mother reported a temperature at home, but the infant had no fever in the ED.”
   Tactile fevers can be difficult to interpret. Nevertheless, one retrospective study indicated that in infants with a documented rectal temperature at home, 92% had a fever either on presentation to the ED or during a subsequent 48-hour hospitalization for observation. The safe course is always to take a parent’s history of fever seriously unless there is a compelling reason not to.

4. “The parents say the child rolled off the bed, which may explain the intracranial hemorrhage.”
   Being responsible for removing an infant from his or her home can be difficult; it is even harder when the diagnosis is unclear. It is important to remember, however, that any suspicion on the part of the emergency clinician requires a DCFS report, and further action is up to the legal system and child abuse teams. Rolling is unlikely in an infant under 4 months of age, making the history suspect. Additionally, although skull fractures and epidural hematomas have been reported in infants after a 3-foot fall, subdural hematomas are highly implausible from that mechanism.

5. “I was waiting for the results of the herpes simplex virus PCR before starting the acyclovir.”
   About 1% of neonates with a fever and CSF pleocytosis have herpes meningitis. It is a high morbidity and mortality disease with improved outcomes when treatment is started early in the disease course. As the side effect profile is minimal for acyclovir, it is reasonable to start the drug in the ED.

6. “I was waiting for the nurses to secure an IV in order to start therapy.”
   It is easy to fall into the trap of waiting for the nurse to obtain an IV, whether the delay involves awaiting the arrival of a NICU nurse or a lack of communication about whether access has been established. In the critically ill neonate, a few attempts should be made before moving on to obtain an intraosseous line. Alternatively, an emergent umbilical line can be attempted in neonates up to 10 days old.

7. “I did everything right with the diagnosis of hypoplastic left heart syndrome. Why did the patient die after transfer?”
   The gut hypoperfusion associated with hypoplastic left heart syndrome can cause bowel ischemia and resultant sepsis. Although some patients clearly die from the cardiac lesion itself, it is important to have a low threshold for diagnosing and/or empirically treating infections in these infants.

8. “The patient’s glucose level was 35 mg/dL. I understood that to be normal in the neonate.”
   Historically, lower values were tolerated. Current recommendations consider values below 45 mg/dL to be hypoglycemic, requiring treatment.

9. “I’ve intubated the child, but I’m still having trouble ventilating her.”
   Neonatal intubations can be difficult, but when one does not have the desired result, following the adult algorithm of reconfirming placement, assessing for equipment failure, ruling out pneumothorax, and checking for obstruction is appropriate. Additionally, it is easy to intubate the right mainstem bronchus in an infant; generally, the tube measurement at the lip should be 3 times the size of the ETT (ie, a size 4 tube will be inserted at 12). Finally, gastric distension from bagging can be significant enough to impair ventilation. Placing an NGT or OGT may improve the ability to ventilate the infant.

10. “An infant brought in died in the department, and it sounds like a clear-cut case of SIDS. The parents are strongly against an autopsy.”
    Unfortunately, any unexpected neonatal death does require an autopsy. Sudden infant death syndrome is a diagnosis of exclusion and requires an autopsy to ensure that other potential causes (such as abuse) have been ruled out.
mild elevation in white blood cell count with a left shift. A catheterized urinalysis showed neither leukocyte esterase nor nitrites. Blood and urine cultures were obtained. This infant was given normal saline 20-mL/kg boluses and started on maintenance fluids. Ampicillin and gentamicin were initiated immediately. When the stability of the neonate was established, a lumbar puncture was performed and showed no evidence of meningitis. The blood culture grew Group B Streptococcus 12 hours into the patient’s hospital stay. Antibiotics were changed to penicillin because of sensitivities, and the drug was continued for 10 days. The infant was discharged without apparent deficits on hospital day 12.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.


Cost-Effective Strategies

- Most neonatal and pediatric patients come in with benign conditions requiring little or no workup beyond the history and physical examination. Cost containment is both possible and important in these infants.
- The critically ill neonate will unquestionably require significant resources. Fortunately, this situation is rare. A carefully obtained history and physical examination, with a targeted rather than shotgun approach, may be helpful but are not always possible. Telemedicine and transfer agreements with tertiary care pediatric hospitals may expedite diagnosis and transport of the ill infant out of the ED to a more appropriate setting.
- The conditions discussed in this article all have a high rate of mortality, even when the patient receives the best care. Emergency clinicians should keep parents abreast of their thoughts, the treatment plan, and the serious nature of the infant’s condition to help prepare them in the event of a poor outcome, thus minimizing their perception of a medical error and resultant legal action.
53. Bonsu BK, Harper MB. A low peripheral blood white blood cell count in infants younger than 90 days increases the odds of acute bacterial meningitis relative to bacteremia. *Acad Emerg Med*. 2004;11(12):1297-1301. (Retrospective; 72 patients)

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4. Standard tests for the ill neonate with suspected sepsis include:
   a. CBC
   b. Urinalysis
   c. CSF parameters
   d. Blood, urine, and CSF cultures
   e. All of the above

5. The latest neonatal advanced life support guidelines suggest which of the following?
   a. 100 compressions per minute
   b. 3:1 ratio of compressions to breaths
   c. 30:2 compression to ventilation ratio

6. For the critically ill neonate with a suspected diagnosis of cardiac disease, which of the following is (are) appropriate?
   a. Start PGE1 at 0.05 ug/kg/min
   b. Correct acidosis
   c. Consider furosemide 1 mg/kg
   d. All of the above

7. For the critically ill neonate with a suspected diagnosis of nonaccidental trauma, of the following are appropriate EXCEPT:
   a. Correct coagulopathy
   b. Consult neurosurgery
   c. Administer antibiotics
   d. Contact the Department of Child and Family Services

8. When intubating neonates, the tube measurement at the lip should generally be 3 times the size of the ETT.
   a. True
   b. False
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### EVIDENCE-BASED PRACTICE RECOMMENDATIONS

**An Evidence-Based Review Of Neonatal Emergencies**

Claudius, I. August 2010; Volume 7, Number 8

The ill neonate is a frightening entity for most emergency clinicians. Neonates are a rare entity at many nonpediatric emergency departments (EDs), and when they are brought in, it is frequently for minor complaints. When critically ill infants do present, appropriate newborn resuscitation equipment and consultations are often unavailable. Even when a general pediatric consultation is readily available, the experience with ill children may be limited. It is easy to understand why the resuscitation of a neonate can be an intimidating and lonely experience for an emergency clinician. This issue of Pediatric Emergency Medicine Practice will discuss recognition of the causes as well as general and disease-specific means of stabilizing the critically ill neonatal patient. For a more detailed and systematic look at the critically ill neonate, see the full text article at www.ebmedicine.net.

#### Key Points

<table>
<thead>
<tr>
<th>Neonatal emergencies are rare. The most common include: serious bacterial infections, congenital cardiac disease, GI emergencies (including malrotation with midgut volvulus and necrotizing enterocolitis), metabolic disorders, and child abuse.</th>
<th>Very different types of illnesses can culminate in the neonate as oral intolerance, vomiting, and lethargy. It is the responsibility of the emergency clinician to treat the unstable neonate, narrow the differential to the most likely diagnoses, begin life-sustaining treatment, and ensure a safe disposition. Laboratory tests and ancillary studies can be helpful, but empiric treatments such as antibiotics and PGE1 must often be started on the basis of suspicion, rather than a definitive diagnosis.</th>
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<td>Important points to consider on history and physical include: feeding, vomiting (and whether bilious or non-bilious), fever or hypothermia, mental status (including lethargy or intractable irritability), neurologic findings (such as seizures or repetitive movements), and evidence of abuse (such as bruising or retinal hemorrhages).</td>
<td>Prenatal and perinatal histories may provide an answer to what is wrong with the patient. Formal structural ultrasounds may give an indication of cardiac disease, although this finding is missed in more than half of cases. After the first day of life, 6 to 8 breastfeedings of approximately 15 minutes per side are normal. Bottle-fed infants require about 5 oz/kg/d, generally taken as a 2- to 3-oz feeding over 15 to 20 minutes. Infants should regain their birth weight by day 10 of life and subsequently gain 15 to 30 gm/d. The average infant produces yellow seedy stools between 3 and 6 times per day (this can vary widely) and about 8 urinations per day after the first 48 hours. One single-center prospective study of neonates with bilious emesis found a surgical cause in 38%, including (in descending order of frequency) Hirschsprung disease, bowel atresia, malrotation, meconium ileus, meconium plug, and other inspissation. Subtle changes in mental status can be difficult to determine. Healthy infants sleep a median of 16.2 hours per day. Several risk factors for abuse can be assessed. Personality disorders or stressed marital situations are not uncommon among abusers. Inquiries into the social support structure available to a neonate are mandatory if NAT is being considered. Table 1 (available at <a href="http://www.ebmedicine.net">www.ebmedicine.net</a>) lists features of neonatal emergencies commonly revealed by the history and physical examination.</td>
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<tr>
<td>Important laboratory findings in all ill neonates include: rapid assessment of glucose, complete blood count, electrolytes, and directed laboratory work-up for the condition considered.</td>
<td>The following adjunctive tests may prove helpful in diagnosis: chest radiograph, ECG, abdominal series, upper GI, head CT, and urine toxicology.</td>
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<td>Principles of treatment in all ill neonates include:</td>
<td>Practice Pearls:</td>
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<tr>
<td>• Rapid assessment of airway and breathing with oxygen, bag-valve-mask, or intubation if deemed necessary</td>
<td>• Sepsis can occur in conjunction with many of the other conditions discussed; don’t hesitate to consider and start antibiotics.</td>
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<td>• Rapid assessment of rhythm and heart rate with chest compressions if pulseless or pulse &lt; 60 beats per minute</td>
<td>• Child abuse can present in subtle ways; don’t fail to consider the diagnosis.</td>
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<td>• IV fluid resuscitation with 10 mL/kg boluses of normal saline</td>
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<td>• IV glucose restoration to 50 mg/dL with 2-4 mL/kg boluses of D10</td>
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<td>• Further treatment should be based on clinical suspicion of condition</td>
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