



## **Common Emergency Department Complaints in the First 28 Days of Life**

The newborn in the emergency department often causes anxiety for even the most experienced emergency physician. The lecturer will review how to distinguish a newborn with a life-threatening emergency from one with overanxious parents and all those in between. The workup and management of common noncritical problems in neonates will be discussed.

- Develop a differential diagnosis of the neonate who presents with jaundice, apnea, vomiting, and irritability.
- Discuss the management and disposition of these patients in the emergency department.

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### **FACULTY**

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# COMMON COMPLAINTS IN THE FIRST 30 DAYS OF LIFE

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## Jaundice

### Definition

Yellow discoloration of the skin, **conjunctiva**, or mucous membranes  
Reflects excess bile pigments and elevated serum bilirubin

### Pathophysiology of Jaundice

Unconjugated bilirubin is largely a product of converted **heme** from  
**senescent RBCs**

Transported by albumin and the reticuloendothelial system to the liver  
Conjugated with glucuronide within the hepatocyte (UDG Transferase)  
Secreted into bile and the small bowel where conversion to  
urobilinogen takes place, excreted into stool and urine

### General Concepts

Unconjugated hyperbilirubinemia (UB) may be physiologic or  
pathologic

High levels of UB may cause kernicterus in certain scenarios:  
prematurity, anemia, sepsis, hypoxia, or acidosis

Conjugated hyperbilirubinemia is almost always pathologic

## Clinical Definitions

Hyperbilirubinemia is direct if the direct conc is  $>2$  mg/dL or  $>30\%$  total

Indirect hyperbilirubinemia = direct  $<2$  or  $<15\%$  total

Visible jaundice is evident at bili concentrations  $>5$

## Causes of Indirect Hyperbilirubinemia

### Physiologic Jaundice

Peaks between 3-5 days of life in full terms

### Birth trauma

Cephalohematoma or scalp bruising

Swallowed maternal blood

### Polycythemia

Delayed cord clamping

Maternal fetal transfusion

### Hemolysis

Rh or ABO incompatibility

### RBC defects (hemolysis on smear)

#### G6PD deficiency

Severe (Asians) and mild (AA) forms

Diagnosed by enzyme assays

#### Hereditary spherocytosis

Defect in RBC membrane

Positive family history

### Intestinal Obstruction

Increases enterohepatic circulation of bilirubin

Duodenal atresia, annular pancreas, meconium plug, pyloric stenosis

### Breast Milk Jaundice

Generally at the end of the first week of life

BM inhibits glucuronyl transferase and produces pregnanediol

Diagnosis and Rx by cessation of breast feeding with subsequent fall in bili

### Approach to the Jaundiced Infant in the ED

Distinguish from carotinemia, yellow staining of skin from yellow or orange vegetables (sclera spared)

Perform a directed history

Feeding patterns- decreased intake, vomiting, lethargy, distention (obstruction)

Breast milk history

Family history of RBC defects

Maternal and infant blood types if known

Directed physical exam

Signs and symptoms of sepsis or obstruction

Hepatosplenomegaly

Cephalohematoma or bruising

### Pertinent Labs

Total and direct bilirubin  
 CBC with smear (hemolysis)  
 Blood type

Coombs testing
+ Coombs and anemia= blood group incompatibility
- Coombs and anemia= RBC defects
- Coombs without anemia= breast milk jaundice

### **Management of the jaundiced infant**

Unconjugated bili > 15, admit for phototherapy  
 Presence of increased bili, anemia or hemolysis= consult  
 Hyperbilirubinemia secondary to pathologic processes resolves  
 with correction of the underlying illness (infection, UTI,  
 obstruction)

### **A Word About Conjugated Hvoerbilirubinemia**

Almost always pathologic  
 Hepatic disorders (biliary atresia)  
 Perinatal Infections (toxoplasmosis, rubella, CMV, HSV, bacterial, UTI)  
 Metabolic disorders (galactosemia, alpha 1 antitrypsin deficiency, cystic  
 fibrosis)

<b>Cough</b>
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Usually benign in nature, overestimated by the parent

If documented however, may indicate the presence of underlying pathology

In many cases is the first symptom of a congenital airway or pulmonary anomaly

<b>Congenital Causes of Cough</b>
Vascular ring
Lobar Emphysema
Pulmonary Cysts
Chronic Aspiration Syndromes (TEF, GER)
Foreign Body Aspiration
Cystic Fibrosis

<b>Infectious Causes of Cough</b>
Chlamydia
Pertussis
Bronchiolitis
Bacterial Pneumonia

**Pertussis**

Infection secondary to inhalation of *B. pertussis*

Duration of illness: 6-8 weeks

Three stages of symptomatology: Catarrhal, Paroxysmal, Convalescent

Incubation period = 6-20 days

### Catarrhal Stage

Rhinorrhea, lacrimation  
Mild cough, conjunctival injection  
Low grade fever

### Paroxysmal Stage (2-4 Weeks)

Repetitive paroxysms of forceful coughs during a single expiration,  
followed by a massive inspiratory effort (whoop)  
Cyanosis, bulging eyes, tongue protrusion, salivation, lacrimation, neck  
vein distention  
Post-tussive emesis commonly occurs  
Attacks triggered by yawning, eating, drinking  
Infants characteristically lack the whoop

### Convalescent Stage (1-2 Weeks)

Less frequent paroxysms, decreasing in severity  
The cough may persist for months

### Complications

Otitis media  
Pneumonia: responsible for 90% of mortality in children < 3 years of  
age; usually a secondary event (*S. pneumoniae*)  
Subarachnoid /intraventricular/subconjunctival hemorrhages  
Umbilical/inguinal hernias

### Clues to the Diagnosis

Witnessing a paroxysm  
Leukocytosis (20-50,000) with marked lymphocytosis (70%) during the  
paroxysmal stage  
Immunofluorescent antibody (IFA) staining of nasopharyngeal  
secretions

## Treatment

Maintain hydration

Supplemental oxygen during paroxysms

Avoid suctioning or any form of oral stimulation

Erythromycin (50 mg/kg/day) eliminates organisms from the nasopharynx in 3-4 days

Antibiotic therapy does NOT shorten the paroxysmal stage

## Disposition

Children under 6 months-admit for observation, apnea monitoring

No fixed current guidelines for the older patient

Treat all contacts

## Chlamydia

Accounts for 15-73% of afebrile pneumonia in infants 3 to 11 weeks of age

Transmission during cervical passage; rates of colonization 2-37%

Of infants colonized, 50-75% will develop conjunctivitis, 11-29% will develop pneumonia

History of conjunctivitis or mucoid rhinorrhea, followed by gradually worsening tachypnea and staccato cough

Most infants are afebrile

Auscultation-coarse rales or minimal wheezing

CXR-hyperexpansion and interstitial infiltrates

## Chlamydia

WBC normal; 70% have eosinophil counts > 300/mm<sup>3</sup>

Diagnosis by perinatal history, clinical picture and eosinophilia

Direct culture from NP swabs (antigen tests are not reliable)

Treatment

Erythromycin estolate- 10mg/kg every 8 hours for 14 days

## Bronchiolitis

Transmitted by direct secretive contact and aerosolization of

Respiratory Syncytial Virus (**RSV**)

May also be caused by parainfluenzae, adenovirus or influenzae virus

The virus is replicated in the small bronchiole

Causes intramural secretions and airway edema

Certain individuals respond by producing IgE-RSV complexes which facilitate the generation of a bronchospastic component

May explain why certain individuals have a more severe presentation and respond to bronchodilators

A wintertime disease

Incidence of 11.4/100 children during the first year of life

More common in poor, crowded, smoke exposed, non breast-fed infants

## Clinical Picture

Prodromal URI for 3-5 days with a worsening cough, decreasing oral intake

Fever is low grade but may be elevated in the presence of OM (quite common in RSV)

Usually an older sibling in the home has a "cold"

Varying degrees of distress- from rhinorrhea and mild cough to full blown ventilatory failure

Tachypnea, retractions, hyperinflation, wheezing and rales

Liver displacement secondary to hyperinflation

Dehydration and listlessness in severe cases

CBC is usually nonspecific

Oximetry is mandatory on presentation and throughout ED course

CXR

Protean findings, including hyperinflation with flattened diaphragms, interstitial edema, atelectasis/infiltrates, peribronchial cuffing

RSV washings are the gold standard

#### RSV Treatment

Supplemental oxygen as necessary (canula or mask)

Cardiac and apnea monitor if ill appearing

Intravenous fluids if indicated

Trial of nebulized Albuterol

Will be helpful in some patients

If response is favorable, administer every 20-30 minutes

Literature is mixed regarding efficacy of Beta 2 agonists in RSV

Oral/IV steroids

No controlled studies

May be helpful in bronchospastic patient subset

Prednisone, 2mg/kg/day PO or Solumedrol, 1mg/kg/dose IV

Ribovarin

#### RSV Disposition

Most resolve as outpatients

Many do well at home on Beta 2 agonists (liquid preparations) +/-

Steroids

## Gastrointestinal Complaints

### Feeding Concepts in Infancy

“Baby’s in charge”- stable pattern by 30 days

To the parent, food= love= weight gain= wellness

Predictable 10% loss from birth weight in first 3-5 days (water)

Gain of 1/2- 1 ounce per day thereafter within the first 2 months

Caloric requirements= 120 kcal/kg/day

Adequate weight gain rules out many pathologic processes

### Soitting

Very common, due to poor post prandial positioning and undeveloped lower esophageal sphincter tone

Presence of choking, coughing and resp distress points to chronic aspiration syndromes

Workup for severe cases= pH probe, fluoroscopy

Management of chronic GER= thicken feeds, positioning, proprolsid

### Diarrhea

Marked variation in stool consistency from infant to infant

Pathogenic forms

*Parenteral* diarrhea- as a nonspecific sign of infection (OM, UTI)

Infectious forms- viral (rotavirus, enteroviral), bacterial (rarely)

Rarely bowel ischemia

Bloody- milk allergy or post antibiotic

Overfeeding or improperly mixed formulas

## Constipation

Quality not quantity

Breast fed have variable patterns

No stool since birth- atresia, meconium ileus, Hirschsprung's

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## **Vomiting**

### Volvulus

May occur anytime from in utero to adulthood

Most cases within the first month of life

Twice as common in males, familial predilection

Congenital abnormality of the fixation of the mesentery (Ladd's bands) and in normal bowel migration, leading to malrotation

Signs and symptoms

Bilious vomiting

Volume depletion/ signs of shock

Grunting respirations

Jaundice (113)

Bloody stools (late)

Plain films

Dilated stomach and proximal duodenum with no gas distal to the obstruction

Air contrast may reveal the double bubble sign

#### Upper GI

More useful than BE as 10% will have high cecum

Reveals narrowing at site of obstruction

Spiraling of small bowel about the superior mesenteric artery (SMA)-  
corkscrew appearance

#### Management

ABC's

Fluid resuscitation

NPO/ NG tube

Antibiotic coverage (Ampicillin/ Clindamycin/ Gentamycin)

#### Pyloric Stenosis

First described by Hirschsprung in 1988

Unknown etiology; 4-5/1000 births

More common in males (5:1) with familial predilection

Frequently occurs in first born males

#### Pathophysiology

Pyloric hypertrophy develops in the first months of life

#### Signs and Symptoms

Vomiting (+/- projectile)

Hungry infant who vomits soon after eating

Bilious vomiting is very rare

May see peristaltic waves moving from left to right after feeding

Eventual dehydration and constipation

Jaundice may ensue (1-2%)

May palpate an olive (round mass) in right upper to mid quadrants,  
next to right border of **rectus** muscle (85%) of patients

#### Laboratory Anomalies

Volume depletion (BUN)

Hypokalemic, hypochloremic metabolic alkalosis

From emetic losses of H<sup>+</sup> and Cl<sup>-</sup>

#### Ultrasound

Increased diameter >17 mm

Increased muscle thickness > 4 mm

Indeterminate ULTZ consider UGI

“String sign” from narrow pylorus

#### Management

ABC's

NPO/ NG suction

Fluid resuscitation as needed

Prompt surgical consultation

May delay repair 24-36 hrs to rehydrate

#### **Intussusception**

Prolapse of one part of the intestine into the other

Most common cause of intestinal obstruction in children <2 years

Usually presents between 3-12 months

Male to female 2: 1

<b>Pathophysiology</b>
Idiopathic (95%)
Lead point
Polyps
Meckel's
Lymphoma
HSP
Hematoma

### Signs and Symptoms

Classic Triad (3 findings in 21%, 2 in 70%)

Colicky abdominal pain (80%)

Striking- often causes knees to pull up

Vomiting (often bilious)

Bloody stools (currant jelly)- 50%

May present as lethargy, pallor, and ALOC

Dance's sign- RUQ mass with no bowel in RLQ

Intussusception may actually be felt in the rectum

### Plain films

Show signs of obstruction with AF levels and decreased gas distal to obstruction

### ULTZ

May be used as a screening tool in low risk patients

Reveals radiolucent donut or pseudokidney

### Barium Enema

Diagnostic and therapeutic in 50-90%

Gold standard

Reveal cervix like mass- coiled spring appearance with reduction

## Crying

### General Concepts

Any unpleasant sensation may cause an infant to cry ranging from hunger to the desire for contact or company to severe illness

Many common minor illnesses need to be excluded by careful Hx and PE

Research on 50 infants studied prospectively from birth

Hours crying per day = 1 at 2 weeks, 3 at 6 weeks, 1 at 12 weeks, in spite of feeding, burping, rocking, and frequent diaper changes

Areas to Focus on in the PE	
Vitals	fever, or tachypnea
Head	trauma, the fontanel
Fundi	retinal hemorrhages
Corneas	foreign body or abrasion
Ears	OM
Abdomen/rectal	obstruction (Hirsch), blood (intussusception), anal fissure
Genitalia	hernia, hair tourniquet
Digits	hair tourniquet
Urine	tox screen for cocaine metabolites

### Establish a Working Definition of Colic

Chronic daily pattern of paroxysmal irritability

Onset 2nd or 3rd week

Lasts for several hours, more commonly late afternoon or evening

**Infant** Will draw up legs, distend abdomen (swallowed air from crying), pass flatus

Colic in the ED

No cure available

Studies of pharmacologic therapies - sedatives=placebo

Empathy, counseling

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## Rashes

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### Cutis Marmorata

Refers to “marbled skin”

Dilation of superficial vessels very close to periphery

Seen predominately in fair skinned infants exposed to cold/heat

### Seborrhea

Considered a defect in sebum

“Cradle cap” in infancy

Greasy yellow scales are typical with the erythematous patches

Scalp involvement is common; both areas need rx

Treatment involves defoliant shampoo (Selenium) and very low strength steroids

### Candida (Monilia)

C albicans is natural flora, will flourish in certain settings

Loves intertriginous areas

May occur at corners of mouth (angular stomatitis)

Intense erythema in thigh creases and pubis with sharp margins-satellite papules and pustules

extend to leg and abdomen help make the clinical dx

Treatment with topical antifungals with/or without steroids

Oral candidiasis often seen in conjunction with it

### **Intertrigo**

Generally a benign condition

Occurs in areas with skin in natural opposition

Maceration of stratum corneum

Consider using mild 1% hydrocortisone for a few days if intensely inflamed

### **Monaolian Spots**

A harmless yet common dyschromia

Common in orientals and blacks, lower back

Often confused with nonaccidental trauma

### **Benign Gynecomastia**

Secondary to maternal hormones, often unilateral

May actually lactate ("witch's milk")

Treatment:

No I and D

Warm compresses

Resolves in 2 week

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## Umbilical Problems

### Umbilical Granuloma

Vestigial remnant after cord separation

No discharge

May be confused with patent urachus or omphalomesenteric duct

Treat with silver nitrate applications

### Omphalitis

Circumferential redness, warmth and tenderness around base of cord

Foul drainage may be seen

Caused by Strep A, Staph aureus, Gram negative rods

Treatment

Admission for IV antibiotics after full septic work up

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## Apnea or "Acute Life Threatening Events" (ALTE)

### General Concepts

Apnea is a common "Final Pathway" in response to various

physiologic and pathologic processes in the young infant

Usually the result of an immature CNS, decreased respiratory reserves, and susceptibility to infections

Defined as a respiratory pause > 15 seconds or of any duration if there is associated pallor, cyanosis, or bradycardia

Distinguished from periodic breathing, a common respiratory pattern in young infants= cycles of short respiratory pauses followed by periods of rapid breathing

#### Respiratory Concepts- Infants vs. Adults

Adult model= Decreases in  $pO_2$  cause increases in RR

Neonatal model= Decreases in  $pO_2$  cause increases in RR, followed by depression of respiratory drive and decreased responsiveness to increases in  $pCO_2$

Other contributors in the infant include hypoglycemia, anemia, and fatiguable diaphragmatic musculature

#### Differential Diagnosis of Apnea

CNS	infection, seizure, IVH, breath holding
Upper airway	reflux, congenital anomaly
Lower airway	pneumonia, RSV
Other	hypoglycemia, hypocalcemia, anemia, sepsis, arrhythmias, SIDS

#### Determine the Significance of the Episode

Where did it take place? (sleep, trauma)

How long did it last? (> 10 seconds)

Awake or asleep? (worse if asleep)

Associated color change? (pallor or cyanosis)

Associated movements or changes in tone? (seizure, "he looked dead")

Any resuscitative efforts?

When last fed?- (reflux)

### Evaluation of Apnea in the ED

Stabilize ABC's (rarely necessary)

Check chemstrip

Labs- CBC, SMA7, Calcium

Consider CSF and CT

ECG

Almost always admitted

### Evaluation of Apnea in the Hospital

24 hour cardiogram, pneumogram, pH probe

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## **Fever**

### Factors Related to Neonatal Infections

Diversity of transmission

Transplacental

Contaminated amniotic fluid

Aspiration of vaginal secretions

Breakdown of barriers (monitors, umbilicus, catheters)

Immunologic Immaturity

Decrease functions of complement system, chemotaxis, and  
macrophage function

Confusion of Presentation

In early stages subtle and non specific

Hypothermia/ Poor PO

Irritable or lethargic

### Clinical Manifestations

General- Alteration in temp, "sleepy" poor PO

GI- distention, V and D, hepatosplenomegaly

Respiratory- apnea, dyspnea, work, flare or grunt, cyanosis

Cardiovascular- pallor or mottling, tachycardia, hypotension,  
bradycardia

CNS- irritable/lethargic, seizures, reflex anomalies, full fontanel, high  
pitched cry

Hematologic- jaundice, pallor, petechiae or purpura, bleeding

### The Pathogens

Viral- CMV, HSV, hepatitis, HIV

Bacterial- Group B strep (30%), E coli (30-40%), Gram - enterics (5-  
20%), Gram positives (10%), Listeria

### Antibiotic Choices

Ampicillin- 100 mg/kg/ day divided q 6 hours

Gentamycin- 7.5 mg/kg/day divided q 8 hours

Cefotaxime- 200 mg/kg/day divided q 6 hours

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## Congestive Heart Failure

Inability of cardiac output to meet metabolic demands

Predominantly the result of congenital heart disease

95% present within the first year of life

Vast majority within the first few months

### Pathophysiology of CHF

Preload (diastolic loading of the ventricles)

Afterload (systolic loading of the ventricles)

Cardiac contractility

Heart rate

### Etiologies of CHF

Volume Overload

Left to right shunt (VSD,PDA)

Anemia

Pressure Overload

LV outflow obstruction (AS, coarct)

Myocardial Dysfunction

Dysrhythmia

Infection

Endocrine

Autoimmune (SLE, ARF)

Poisoning (TCA, digitalis)

### CONGENITAL HEART DISEASES **PRESENTING** AS FAILURE IN INFANCY

LESION	USUAL TIME OF ONSET OF FAILURE
Hypoplastic left heart syndrome	First week
Coarctation of the aorta	First week
Complete atrioventricular canal	First 2-3 weeks
Ventricular septal defect	Weeks 2-12
Patent ductus arteriosus	Weeks 1-4
Complex lesions	Weeks 1-12

**CONGENITAL LESIONS USUALLY ASSOCIATED WITH CYANOSIS**

<b>LESION</b>	<b>USUAL TIME OF ONSET OF CYANOSIS</b>
Transposition of the great arteries	Birth to first week
Total anomalous pulmonary venous return	First week
Tricuspid atresia	Weeks 1-4
Ebstein's anomaly of the tricuspid valve	First week
Tetralogy of <b>F</b> allot	Weeks 1-12
Severe pulmonic stenosis	Weeks 1-4

**Symptoms**

- Poor feeding, accompanied by increases in respiratory work
- Poor weight gain (FTT) secondary to excessive metabolic work and diminished overall caloric intake
- Diaphoresis is common (adrenergic discharge)
- Prone to lower respiratory infections

**Signs**

- Pallor, diaphoresis and tachypnea
- Tachycardia, gallop and cardiomegaly
- Hepatomegaly and edema are late findings
- Cool, mottled extremities with prolonged capillary refill

## Studies

Cardiomegaly on CXR (CT ratio greater than 0.55 in infants)

Pulmonary edema (fluffy infiltrates and Kerley lines)

ECG may not be very useful

Metabolic acidosis, hyponatremia, hypoglycemia

## Management of CHF

ABC'S

Thermoregulation and supplemental oxygen

Drug therapy

Digoxin

Dopamine

Diuretics

Vasodilators

Morphine Sulfate

## Prostaglandins

Employed in cases of ductus-dependent lesions

Severe coarct

Hypoplastic left heart

Interrupted aortic arch

Prostaglandin E1 is the drug of choice

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## Rapid Heart Rate

### Arrhythmia Mechanisms

Reentry pathways

Ectopic pacemakers (automaticity)

### Reentry phenomena

- Propagating impulse fails to die out after normal cardiac activation
- Finds a second pathway, which is no longer refractory, reexciting the heart
- May involve only one beat (PVC) or more (SVT)
- Utilizes accessory pathway which bypasses the AV node
- Multiple accessory pathways= **WPW** Syndrome

### **Common Tachycardias**

#### Supraventricular Tachycardia

- The most common symptomatic dysrhythmia in infants and children
- May be secondary to an alternative pacemaker or as part of WPW Syndrome
- Common in infancy, but occurs in adolescents as well

#### ECG Features of **SVT**

- The heart rate varies widely
- Rates in infancy may reach 300
- Adolescents reach 120-160
- QRS complexes are narrow in more than 90% children with SVT
- Tachydysrhythmias with widened QRS should be considered to be ventricular in origin

#### SVT vs Sinus Tachycardia

- HR in infants rarely exceeds 220 beats/minute
- Lack of beat to beat variability in SVT
- SVT converts in response to therapy
- P waves are visible in 50% infants with SVT

## Acute Therapy of SVT

### Cardioversion

Reserved for patients in shock or CHF

Initial dose= 0.5-1.0 watt sec/kilogram

Subsequent doses may be doubled

### Vagal

The diving reflex is most successful in infants

Utilize an iced washcloth or a bag of ice, held over the infants

nose and mouth for 15 to 30 seconds

Gagging and eyeball pressure should be avoided

### Digoxin

Effective, safe, and the longest track record

Major drawback is its onset of action

IV dose= loading (15mics/kg) followed by 2 more doses 4-5 hours apart

### Adenosine

An endogenous neucleoside, causes transient block thru the AV node

May be the drug of choice in infants with SVT

Very short half-life (10 seconds)

Initial dose= 0.1 mg/kg, may be doubled

### Verapamil

Slows conduction and prolongs the refractory period of the AV node

Should be avoided in infants (hypotension) and in rhythms with a bypass tract (wide-complex tachycardia)

Dose= 0.1-0.3 mg/kg