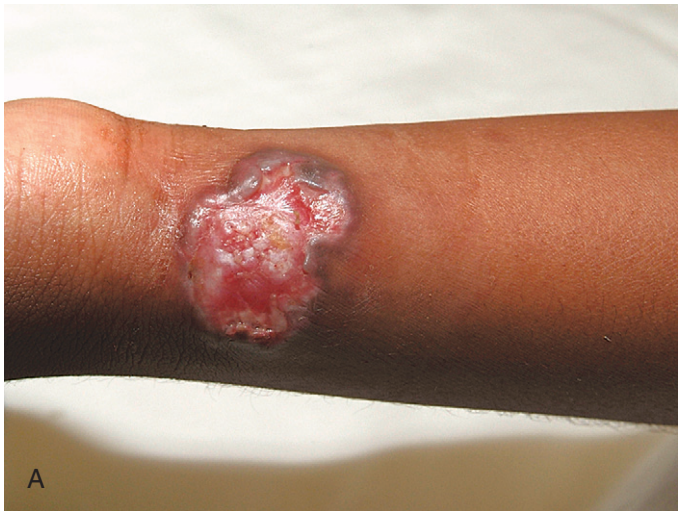
Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.



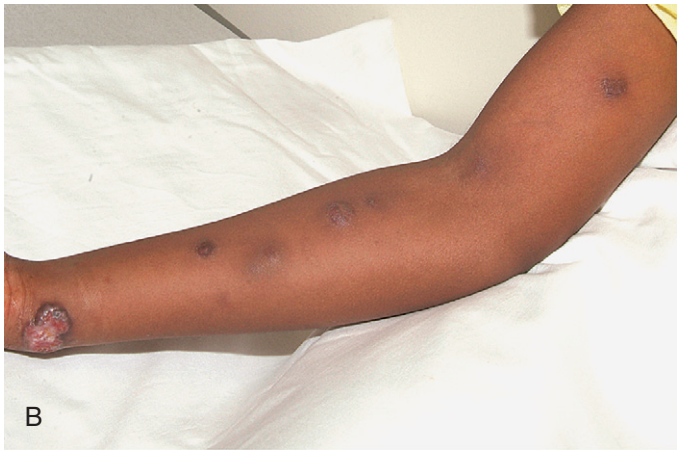


Figure 22-2. One adolescent girl with a 4-month history of an ulcerating skin lesion on her wrist (A) and nodular lymphangitis (B). *Sporothrix schenkii* was isolated. Her only exposure to roses was from a florist. (Courtesy of Sarah Long, M.D.)

Therapy

Because *Streptococcus pyogenes* is the predominant cause of acute lymphangitis, penicillin is the preferred initial treatment. Children with mild disease can be treated with oral penicillin V (25 to 50 mg/kg per day). Those with prominent systemic symptoms have a high risk of concurrent bacteremia and should receive intravenous therapy (penicillin G, 100,000 to 250,000 U/kg per day). Penicillin is also the drug of choice for *Pasteurella* lymphangitis and *Spirillum minor* rat-bite fever.

Lack of familiarity with the syndrome of nodular lymphangitis often leads to delays in correct diagnosis and inappropriate antibiotic therapy directed at pyogenic bacteria. Conservative measures, such as local application of a heating pad, may contribute significantly to resolution of lesions associated with sporotrichosis, *M. marinum* infection, or cutaneous leishmaniasis. Itraconazole (100 to 200 mg/day) has become the drug of choice for lymphocutaneous sporotrichosis, supplanting saturated solution of potassium iodide (SSKI) because of a lower toxicity.[27] Treatment should be continued for 4 weeks beyond resolution of lesions (2 to 3 months total). Antimicrobial agents (e.g., trimethoprim-sulfamethoxazole, minocycline, rifampin plus ethambutol) are variably effective against *M. marinum* lymphangitis, and some excisional surgical debridement is often required. *Nocardia* infection generally responds readily to a sulfa drug; amoxicillin-clavulanate is an option for patients allergic to sulfa drugs. Cutaneous leishmaniasis often heals spontaneously with topical care, but therapy with pentavalent antimony should be used if lesions evolve to the mucocutaneous form.

CHAPTER 23

Respiratory Tract Symptom Complexes

Sarah S. Long

MUCOPURULENT RHINORRHEA

Mucopurulent rhinorrhea, or purulent nasal discharge, denotes nasal discharge that is thick, opaque, and colored. It occurs at any age, usually as a manifestation of self-limited, uncomplicated viral upper respiratory tract infection (URI). Mucopurulent rhinorrhea is most problematic in children younger than 3 years because of: (1) protracted course and frequent recurrence, especially in those in out-of-home child care;[1] (2) parental concern about and misperception of etiology; and (3) overprescription of antibiotics by healthcare providers.[2–5] Occasionally, this symptom is a clue to diagnosis of a treatable bacterial infection or underlying condition.

Acute, sporadic mucopurulent rhinorrhea has an infectious cause and almost always is the manifestation of the uncomplicated "common cold" due to rhinovirus, coronavirus, or other circulating viruses.[6] When the problem is chronic or recurrent, or persistent and unilateral, broader underlying anatomic, obstructive, immunologic, and allergic disorders are considered (Table 23-1).[7–10] Onset in an infant younger than 3 months heightens suspicion of anatomic anomaly, ciliary dyskinesia, or cystic fibrosis. Accompanying sinusitis, otitis media, or pneumonia raises consideration of an immunologic deficiency (especially immunoglobulin deficiency or dysfunction, as in

TABLE 23-1. Causes of Mucopurulent Rhinorrhea

	Chronic or Recurrent	
Acute	**Underlying Conditions**	**Obstructing Lesions**
Viral nasopharyngitis	Allergy[a]	Polyps
Bacterial sinusitis	Medications[a] (antihypertensives, oral estrogens, aspirin and nonsteroidal anti-inflammatory drugs)	Congenital nasal anomalies (choanal atresia or stenosis, Tornwaldt cyst, deviated septum)
Acute otitis media	Pregnancy[a]	Neuroembryonal mass (dermoid, encephalocele, glioma, teratoma)
Streptococcal nasopharyngitis	Hypothyroidism[a]	Tumor (hemangioma, angiofibroma, neurofibroma, lipoma, craniopharyngioma)
Anaerobic bacterial nasopharyngitis (nasal foreign body)	Rhinitis medicamentosa[a] (α_1-adrenergic agonists)	Neoplasm (lymphoma, rhabdomyosarcoma, nasopharyngeal carcinoma)
Adenoiditis	Immunoglobulin deficiency	
Syphilis	Human immunodeficiency virus infection	
Pertussis	Cystic fibrosis	
	Ciliary dyskinesia	

[a]Rhinorrhea is characteristically clear, but opaque white discharge is not unusual.

hypogammaglobulinemia or human immunodeficiency virus (HIV) infection), neutrophil defect, cystic fibrosis, or ciliary dyskinesia. URIs are conspicuously severe in such instances, with recrudescence almost immediately after discontinuation of antibiotic therapy. Unilateral nasal discharge and obstruction should prompt investigation for a foreign body, mass lesion, or unilateral posterior choanal atresia.

Table 23-2 shows differentiating features of important or common causes of acute mucopurulent rhinorrhea; allergic rhinitis is included because it is frequently part of the differential diagnosis in older children and adolescents.

Causes of Acute Mucopurulent Rhinorrhea

Viral Nasopharyngitis

In uncomplicated viral nasopharyngitis or rhinitis, nasal discharge is initially clear but can become white, yellow, or green (related to mucous secretions, dryness, blood, exfoliation of damaged epithelial cells and cilia, and leukocytic inflammatory response). Presence of high fever and persistence of discharge depend on the specific viral cause but are more common in uncomplicated infection than generally perceived.

In a study of hospitalized children, more than 50% of those with uncomplicated adenovirus, influenza, parainfluenza, or respiratory syncytial virus infection had temperatures >39°C, and 12% had temperatures >40°C; height of fever in these children was not different from that in children with serious bacterial infection.[11] Fever persisted for 5 days or longer in 37% of the children in the study; 20% to 30% of those with adenovirus or influenza A infection had fever for 7 days or longer. In another study, nasal discharge or congestion associated with uncomplicated URI persisted for 6.6 days in 1- to 2-year-old children who were in home care and for 8.9 days in children younger than 1 year in daycare centers.[1] In this study, 13% of 2- to 3-year-old children in out-of-home childcare had symptoms for more than 15 days.

The bacteriology of nasopharyngeal flora in children with uncomplicated viral respiratory illnesses, mucopurulent rhinorrhea, acute otitis media, and sinusitis has been evaluated and compared

TABLE 23-2. Differentiating Among Causes of Nasal Discharge[a]

	Viral Nasopharyngitis[1,11–15]	Acute Bacterial Sinusitis[16,17,24]	Streptococcal Nasopharyngitis[25]	Foreign Body-Related Rhinitis (Bacterial)[18]	Allergic Rhinitis[10]
HISTORY					
Peak age	Peak in first 2 years after "new recruitment" into childcare or school	Any	< 3 years	< 3 years	> 2 years; peak in adolescence
Onset	Dryness, burning in nose or nasopharynx	Insidious, with cough day and night; occasionally, acute, febrile, toxic	Insidious; occasional acute, febrile, toxic	Insidious	Seasonal; precipitants
Associated symptoms	Nasal congestion, sneezing malaise	Malodorous breath; head or facial pain, edema		Malodorous breath ± hyponasal voice	Sneezing; nasal or palatal pruritus; tearing; snoring
Fever	Yes/no	No/yes	Low/high	No	No
Duration of discharge	3–8 days	≥ 10 days	> 5 days	Chronic	Chronic, recurrent
PHYSICAL EXAMINATION					
Associated findings	Red, excoriated nares; sometimes, acute otitis media	Periorbital swelling, facial tenderness; mucopurulent postnasal discharge	Anterior cervical lymphadenitis; impetiginous lesions below nose	Mouth-breathing	Transverse nasal or lower eyelid crease; periorbital hyperpigmentation; cobblestone conjunctivae or posterior pharynx
Character of discharge	Clear or colored, watery or thick	Thick, colored	Thick, colored	Unilateral, purulent, putrid blood-stained	Watery, clear, or white
Rhinoscopy	Hyperemic mucosa; dry or glazed early, edematous later; crusted discharge	Normal mucosa; discharge from middle meatus	Normal, hyperemic, or excoriated mucosa	Identifiable object (button, pit, nut), boggy mass (vegetable), or rhinolith	Pale or blue, edematous turbinates
DIAGNOSTIC TESTS	None; nasal smear shows polynuclear and mononuclear cells ± inclusion bodies, pyknotic epithelial cells	None; sinus radiograph (> 6 years of age)	Nasopharyngeal culture for streptococcus only	Rhinoscopy	Nasal smear shows goblet cells and eosinophils; skin test or radioallergosorbent test (RAST)
CAUSE	Multiple agents, depending on age and season	*Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis*	*Streptococcus pyogenes*	Normal nasopharyngeal facultative and anaerobic bacteria	Allergens in predisposed individual
THERAPY	Saline nasal drops, humidification; amoxicillin if acute otitis media	Amoxicillin; β-lactamase stable agent	Penicillin V	Removal of obstruction; amoxicillin-clavulanate or clindamycin if tissue or sinus complication	Avoidance; oral antihistamine/ decongestant; or topical corticosteroid; cromolyn

[a]Superscript numbers indicate references.

with that in normal children.[6,12–23] Viral infection is associated with acquisition of new serotypes of *Streptococcus pneumoniae* and with temporally increased risk of acute otitis media.[21] Quantitative, and some qualitative, differences in nasopharyngeal flora have been found in children with purulent nasopharyngitis (and uncomplicated viral upper respiratory illnesses), with excessive isolation rates reported for *S. pneumoniae* and *Haemophilus influenzae,*[13,18] *Peptostreptococcus* spp., *Fusobacterium* spp., and *Prevotella melaninogenica.*[18,19] The significance of such findings is unclear; isolation of such organisms may reflect exuberant proliferation in virus-induced inflammatory mucus or acquisition of a more robust specimen than is collected in healthy subjects. Furthermore, "high" rates of isolation of *S. pneumoniae* in 25% to 46% of subjects do not exceed those in normal young children when fastidious technique is used.[22]

Only two systematically performed studies on the course of mucopurulent rhinorrhea have been published. In one study, prospective evaluation showed that there was no difference in duration of illness or complications in children with clear or purulent nasal discharge.[14] In a placebo-controlled, blinded study of 142 children 3 months to 3 years old with mucopurulent rhinorrhea of any duration, antibiotic therapy (cephalexin), systemic use of an antihistamine-decongestant, or both had no effect on the course or complications of mucopurulent rhinorrhea.[12] In a small pilot study of 13 children younger than 2 years whose purulent nasal discharge had persisted for at least 10 days without improvement, amoxicillin-clavulanate (40 mg/kg per day divided into 3 doses for 10 days) was significantly associated with resolution of symptoms in comparison with placebo.[15]

Response to antimicrobial therapy does not necessarily validate an entity of bacterial nasopharyngitis, however; it seems more likely that children with such responses have an incomplete symptom complex of ethmoid sinusitis. Acute bacterial adenoiditis is postulated to be another cause of purulent nasal discharge when: (1) tympanic membranes are normal; (2) *S. pyogenes* is not found in culture specimens; and (3) radiographs show an enlarged adenoid shadow but no sinus abnormality.[23,24] Critical study has not been performed to validate this entity. A comparison of clinical and radiographic assessments of adenoidal enlargement may be an important first step.[25]

Bacterial Sinusitis

Mucopurulent rhinorrhea of 10 or more days' duration without improvement (or recrudescence after improvement) that is associated with daytime cough (which is frequently worse at night), or malodorous breath, facial pain, edema, headache, or fever is highly suggestive of paranasal sinusitis.[17,26] Sinus radiographs show significant abnormalities in nearly 90% of children 2 to 6 years old with such findings (see Chapter 34, Sinusitis), and thus support the validity of clinical diagnosis without need for imaging.

Streptococcal Nasopharyngitis

In children younger than 3 years, *S. pyogenes* has been associated with high fever, toxicity, and clear rhinorrhea or indolent infection with irregular fever and purulent nasal discharge, sometimes with associated excoriation of nares or tender anterior cervical lymphadenitis.[13,18,25] In a streptococcal outbreak studied in a childcare facility for school-aged and young children, 26% of children younger than 3 years were affected, but pharyngitis was predominant, with no case of nasal streptococcosis.[27]

Other Infectious Causes

Bacterial nasopharyngitis associated with nasal foreign body is typified by the young age of the patient and putrid, commonly blood-stained unilateral nasal discharge. Fever is unusual unless infection has spread to contiguous sinuses or distant sites. *Prevotella, Fusobacterium,* and *Peptostreptococcus* spp. as well as facultative flora are responsible. Nasal discharge can be the first manifestation of congenital syphilis and a later finding in nasal diphtheria, in which discharge is putrid and sanguineous and contains pieces of pseudomembrane.

Allergic Rhinitis

Allergic rhinitis typically begins in the second decade of life, is uncommon before age 3 years, and may be rising in incidence in children between these ages. Diagnosis is suspected from the season, environmental precipitants, personal and family history of allergy, other associated symptoms and physical findings, and the response to specific interventions of avoidance or pharmacotherapy (see Table 23-2). Nasal secretions are usually clear or whitish. Diagnostic usefulness of nasal cytologic analysis is controversial.[10,28] Relative eosinophilia (above 20%) is suggestive but not diagnostic of allergic rhinitis. The findings in vasomotor rhinorrhea, which is thought to be due to increased parasympathetic tone of the nasal mucosa, are similar to those in allergic rhinitis, except that symptoms of allergy and nasal eosinophils are absent. In severe allergic rhinitis, the inflammatory phase of response can cause accumulation of neutrophils and mononuclear cells.[10]

Management of Acute Mucopurulent Rhinorrhea

In the vast majority of children with purulent nasal discharge (even if thick and green) of up to 1 week in duration, history and setting of illness, associated symptoms, and physical findings suggest uncomplicated viral URI. Antimicrobial therapy is inappropriate unless acute otitis media or sinusitis is diagnosed from additional findings (see Chapter 34, Sinusitis). Symptomatic therapy with saline nose drops or lavage facilitates expulsion of secretions and provides humidification. Its effectiveness reduces parental pressure to prescribe an antibiotic.[29]

If mucopurulent rhinorrhea persists for more than 5 days, and especially if some findings (e.g., anterior cervical lymphadenitis, scarlatiniform rash, excoriation around nostrils) or the epidemiology heightens the likelihood of group A streptococcal disease, nasopharyngeal specimens should be obtained for culture of *S. pyogenes* only. If findings are positive, penicillin V is given for 10 days. Routine culture for, or recovery of, *S. pneumoniae, H. influenzae, Moraxella catarrhalis*, or *Staphylococcus aureus* has no meaning and is an opportunity for misinterpretation.

If mucopurulent rhinorrhea persists for more than 10 days without diminution, and especially if other symptoms are present, paranasal sinusitis is likely. Nasal mucosa is examined after use of single or second (5 minutes after the first) application of a topical vasoconstrictor such as oxymetazoline.[15] If purulent secretions flow from the middle meatus, the diagnosis of acute sinusitis is confirmed. Signs of allergic rhinitis can also be confirmed. Radiographs may be helpful in patients older than 6 years to confirm sinusitis (or possibly to suggest adenoiditis). Pending definitive efficacy studies, many clinicians would treat children who have purulent nasal discharge of greater than 10 days' duration as for acute sinusitis, usually with amoxicillin initially. When antimicrobial therapy is effective, substantial improvement of symptoms is expected within 48 to 72 hours. Therapy is continued for 1 week beyond complete resolution of respiratory symptoms.

STRIDOR

Characteristics

Stridor is a rough, crowing sound caused by passage of air through a narrowed upper airway, which includes the extrathoracic trachea, larynx, and hypopharynx. Because the extrathoracic airway normally narrows during the inspiratory phase of respiration, stridor due to upper-airway disease occurs during inspiration (or is more pronounced during inspiration if severe narrowing causes obstruction during

inspiration and expiration). Because the intrathoracic trachea normally narrows during expiration, obstruction of the intrathoracic trachea, such as that due to extrinsic compression of vascular ring or intraluminal obstruction of foreign body, inflammation, or tracheomalacia, causes a loud noise, acoustically like stridor, heard during both phases of respiration but more pronounced on expiration. Extrathoracic obstruction (inspiratory stridor) is associated with prolonged inspiration and underaeration of the chest, whereas intrathoracic obstruction (expiratory stridor or wheezing) is associated with prolonged expiration and overinflated chest. Stridor can be associated with mild tachypnea, but a respiratory rate > 50 breaths/minute should not be ascribed to upper-airway obstruction alone.

The timbre of the stridulous sound provides a clue to etiology; for example, (1) the high-pitched, fixed, dry sound of congenital subglottic stenosis; (2) the wet, rhonchal changing sound of inflammatory laryngotracheitis; and (3) the low-pitched, vibratory, somewhat positional sound of laryngomalacia. Associated voice changes are useful in specifying disease as well. Vocal cord paralysis causes a weak, dysphonic cry; supraglottic obstruction, a muffled voice; and laryngotracheitis, hoarseness or aphonia, frequently with a barking cough.

Etiology

Categorization of the setting and duration of stridor as acute, persistent, or recurrent or episodic provides a framework for considering likely causes (Table 23-3).[30–34] Infectious agents cause most acute upper-airway obstruction, from intraluminal, epithelial inflammation or by encroachment on the airway by reactive or infected lymphoid tissue in parapharyngeal or paratracheal spaces. Fungal or viral tracheobronchitis must be considered when stridor occurs in an immunocompromised child; odynophagia and dysphagia are also commonly present.[31] Congenital anatomic abnormalities are considered, especially in infants whose persistent stridor began neonatally. Acquired obstruction can have abrupt onset and an obvious cause (such as foreign-body aspiration or necrotizing tracheobronchitis in ventilated neonates) or more insidious onset and inapparent cause (such as expanding laryngotracheal papillomas or hemangioma or an extrinsic compressing mass). The younger the infant, the more likely that sudden obstruction, apnea, or feeding difficulties overshadow a singular complaint of stridor.

TABLE 23-3. Causes of Upper-Airway Obstruction and Stridor[a]

Acute	Persistent[30]
Infectious	**Congenital**
Viral laryngotracheitis (croup)	Laryngotracheal web, cleft, cyst, hemangioma
Bacterial tracheitis	Tracheal stenosis
Epiglottitis, supraglottitis	Vascular ring
Peritonsillar, retropharyngeal, or parapharyngeal abscess	Laryngotracheal malacia
Tracheobronchitis associated with immunodeficiency[31]	Neuromuscular disorder
	Cystic hygroma
Noninfectious	**Acquired**
Angioedema	Posttraumatic tracheal stenosis
Foreign body	Foreign-body aspiration
Necrotizing tracheobronchitis in neonates[32,33]	Mediastinal mass (tumor, lymphatic, vascular)
Recurrent/episodic	Papilloma (perinatally acquired)
Spasmodic croup	Posttraumatic spinal cord, vagal or glossopharyngeal nerve, or vocal cord damage
Gastroesophageal reflux[34]	Bulbar neuropathy (infectious, postinfectious, malignant)

[a]Superscript numbers indicate references.

Clinical Features of Acute Infectious Causes

Recognition, care to avoid precipitating sudden airway occlusion, and urgent, expert intervention to establish an airway when indicated are paramount to avert disastrous outcomes of acute upper-airway obstruction. Table 23-4 shows characteristic features of infectious causes of stridor and acute airway obstruction.[35–45] Viral laryngotracheitis (infectious croup) or laryngotracheobronchitis due to parainfluenza viruses is by far the most common.[35,36] Influenza viruses, respiratory syncytial virus, adenoviruses, and other viruses typically cause symptomatic disease elsewhere in the respiratory tract, but during epidemic seasons, stridor is the predominant feature in a minority of infected children. Bacterial tracheitis is usually a complication of viral laryngotracheitis (with concordant peak age and season) but can occur at any age or as a complication of oropharyngeal surgery.[46] *Staphylococcus aureus* is the most common cause, followed by *Streptococcus pyogenes*; the role of anaerobic bacteria is less clear.[43,46] With the universal use of *H. influenzae* b vaccine, epiglottitis is a rare cause of stridor; current cases of supraglottitis are more likely to affect the aryepiglottic region and to be caused by streptococci. Parapharyngeal and retropharyngeal infections in young children must also be considered; their incidence is increasing[45,47,48] (see Chapter 30, Infections Related to the Upper and Middle Airways).

The history surrounding the onset of stridor and the patient's age and demeanor are the most helpful clues to the likely site and cause of infection. The child with viral laryngotracheitis usually has had 2 to 3 days of typical upper respiratory tract illness when cough worsens and stridor begins. The child with bacterial tracheitis has usually had a similar background illness and then has sudden high fever, toxicity, and rapid progression of airway obstruction. The young child with retropharyngeal abscess or adolescent with peritonsillar abscess has less stridor but refuses to swallow, has a muffled voice, and a guarded posture to maximize the oropharyngeal airway. Trismus is an expected and useful finding in patients with peritonsillar abscess as well as in some with lateral pharyngeal space infections of odontogenic origin.[38] Epiglottitis and supraglottitis cause the patient to guard anxiously in a sitting posture with arms back, jaw forward, and chin raised ("sniffing dog") to maximize "lift" of the epiglottis away from the airway. In contrast, subglottic, tracheal obstruction cannot be lessened by position; patients with laryngotracheitis or bacterial tracheitis thrash about with the anxiety of suffocation.

The expected course and sequelae of acute infectious airway obstruction are shown in Table 23-5.[49,50] Children with viral laryngotracheitis are less prone to sudden complete obstruction; hourly course is predictable by degree of stridor and adequacy of aeration; response to racemic epinephrine and corticosteroid therapy usually averts intubation. Establishment of an artificial airway is urgently required for almost all patients with stridor due to acute supraglottic and bacterial tracheal infection, and for many with retropharyngeal infection. The course of disease in children with bacterial tracheitis can be further complicated, because infection (and obstructive consequences) commonly extends for the length of the trachea and below.

COUGH

Cough is a critical protective mechanism to expel particulate matter from the larynx and trachea as well as a cardinal sign of infectious and noninfectious respiratory tract and nonrespiratory tract disorders. Although the vast majority of coughs are related to self-limited infections, occasional life-threatening infectious and noninfectious causes may be overlooked unless the clinician adopts a disciplined approach. Careful assessment of a pathologic cough – its onset, duration, clinical context, and association with other findings as well as its specific timbre, pattern, and productivity – frequently predicts the site of pathophysiology and narrows the differential diagnosis to a limited number of entities. Table 23-6 provides a framework for assessment and lists the differentiating features of the various causes of cough.

TABLE 23-4. Differentiating Among Infectious Causes of Upper-Airway Obstruction[a]

	Viral Laryngotracheitis[35–37]	Supraglottitis[38,39]	Bacterial Tracheitis[40–43]	Retropharyngeal Abscess[38,40,44,45]
HISTORY				
Peak age	1–2 years	3–6 years, any	2–4 years, any	< 3 years
Peak season	Late fall, late spring	Any	Late fall, late spring; any	Any
Prodrome	Viral illness	Uncommon	Viral illness	Uncommon
Onset of stridor	Gradual	Abrupt	Abrupt	Abrupt
PHYSICAL EXAMINATION				
Peak temperature (°C)	38–39	> 39	> 39	> 39
Predominant findings	Brassy cough, stridor	Toxicity, stridor	Toxicity, stridor	Toxicity, stridor
Associated findings	Bark, rhinorrhea	Sore throat, odynophagia, dysphagia, anxiety, drooling	Brassy cough, anxiety	Lethargy
Voice	Hoarse, raspy	Normal, muffled, mute	Hoarse, raspy	Muffled, mute
Position	Any; thrashing	"Sniffing dog"; still	Any; thrashing	"Sniffing dog"; still
Airway occlusion	Predictable from degree of stridor	Sudden	Sudden	Sudden
Response to racemic epinephrine?	Yes, with rebound	No	No or partial	No
LABORATORY TESTS				
Peripheral neutrophils	Normal or low	High	Immature	Immature
RADIOGRAPH				
Hypopharynx	Distended	Distended	Distended	Anteriorly displaced
Airway	Subglottic narrowing; edema cords	Swollen epiglottitis, aryepiglottic folds	Subglottic narrowing; irregular trachea ± intraluminal mass	Prevertebral soft-tissue mass with anterior displacement of airway (not valid sign if expiratory film, flexed neck)
Chest	Underaerated ± cardiomegaly	Underaerated ± cardiomegaly	Patchy parenchymal peribronchial infiltrate	Underaerated ± cardiomegaly
ENDOSCOPY	Red, edematous subglottis; crusting pseudomembrane	Red, edematous supraglottic structures	Red, edematous, eroded trachea and bronchi; purulence, pseudomembrane	Bulging mass in posterior pharyngeal wall; purulence
CAUSE	Parainfluenza viruses (epidemic); other viruses (sporadic)	*Streptococcus pyogenes, Streptococcus pneumoniae, Haemophilus influenzae* b	*Staphylococcus aureus, Streptococcus pyogenes, Streptococcus pneumoniae*	*Streptococcus pyogenes; Staphylococcus aureus;* rare *Streptococcus pneumoniae*

[a]Superscript numbers indicate references.

TABLE 23-5. Expected Course and Sequelae of Acute Infectious Upper-Airway Obstruction

	Viral Laryngotracheitis[37,49,50]	Supraglottitis, Epiglottitis	Bacterial Tracheitis[41,42]	Retropharyngeal Abscess
Artificial airway (% of cases)	< 20	> 90	> 75	≥ 75
Median intubation period	4 days	2 days	6 days	2 days
Airway occlusion after intubation	Rare	No	Yes	No
Death during hospitalization	No	No	Yes	No
Airway sequelae (% of cases)	< 3	Rare	<3	No

[a]Superscript numbers indicate references.

Cough should not be accepted as a sign of self-limited URI in infants younger than 3 months. The mnemonic CRADLE may be useful to call to mind important considerations for such patients:[51] *C*, cystic fibrosis; *R*, respiratory tract infections (especially pneumonia and pertussis); *A*, aspiration (swallowing dysfunction, gastroesophageal reflux, tracheoesophageal fistula); *D*, dyskinesia of cilia; *L*, lung, vascular, or airway malformations; *E*, edema (heart failure, pulmonary lymphangiectasia).

TABLE 23-6. Differentiating Among Causes of Cough

	Peak Age	Nature of Cough	Cough Dominant Feature?	Anticipated/Associated Findings
INFECTIONS OF THE RESPIRATORY TRACT				
Viral laryngotracheitis	> 5 years	Brassy, painful	Yes	Hoarse, raspy voice; viral URI complex[a]
Viral laryngotracheitis/ laryngotracheobronchitis	4 months–3 years	Barking, brassy	Codominant with stridor	Stridor, hoarseness, viral URI complex[a]
Mycoplasmal tracheobronchitis	Adolescent	Hacking, paroxysmal, painful	Yes	Prodromal, fever, headache, myalgia; then gradual worsening cough
Pertussis	Infancy, adolescence	Sudden paroxysm of explosive machine-gun bursts (15–30 per breath)	Yes	Bulging, watering eyes during paroxysm, posttussive emesis; skin and conjunctival hemorrhages; afebrile, without lower respiratory tract symptoms or symptoms between paroxysms
Chlamydia trachomatis pneumonia	1–3 months	Staccato, dry (single cough per breath)	Yes	History can include conjunctivitis; afebrile, tachypnea, rales
Bronchiolitis	4 months–2 years	High-pitched or grunt	No	Wheezing, rhinorrhea, respiratory distress; ± fever
Pneumonia (bacterial or viral)	Any	Wet, productive or nonproductive	Codominant with respiratory distress	Tachypnea, rales, respiratory distress; fever
Pleurodynia	Any	Inspiratory hitch; expiratory grunt	Codominant with chest pain	Chest pain; costochondral tenderness
Sinusitis	Any	Irritative; occurs in day and worsens at night	Sometimes	Mucopurulent rhinorrhea, postnasal discharge; facial pain, swelling, or tenderness; headache; ± fever
Tracheoesophagitis (fungal or viral)	Any	Irritative	No	Odynophagia or dysphagia; immune-compromised host; hoarseness; oropharyngeal lesions
Cystic fibrosis	< 2 years; any	Wet, productive; paroxysmal, hacking	Sometimes	Poor growth; persistent and recurrent sinusitis, pneumonia; digital clubbing
CARDIAC CONDITIONS				
Purulent pericarditis	Any	Grunt	Sometimes	Fever, toxicity, respiratory distress/dyspnea; displaced point of maximum impulse; muffled heart sounds
Myocarditis	Any	Grunt	Sometimes	Fatigue, dyspnea, tachypnea; ± fever
Congestive heart failure	Any	Grunt, wet, or brassy	Sometimes	Fatigue, dyspnea, sweating, tachycardia, tachypnea; ± fever; distended neck veins, liver
NONINFECTIOUS AIRWAY ABNORMALITIES				
Gastroesophageal reflux	6 weeks–6 months	High-pitched, dry	Codominant with other symptoms	Stridor, choking, gagging, irritability, arching (Sandifer syndrome) ± regurgitation, pneumonia
Reactive airway	6 months–adolescence	Irritative dry, repetitive (not paroxysmal); night especially	Sometimes	Atopic, precipitants, seasonal; ± wheezing; response to β-agonist
Congenital vascular rings, pulmonary sling	Infancy	Brassy	No	Stridor; onset of symptoms in first month of life
Compression on airway or glossopharyngeal or phrenic nerve	Any	Irritative, dry	Sometimes initially	Can be positional (tumors, other masses), associated with other neuropathies, stridor, changes in phonation
PSYCHOGENIC	Adolescence	Vibratory, low-pitched, honking	Yes	Family dynamics and other somatization

[a]Viral upper respiratory tract infection (URI) complex consists of fever, rhinorrhea, sore throat, conjunctivitis, exanthem, enanthem.

Infectious Causes

There is considerable overlap in symptomatology of cough caused by certain infectious agents, such as *Bordetella pertussis* or *Mycoplasma pneumoniae* in adolescents,[52] because of a common tracheobronchial site of pathophysiology and frequent dual infection by microbes and viruses.[53] *Chlamydophila pneumoniae* also can cause similar symptoms.[54] Although both *B. pertussis* and *Chlamydia trachomatis* infections are associated with prominent cough in the absence of fever in young infants, there should be little difficulty in distinguishing the two entities. *B. pertussis* causes a dramatic, debilitating paroxysmal cough without airway or lower tract abnormalities (unless secondary pneumonia occurs, leading to fever and toxicity), whereas *C. trachomatis* causes pneumonia with prominent tachypnea: the cough is only important because it brings the child to medical attention (see Chapter 162, *Bordetella pertussis* [Pertussis] and Other Species; Chapter 167, *Chlamydia trachomatis*).

Diagnosis of pneumonia is based on signs of lower respiratory tract involvement, such as tachypnea and retractions, in addition to cough, and the likely causative agent is determined from the constellation of clinical findings (Tables 23-7 and 23-8). Protracted cough is the major symptom of recurrent or persistent pneumonia (see Chapter 37, Persistent and Recurrent Pneumonia). Cystic fibrosis can masquerade as pertussis, asthma, or bronchitis because of prominence of cough. Diagnosis of purulent pericarditis is frequently delayed, symptoms being misinterpreted as those of respiratory tract infection. Stretch of the pericardium, ischemic compression of the myocardium, or compression of pericardial mass on the airway can cause "cough" that is a hitch at the end of inspiration or, more frequently, a grunting expiratory sound.

Noninfectious Causes

Lack of context of an acute infection, prolonged duration of cough, presence of inciting factors, or specific physical findings suggest noninfectious causes. Infants with congenital anomalies of great vessels that compress and confine the trachea, esophagus, or both usually have respiratory symptoms (stridor, cough, or difficulty breathing during feeding) dated "from birth." Secondary pneumonia or aspiration can complicate the disorder or confuse the diagnosis. Coughing as a manifestation of milk allergy in infants is not well established. Increased postprandial coughing has been documented when thickened feedings are used as therapy for suspected or proven gastroesophageal reflux; the mechanism is ill defined.[55] Cough,

TABLE 23-7. Symptoms and Signs of Pneumonia

Symptoms	Signs	Physical Examinations
Fever	Fever	Rales
Cough	Cough	Wheezes
Rapid breathing	Tachypnea	Diminished breath sounds
Difficulty breathing	Dyspnea	Tubular breath sounds
Vomiting	Retractions	Dullness to percussion
Poor feeding	Nasal flaring	Decreased tactile and vocal fremitus
Irritability	Grunting	
Lethargy	Splinting	Meningismus
Chest pain	Cyanosis	Ileus
Abdominal pain		Pleural friction rub
Shoulder pain		

TABLE 23-8. Clinical Features of Pneumonia in Infants Younger Than 3 Months

	Respiratory Syncytial Virus	Other Respiratory Viruses	*Chlamydia*	Cytomegalovirus	Pertussis[a]
HISTORY					
Season	Winter	Unique to each	Any	Any	Any; peak July–October
Onset	Acute, days	Acute, days	Insidious	Insidious	Progressive, days
Illness in others	URI	URI, "flu," croup	No	No	Cough
Fever	Half of cases	Majority of cases	No	Unusual	No
Cough	Yes	Yes	Yes/staccato	Yes	Yes/paroxysmal
Associated features	Apnea, URI	URI, croup, conjunctivitis	Conjunctivitis (prior or current)	Failure to thrive, hepatosplenomegaly	Apnea, cyanosis, posttussive vomiting
PHYSICAL EXAMINATION					
Predominant feature	Respiratory distress	Respiratory distress	Cough	Failure to thrive	Cough
General appearance	Ill, not toxic	Ill, not toxic	Well, tachypneic	Chronically ill	Well between paroxysms
Degree of illness: respiratory findings	Degree of illness = findings	Degree of illness = findings	Findings > degree of illness	Ill general appearance > respiratory illness	Ill only during cough
Auscultation	Wheezes, coarse crackles	Crackles, wheezes	Diffuse crackles	Crackles, ± wheezes	Clear
LABORATORY STUDIES					
Chest radiograph	Hyperaeration, sub-segmental atelectasis	Hyperaeration, ± peribronchial thickening, ± diffuse interstitial infiltrates	Hyperaeration, diffuse alveolar and interstitial infiltrates	Diffuse interstitial infiltrates	Normal or perihilar infiltrate
White blood cell count	Normal or lymphocytosis	Normal, lymphocytosis, neutropenia	Eosinophilia	Normal, eosinophilia, lymphocytosis, neutropenia	Lymphocytosis; eosinophilia unusual
Other findings	Hypoxemia		Increases in IgG, IgA, IgM	Increases in IgG, IgA, IgM; thrombocytopenia	
Diagnostic tests	Nasal wash EIA, DFA, culture	Nasal wash EIA, DFA, culture; throat culture	Conjunctival, NP DFA, EIA	Throat, bronchoscopy, lung biopsy, or urine culture	NP DFA, culture, PCR

DFA, direct fluorescent antibody (test); EIA, enzyme immunoassay; Ig, immunoglobulin; NP, nasopharyngeal specimen; PCR, polymerase chain reaction; URI, upper respiratory tract infection.
[a]Pertussis is included in this table because it should be considered in young infants with cough and respiratory distress, although pneumonia is characteristically absent.

stridor, or choking spells, without regurgitation, can be a manifestation of gastroesophageal reflux in infants.[34,56] Clues to this diagnosis are: (1) typical age of onset at 6 weeks to 6 months; (2) postprandial occurrence of cough; and (3) history of pneumonia. Diagnosis is best confirmed by esophageal pH study.

Cough can be the result of irritation of normal airways by mucus or purulent secretions (e.g., postnasal discharge or sinusitis) or of hyperreactive airways by secretions, infection, environmental stimuli, or smoke. Dry cough and frequent throat-clearing are clues to irritation of postnasal secretions. Sinusitis and cough-variant asthma are the most common causes of chronic cough in children, even for those younger than 2 years (with a normal chest radiograph).[57] Pertussis and tracheal anomalies are frequently missed diagnoses when protracted cough is incorrectly ascribed to sinusitis or cough-variant asthma. Sinusitis as a cause can usually be uncovered by noting its association with infectious prodrome, the occurrence of cough day and night, or associated symptomatology; radiographs or limited computed tomographic study frequently clarify a confusing situation (see Chapter 34, Sinusitis).

Cough of asthma is suspected when there is family or patient history of allergies and symptoms are recurrent, are not associated with acute illness, are exaggerated at nighttime, or are provoked by exercise, cold, smoke, specific allergens, or pollutants. A diagnostic trial of bronchodilator therapy can be used in young children if the clinical history is compelling and other diagnoses are excluded or highly unlikely. Respiratory function is tested in older children, with a diagnostic trial of bronchodilator therapy if the result is abnormal, or methacholine challenge if the result is normal.[58]

Additional considerations in children whose cough is not explained by acute infection or allergic process include compression of the trachea by tumor mass, lymph nodes, or enlarged vessels; irritation of the diaphragm by an abdominal disease process; and irritation of the pleura or phrenic nerve by tumor, inflammatory fluid, mass, or blood. Cough is usually irritative (dry, occurring at end of inspiration, diminished by voluntarily decreased inspiratory excursion). Neuropathy, myopathy, and bulbar involvement in infectious, metabolic, and immunologic disorders or malignancy (especially neuroblastoma and rhabdomyosarcoma) are considered when symptoms are otherwise unexplained. Cough is almost invariably associated with other signs, such as gurgling, weak cry, hoarse or quiet voice, and stridor.

Habit cough has a classic presentation, usually after an uncomplicated "starter" URI in an adolescent (commonly a girl). The cough is loud, rattling, resonant, and low-pitched, occasionally with canine or seal-like bark. It never awakens the patient from sleep. Others, but not the patient, are bothered by the cough. This diagnosis is not tenable in the presence of weight loss or systemic illness. Invasive diagnostic procedures and narcotic cough suppressants are inappropriate and ineffective, and further foster the family's misplaced focus. Reassurance, redirection of focus, and frequent visits to the primary care provider for examination and caring support are curative.

TACHYPNEA AND OTHER SIGNS OF LOWER RESPIRATORY TRACT DISORDERS

Tachypnea can be a voluntary or involuntary response to anxiety, fright, or pain; an abnormal breathing pattern related to central nervous system dysfunction; or the physiologic response to increased temperature or metabolic state. It is most usually the response to respiratory acidosis or hypoxemia of acute infection or the attempt to restore pH balance during metabolic acidosis (e.g., diabetes, salicylate poisoning, dehydration). Metabolic causes should not be forgotten, while the clinician pursues the much more likely primary pulmonary causes. Additionally, tachypnea can result from primary cardiac abnormalities (congestive heart failure, cyanotic congenital heart disease), pulmonary vascular abnormalities (cardiac shunts, capillary dilatation, hemorrhage, obstructed return to the heart, or infarction), impaired lymphatic flow (congenital lymphangiectasia, tumor) or pleural fluid collections (hemorrhagic, purulent, transudative, or lymphatic fluid or a misplaced infusion from a vascular catheter).

Tachypnea is thought to be the best clinical predictor of lower respiratory tract infection in children. Reference values for normal respiratory rates have been reconfirmed in healthy and febrile infants and young children.[59–62] Roughly, respiratory rates >60 breaths/minute in infants younger than 6 months, >50 breaths/minute in infants 6 to 11 months old, and >40 breaths/minute in children 12 to 59 months old have a sensitivity of 50% to 85% for diagnosis of lower respiratory tract infection with specificity of 70% to 97%. A useful cutoff respiratory rate for febrile children 5 years of age and older might be 30 breaths/minute. For infants younger than 24 months, the younger the patient, the less likely that pneumonia is present if tachypnea is absent. Performance of a chest radiograph in febrile infants without an apparent focus of infection to exclude pneumonia "missed" by physical examination has extremely low yield in the absence of tachypnea.[63,64] In one study, for infants younger than 2 months, respiratory rate of 60 breaths/minute, retractions, or nasal flaring had sensitivity for diagnosis of pneumonia of 91%.[62]

Other symptoms and signs associated with pneumonia, such as cough, are more sensitive but are nonspecific; nasal flaring, intercostal retractions, and cyanosis have less sensitivity (25%, 9%, and 9%, respectively) but high specificity (87%, 93%, and 94%, respectively).[61]

Grunting is an expiratory sound produced in the larynx when vocal cords are adducted to generate positive end-expiratory pressure (self-induced PEEP) and increased resting volume of the lung. Its causes are myriad but never trivial. Grunting can be a sign of surfactant deficiency in the neonate, or of pulmonary edema, foreign-body aspiration, severe pneumonia, mediastinal mass or severe mediastinal shift from any cause, pleuritic or musculoskeletal chest pain, or myopericarditis or other cardiac abnormalities at any age.[65] Care must be taken with sedation, positioning, or intubation of such patients; the sudden removal of the self-induced PEEP can cause hypoxemia and respiratory arrest.

Adventitial respiratory sounds usually indicate lower respiratory tract disease, pulmonary edema, or hemorrhage. *Wheezes* are musical continuous sounds present predominantly on expiration and are a sign of airway obstruction. Widespread bronchiolar narrowing, as most commonly occurs with the inflammation of virus-associated lower respiratory tract infection, produces heterophonous high-pitched, sibilant wheezes of variable pitch and presence in different lung fields. Fixed obstruction in a larger airway, as from foreign body or anomaly, produces homophonous, monotonous wheeze. *Rhonchi*, sometimes also termed low-pitched wheezes, or coarse crackles, are nonrepetitive, nonmusical, low-pitched sounds frequently present on early inspiration and expiration; they are usually a sign of turbulent airflow through secretions in large airways. *Fine crackles* (the term preferred by pulmonologists for rales, which has a variety of meanings across languages)[66] are high-pitched, low-amplitude, end-inspiratory, discontinuous popping sounds indicative of the opening of peripheral air–fluid interfaces. Fine crackle is the auscultatory finding suggestive of the diagnosis of pneumonia. Auscultatory abnormalities of crackles and wheezing have disparate diagnostic usefulness among various studies, depending on the categorization of bronchiolitis. Tachypnea is a more sensitive finding than crackles for bacterial pneumonia; wheezing is more sensitive than tachypnea for bronchiolitis.

Diminished or distant breath sounds, dullness to percussion, and decreased vocal fremitus indicate peripheral pulmonary consolidation, pleural mass, or fluid collection. *Tubular breath sounds* (low-pitched sound of similar intensity throughout inspiration and expiration, as normally heard in the intrascapular area), dullness to percussion, and increased vocal fremitus indicate parenchymal consolidation, atelectasis, or the presence of another continuous tissue or fluid density abutting both a bronchus and the chest wall.

DIFFERENTIATING FEATURES OF PNEUMONIA

Table 23-7 shows symptoms and signs of pneumonia in infants and children. Although fever, cough, and tachypnea are cardinal features,

any or all of them can be overshadowed or overlooked in patients who come to medical attention for pneumonia-associated stiff neck, abdominal pain, or chest pain or for nonspecific symptoms of illness as well as in infants with feeding difficulty. Classic symptoms of pneumonia reported in adolescents and adults are fever, chills, pleuritic chest pain, and cough productive of purulent sputum, with less noticeable tachypnea.[67]

Pneumonia in Young Infants

In young infants, acute infection with bacterial and nonbacterial respiratory tract pathogens frequently leads to lower respiratory tract infection. Except in the first few days of life, when pneumonia is due predominantly to bacteria acquired from the mother's genital tract or to organisms acquired transplacentally, nonbacterial pathogens are overwhelmingly predominant.[68] As perinatally acquired agents persist, community exposures increase, and maternally derived antibody protection wanes, the infant between 3 weeks and 3 months old is vulnerable to a unique array of lower respiratory tract pathogens.[69] Clinical setting, specific symptom complex, and severity of illness in proportion to findings on physical examination aid distinction of likely causes and guide the diagnostic and therapeutic approach (see Table 23-8). Although the pathogens listed in Table 23-8 are frequently referred to as causing "afebrile pneumonia," this is a misnomer, because *Bordetella pertussis* infrequently causes lower respiratory tract abnormalities,[70,71] and respiratory syncytial virus and especially other respiratory viruses frequently cause fever.[11,68,72,73] A causal role for *Ureaplasma urealyticum* is not completely defined, because the situation is confounded by the asymptomatic presence of this organism in women and young infants. Pneumonia due to *Pneumocystis carinii* is probably confined to infants with severe debilitation or immune defects.

Pneumonia in Older Infants, Children, and Adolescents

A number of studies using complex diagnostic methodologies have confirmed the specific cause of pneumonia in 45% to 85% of cases.[74–78] Viral etiologies predominate, and, currently, most are amenable to diagnosis. Table 23-9 categorizes the features of acute pneumonia in older infants, children, and adolescents by etiology. No single fact in history or finding on examination is unique for any agent, but when they are taken together, a working diagnosis emerges and guides intervention or further diagnostic testing. Chest radiography and laboratory tests are reserved for patients who are ill or whose clinical picture is not compelling for a category of etiologic agents. The efficacy trial and postmarketing studies of heptavalent

TABLE 23-9. Clinical Features of Acute Pneumonia in Children and Adolescents

	Bacteria	Virus	Mycoplasma	Tuberculosis
HISTORY				
Age	Any; infants especially	Any	School age	Any; < 4 years and 15–19 years especially
Temperature (°C)	Most ≥ 39	Most < 39	Most < 39	Most < 39 (unless empyema)
Onset	Abrupt	Gradual	Worsening cough	Insidious cough
Others in home ill	No	Yes, concurrent; upper respiratory tract infection, rash, conjunctivitis	Yes, weeks apart; pharyngitis, "flu," cough	Yes, persistent cough
Associated signs, symptoms	Toxicity, rigors	Myalgia, rash, mucous membrane involvement	Headache, sore throat, chills, myalgia, rash, pharyngitis, myringitis	Weight loss, night sweats (late)
Cough	Wet, productive	Nonproductive	Hacking, paroxysmal, usually nonproductive	Irritative or productive
PHYSICAL EXAMINATION				
Predominant feature	Toxicity, respiratory distress	Respiratory distress	Cough	Persistent cough
Degree of illness: respiratory finding	Degree of illness > findings	Degree of illness ≥ findings	Degree of illness < findings	Well → no findings (± cough); ill → findings
Pleuritic chest pain	No/yes	No	No	No/occasional
Auscultation	Unilateral, anatomically confined or no crackles; dullness, diminished or tubular sounds	Diffuse, bilateral crackles, wheezes	Unilateral, anatomically confined crackles; ± wheezes	Most normal; or unilateral crackles ± dullness
LABORATORY STUDIES				
Chest radiograph	Hyperaeration, patchy alveolar infiltrate or consolidation in lobe, segment, subsegment	Hyperaeration, interstitial infiltrate in diffuse or perihilar distribution; "wandering" atelectasis	Patchy alveolar and/or interstitial infiltrate in single or contiguous, usually lower lobe(s), unilaterally; perihilar adenopathy	Patchy alveolar infiltrate in single or contiguous lobes with disproportionate hilar adenopathy; or miliary or lobar consolidation
Pleural fluid	No/yes → large	No/yes → small	No/yes → small	No/yes → small, large
Peripheral white blood cell count (cells per mm^3)	Majority > 15,000; neutrophils ± bands	Majority < 15,000; lymphocytes	Majority < 15,000; neutrophils	Majority < 15,000; neutrophils, monocytes
Sedimentation rate > 40 mm/hour	Usual	Infrequent	Infrequent	Frequent
Sputum	Copious, purulent; neutrophils, abundant bacteria	Scant mucoid; epithelial, Mononuclear cells	Scant mucoid; mixed mononuclear cells/neutrophils	Scant → copious; neutrophils (if copious)
Diagnostic tests	Sputum Gram stain, culture; blood culture	Nasal wash, throat, bronchoscopy specimen for antigen detection, culture; acute and convalescent serology	Cold agglutinin; acute and convalescent specific serology; throat culture, antigen detection, DNA techniques	Gastric aspirate; sputum stain and culture

conjugate pneumococcal vaccine infers *Streptococcus pneumoniae* as a relatively common cause of pneumonia with patchy or consolidative infiltrates.[79,80] Urine antigen detection test in children with lobar pneumonia also supports the important role of *S. pneumoniae*.[81] Currently, ascribing a causal role to *S. pneumoniae* is confounded by the findings of prolonged asymptomatic carriage and inconsistent serologic results among studies.[82]

HEMOPTYSIS

Hemoptysis, defined as coughing up of blood that originated below the larynx, is uncommon in children; most commonly, supposed episodes are due to a posteriorly draining nosebleed. Mechanisms of hemoptysis include bleeding from: (1) congenital or acquired abnormal bronchial or pulmonary blood flow, venous obstruction, or vascular abnormalities; (2) immune-mediated endothelial damage; or (3) infectious or traumatic erosion of tracheal, bronchial, or bronchiolar epithelium. Hemorrhage can be mild (tracheitis, tracheobronchitis) or massive (congenital malformations, foreign body, bronchiectasis, pulmonary hemosiderosis). Causes of hemoptysis in children are listed in Table 23-10. Infection is the most common cause of mild hemoptysis. Panton–Valentine leukocidin-producing *Staphylococcus aureus* pneumonia is specifically associated with hemoptysis.[83] Epstein–Barr virus was implicated in a single case.[84] For more severe hemoptysis, bronchiectasis associated with cystic fibrosis accounts for as many cases as all other causes combined.[85]

Rigid bronchoscopy, computed tomography, and magnetic resonance imaging are useful diagnostic modalities in most cases of hemoptysis. Digital subtraction angiography and, occasionally, cardiac catheterization or arteriography are required.

TABLE 23-10. Causes of Hemoptysis in Children

Epithelial Damage	Vascular Abnormality/Damage
Acute infection Bronchiectasis (cystic fibrosis, immunodeficiency, retained foreign body) Trauma (airway or chest) Foreign body Tumor (primary airway or pulmonary, metastatic)	Congenital heart disease or pulmonary vascular anomalies (venous obstruction, arteriovenous fistulae) Congenital malformation (pulmonary sequestration) Autoimmune vasculitis (systemic lupus erythematosus, Wegener granulomatosis, inflammatory bowel disease, Goodpasture syndrome) Sickle-cell disease Pulmonary hemosiderosis Nonspecific endothelial damage (chemical, drug)

CHAPTER 24

Abdominal Symptom Complexes

Robert S. McGregor

To simplify the clinical approach to abdominal symptom complexes, abdominal pain is usually classified as acute or recurrent abdominal pain (RAP). Acute abdominal pain demands rapid diagnosis and appropriate intervention so that catastrophic outcomes can be avoided.

ACUTE ABDOMINAL PAIN

Signs and symptoms of medical and surgical conditions that cause acute abdominal pain have considerable overlap. Even though Scholer and associates[1] determined that only 1.5% of 1141 nonscheduled healthcare visits for acute abdominal pain resulted in a surgical diagnosis, rapid diagnosis and intervention should always be a primary goal to avoid an adverse outcome. Cope,[2] in a classic monograph, pointed out that the first principle in approaching the patient with acute abdominal pain is the necessity of coming to a "best," albeit not "certain," diagnosis because severe abdominal pain of 6 hours' duration occurring in a previously well child is frequently caused by a condition of surgical importance.

History

The history and character of the patient's acute abdominal pain are elicited with specific consideration of anatomy, embryology, and physiology. Diaphragmatic irritation, for example, causes shoulder pain, because the diaphragm, a high thoracic structure embryologically, shares cervical nerve innervation with the shoulder. History of therapies already provided is elicited, and potential effects integrated. Anti-inflammatory agents, especially corticosteroids, can substantially alter expected clinical findings, and potent analgesics or pretreatment with antimicrobial agents can mask otherwise clarifying symptoms. Regimentation in history-taking is essential. The three features of pain of particular importance are location, migration, and radiation sites.

Location of Pain

Pain over the entire abdomen suggests a diffuse peritoneal process. Pain relative to disease in the small intestine is chiefly felt in the epigastric and umbilical areas, and because innervation of the appendix is similarly derived embryologically, the initial pain of acute appendicitis is located periumbilically. Pain relative to disease in the large intestine is usually felt in the hypogastrium or over the site of colonic abnormality. Pain of pelvic structures is also appreciated in the hypogastrium.

Migration of Pain and Radiation Sites

Migration of pain and sites of radiation are useful clues.[3] The early epigastric pain of appendiceal obstruction is carried by visceral pain fibers. Once the inflamed appendix irritates or adheres to the abdominal wall, somatic pain fibers in the parietal peritoneum cause migration of pain to the right lower quadrant. Similarly, biliary colic begins with epigastric pain but moves to the right upper quadrant when the inflamed gallbladder contacts parietal peritoneum. Because the eighth thoracic nerve innervates both the bile ducts and the infrascapular area of the posterior thorax, pain of biliary colic is often perceived just inferior to the right scapula. Renal and ureteral colic radiates to the ipsilateral testicle. Vertebral pain, as in osteomyelitis, radiates to the corresponding site of abdominal innervation.