Wheezing in infants: Working efficiently to the cause

JAMES D. TUTOR, M.D. and ROBERT A. SCHOUMACHER, M.D.
Tachypnea during sleep suggests significant disease

Wheezing in infants: Working efficiently to the cause

ABSTRACT: Among the most common causes of wheezing in young children are bronchiolitis, asthma, foreign-body aspiration, and various aspiration syndromes. When taking the history, ask about onset, frequency, and duration of wheezing; associated symptoms (for example, stridor); feeding pattern; and exposure to allergens or respiratory irritants. Focus the physical examination on assessing respiratory compromise, observing the chest configuration, auscultating the chest, and evaluating other relevant findings (for example, clubbing or eczema). The chest film, results of bronchodilator therapy, and possibly pulmonary function or reflux testing may provide additional clues to the differential diagnosis. (J Respir Dis 1996;17(11):977-997)

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Wheezing—a continuous musical or whistling sound produced by turbulent airflow through narrowed airways—occurs in as many as 30% of infants. Often caused by asthma or a viral infection, wheezing in infants may be a sign of a benign, self-limited process or a life-threatening condition. Appropriate management of the wheezing infant depends on systematic evaluation by a physician with a sound knowledge of the pathophysiologic mechanisms that cause wheezing.

In this issue of The Journal of Respiratory Diseases, we will review the diagnostic work-up of the wheezing infant, including how to focus the history and physical examination and how to select the most effective diagnostic tests to pinpoint the cause. We will also discuss the conditions that most commonly present with wheezing in very young children, including bronchiolitis, asthma, and aspiration syndromes. In a future issue of this journal, we will consider the differential diagnosis and management of less common causes of infant wheezing, such as bronchopulmonary dysplasia, immune deficiencies, cystic fibrosis (CF), and structural abnormalities.

MECHANISMS
Compared with airways in adults, those in infants are intrinsically narrow. Any decrease in lumen size exponentially increases the resistance to airflow.

Four general mechanisms produce airways narrowing in infants, resulting in wheezing:

• Accumulated secretions: The airways may become obstructed because of the accumulation of air-
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way secretions. Irritation that is chronic or recurrent may lead to increases in the size and number of airway mucous glands, causing a greater quantity of mucus to be produced.

The mucus may be abnormally viscous because of dehydration or inflammation, as in CF. The mechanisms for clearing mucus from the airways may also be faulty—for example, weak cough (as in muscular dystrophy) or abnormal action of the cilia (as in primary ciliary dyskinesia).

• **Interstitial edema:** When the tissues in the interstitium become edematous and compress the airways, peribronchial cuffing occurs. The interstitial edema may be caused by increased production of interstitial fluid or decreased pulmonary lymphatic drainage. This type of airway narrowing may be seen in some infants who have congestive heart failure.

• **Muscular contraction:** The airways may be narrowed by contraction of bronchial smooth muscle.

This mechanism is seen in infants who have such diseases as asthma or bronchopulmonary dysplasia.

• **Airway collapse:** Narrowing may result from airway collapse caused by malformed or absent cartilage in that segment, as in tracheomalacia. Such changes may be secondary to extrinsic compression by another structure, as in the case of the infant whose trachea is compressed by a vascular ring.

**INITIAL EVALUATION**

Judicious use of diagnostic tests, guided by the findings of a comprehensive history and physical examination, should enable you to evaluate the wheezing infant in systematic fashion.

**History**

The age at onset of wheezing, its duration, and any associated symptoms are among the points to cover in the infant’s history (Table 1). Frequent or persistent wheezing starting soon after birth, particularly if stridor is also present, suggests a congenital structural abnormality that involves the respiratory tract.

Ask about factors that may precipitate or relieve the infant’s wheezing. A history of sudden choking and wheezing may indicate that aspiration of a foreign body has occurred. Episodic wheezing that responds to bronchodilator therapy is common in asthma. Note any history of seasonal wheezing, recurrent sinopulmonary infections, or exposure to tuberculosis.

Investigate the infant’s birth and nursery course for factors that predispose to wheezing, such as meconium aspiration (a rare cause), prematurity, or bronchopulmonary dysplasia. The child’s feeding pattern and growth rate may provide clues to the cause of wheezing. Infants who choke when swallowing or often regurgitate their feedings may have swallowing dyskinesia or gastroesophageal reflux (GER) leading to chronic aspiration.

Elicit any history of chronic exposure to allergens or respiratory irritants, such as house dust or tobacco smoke. Be sure to assess the family medical history for the presence of atopy or chronic lung diseases (for example, CF or primary ciliary dyskinesia).

Finally, elicit any history of disease involving other organ systems that may have respiratory tract manifestations. An infant who has congenital heart disease with enlargement of the left atrium may be wheezing because of compression of the left main-stem bronchus.

**Physical examination**

Focus the first part of the physical examination on assessing the patient’s respiratory compromise and work of breathing (Table 2). Evaluating the respiratory rate during sleep is a noninvasive mea-

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**Table 1—Wheezeing in infants: Points to cover in the history**

<table>
<thead>
<tr>
<th>Point to Cover</th>
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<tbody>
<tr>
<td>Age at onset</td>
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<tr>
<td>Frequency</td>
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<tr>
<td>Duration</td>
</tr>
<tr>
<td>Associated symptoms (for example, stridor)</td>
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<tr>
<td>Precipitating and/or ameliorating factors (for example, seasonal wheezing or improvement in response to a bronchodilator)</td>
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<tr>
<td>Occurrence of a sudden episode of choking and wheezing (suggests foreign-body aspiration)</td>
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<tr>
<td>Recurrence of sinopulmonary infections</td>
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<td>Exposure to tuberculosis</td>
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<tr>
<td>Predisposing factors in the birth and nursery course (for example, meconium aspiration or prematurity)</td>
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<tr>
<td>Feeding pattern and growth rate (for example, choking while swallowing or regurgitation)</td>
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<tr>
<td>Exposure to allergens and/or respiratory irritants (for example, house dust or tobacco smoke)</td>
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<tr>
<td>Atopic conditions or chronic lung diseases in other family members (for example, cystic fibrosis or primary ciliary dyskinesia)</td>
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<tr>
<td>Other organ system diseases that may have respiratory tract manifestations (for example, congenital heart disease with enlargement of the left atrium)</td>
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</tbody>
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Table 2—Wheezing in infants: Steps in the physical examination

<table>
<thead>
<tr>
<th>Assessment of respiratory compromise and work of breathing</th>
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<tbody>
<tr>
<td>Respiratory rate during sleep (if possible)</td>
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<tr>
<td>Signs of distressed breathing: abnormal chest wall</td>
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<tr>
<td>retractions, bulging of the intercostal spaces, visible</td>
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<tr>
<td>use of accessory muscles of respiration and alae nasi,</td>
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<tr>
<td>paradoxical chest and abdominal wall movements, head</td>
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<tr>
<td>bobbing, and grunting</td>
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<tr>
<td>Clinical signs of hypoxia (for example, tachypnea,</td>
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<tr>
<td>tachycardia; cyanosis; hypertension; behavioral changes,</td>
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<tr>
<td>such as agitation or restlessness)</td>
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<tr>
<td>Clinical signs of hypercapnia (for example, flushed,</td>
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<tr>
<td>hot hands and feet; bounding pulses; drowsiness)</td>
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<tr>
<th>Assessment of the chest configuration</th>
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<tbody>
<tr>
<td>Trachea position</td>
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<tr>
<td>Asymmetric percussion or tactile fremitus (palpable</td>
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<tr>
<td>voice-generated vibrations)</td>
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<table>
<thead>
<tr>
<th>Auscultation of the lungs</th>
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<tr>
<td>Intensity, phase, and pitch of the breath sounds in each</td>
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<tr>
<td>lobe or segment compared with those heard in the homologous</td>
</tr>
<tr>
<td>contralateral lobe or segment</td>
</tr>
<tr>
<td>Presence and location of any adventitious lung sounds</td>
</tr>
<tr>
<td>Type of wheezing: high- or low-pitched, inspiratory or</td>
</tr>
<tr>
<td>expiratory, short or long, or single (monophonic) or</td>
</tr>
<tr>
<td>multiple (polyphonic)</td>
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<tr>
<th>Evaluation of other relevant findings</th>
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<tr>
<td>Clubbing</td>
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<tr>
<td>Eczema</td>
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<tr>
<td>Other signs of atopy</td>
</tr>
<tr>
<td>Tonsillar enlargement</td>
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<tr>
<td>Cardiac signs and symptoms</td>
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<tr>
<td>Lymphadenopathy</td>
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<tr>
<td>Hepatosplenomegaly</td>
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Sure of pulmonary and thoracic mechanics: the normal rate is 23 to 38 breaths per minute in children up to 1 year of age and 22 to 31 breaths per minute in children aged 1 to 2 years.

Consider training parents to count and record at home the respiratory rate of their sleeping infant over a full minute. Ask them to count the rate for two or three separate determinations and to record the average of the determinations. If a sleeping infant is tachypneic (that is, has a respiratory rate greater than 40 breaths per minute), significant respiratory tract disease is probably present.

Findings that reflect distressed breathing include abnormal substernal, intercostal, and suprasternal chest wall retractions; bulging of the intercostal spaces; visible use of the accessory muscles of respiration and the alae nasi; paradoxical chest and abdominal wall movements; head bobbing; and grunting. Clinical signs of hypoxia include tachypnea, tachycardia, cyanosis, hypertension (systolic blood pressure greater than 105 mm Hg, diastolic blood pressure greater than 70 mm Hg), and such behavioral changes as agitation or restlessness. Clinical signs of hypercapnia include flushed, hot hands and feet; bounding pulses; and drowsiness. Confirm suspected hypoxia or hypercapnia by pulse oximetry, blood gas measurement, or capnography.

Observe the infant’s chest configuration, and palpate the position of the trachea. Asymmetric percussion or tactile fremitus (palpable voice-generated vibrations) indicates a unilateral intrathoracic abnormality, such as a pneumothorax or pleural effusion.

Auscultate each lung segment, and compare the intensity, phase, and pitch of the breath sounds with those heard in the homologous contralateral segment. Note the presence and location of any adventitious lung sounds.

Wheezees are described as high- or low-pitched, inspiratory or expiratory, short or long, and single (monophonic) or multiple (polyphonic). Monophonic wheezing indicates obstruction in a single airway, as noted with foreign-body aspiration or bronchial compression by an adjacent structure. Polyphonic wheezing indicates obstruction of several airways, as in asthma or bronchiolitis.

Other relevant physical findings include:
- Clubbing
- Eczema or other signs of atopy
- Cardiac findings, such as an accentuated P₂, a narrowly split S₂, or a systolic heart murmur
- Lymphadenopathy
- Enlargement or displacement of the liver or spleen

Signs and symptoms of viral infection are relevant to bronchiolitis, which we discuss below.

Using the findings from the history, the physical examination, and a chest film, try to identify the site of the airway obstruction. Also determine whether the infant’s wheezing is responsive to bronchodilator therapy. If it is, provide aggressive treatment for possible asthma.

If you cannot determine the site of obstruction—or if response to a bronchodilator is equivocal and the history, physical examination,
and chest film do not suggest a specific diagnosis other than asthma—consider referral to a pediatric pulmonologist for other diagnostic studies, which may include pulmonary function tests. Infant pulmonary function testing is now available at many tertiary-care referral centers.

As in older patients, pulmonary function testing in infants can reveal the site and severity of the airway obstruction and the degree of responsiveness to bronchodilator therapy. This information can

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**Figure 1 – Evaluating and managing the wheezing infant**

1. Infant with wheezing
   - History, physical examination, chest film
     - Severe distress, hypoxemia, weight loss, fever, pneumonia
       - Yes: Consider hospitalization: supplemental O₂, parenteral fluids, and (if indicated) ribavirin or antibiotic therapy
       - No: Acute presentation, associated coryza, normal past medical history, normal growth and development
         - Yes: Bronchiolitis: manage on an outpatient basis
         - No: Chronic unremitting wheezing (4 or more weeks)
           - Yes: Consider any of the diagnoses in Table 3 except bronchiolitis; consult/refer as indicated
           - No: Wheezing responsive to a bronchodilator
             - Yes: Probable asthma: begin long-term management
             - No or poor response: Feeding difficulties, temporal relationship of wheezing and feeding, nocturnal symptoms
               - Yes: Evaluate for aspiration, GER; manage if confirmed
               - Unclear or no response: Consult/refer as indicated

GER, gastroesophageal reflux.
help guide the further evaluation and therapy of the wheezing baby (Figure 1).

**DIAGNOSTIC STUDIES**
The results of the history, physical examination, chest film and, possibly, pulmonary function testing enable you to choose from a limited number of sensitive diagnostic studies to further assess the cause of wheezing in an infant. They include barium esophagography, flexible fiberoptic bronchoscopy, sweat chloride testing, pH probe study, serum immunoglobulin measurement, tuberculin skin testing, testing for viral respiratory pathogens, and echocardiography.

The chest film may demonstrate hyperinflation of the lung fields secondary to air trapping. This can be seen in bronchiolitis, asthma (Figure 2), and CF, for example. The chest film is also helpful in assessing for pneumonia or other parenchymal lung disease, as well as congenital anomalies and foreign-body aspiration (Figure 3).

The barium esophagogram is useful in detecting extrinsic compression of the airway by a structure, such as a vascular ring or bronchogenic cyst. It is also useful for the detection of aspiration syndromes, such as swallowing dyskinesia, GER, laryngotracheal cleft, or tracheoesophageal fistula.

The pH probe study may be extremely helpful in evaluating GER. Serum immunoglobulin levels and the sweat chloride test may be needed to exclude immunodeficiency or CF in the wheezing infant who also has recurrent sinopulmonary infections. The echocardiogram is useful in evaluating the wheezing infant in whom congenital or acquired cardiac disease is suspected.

A tuberculin skin test is indicated if the child has been exposed to the first 2 years of life. It is seen most frequently in the winter and early spring months. More than 50% of cases are caused by infection with RSV, although the other common respiratory viruses can also cause bronchiolitis.

Bronchiolar obstruction in acute bronchiolitis is caused by edema and the accumulation of mucus and cellular debris due to invasion by viral particles. A marked increase in airflow resistance develops in the infant’s small airways, with air trapping. Atelectasis can occur when an obstruction becomes complete and the trapped air is absorbed. These pathologic processes impair normal gas exchange, and ventilation-perfusion mismatch results in hypoxemia.

The typical infant with bronchiolitis has cough and coryza for a few days followed by audible wheezing, poor feeding, and fever. On physical examination, the infant may be tachypneic and dyspneic and have other signs of respiratory compromise. On auscultation of the chest, widespread, fine, end-inspiratory crackles may be present. The expiratory phase is often prolonged, and wheezes are usually heard.

The chest film reveals hyperinflation of the lungs. Multifocal areas of atelectasis are seen in about 30% of cases.

Pulmonary function tests demonstrate obstruction of airflow in the small airways that is usually poorly reversible with bronchodilators. The presence of a virus can be demonstrated in nasopharyngeal secretions by antigen detection or culture.

Treatment of bronchiolitis is generally supportive and can usually be provided on an outpatient basis. However, consider hospitalization if the infant is:

- Hypoxic (that is, has an oxy-
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design saturation level less than 93% measured on room air by pulse oximetry or arterial oxygen tension less than 70 mm Hg measured on room air) and requires supplemental oxygen.

• Dehydrated and requires parenteral fluids.

The antiviral drug ribavirin appears to interfere with the expression of messenger RNA and to inhibit RSV protein synthesis. It is mixed with preservative-free, sterile water to a final concentration of 20 mg/mL and delivered by small-particle aerosol for 12 to 18 hours per day for 3 to 7 days, depending on the clinical course.

Consider using ribavirin in hospitalized infants at high risk for serious RSV disease: those with complicated congenital heart disease, bronchopulmonary dysplasia, CF, or immunodeficiency; those who are severely ill, with or without mechanical ventilation; and those who are at increased risk for a course that progresses from mild to more complicated (for example, infants younger than 6 weeks, premature infants, or infants with multiple congenital anomalies or certain neurologic or metabolic diseases).10 With or without ribavirin therapy, the infant who has recovered from the acute phase of RSV bronchiolitis may wheeze for several weeks.

Corticosteroids currently have no consistent role in the treatment of bronchiolitis. In large, controlled studies, they have failed to demonstrate any significant clinical effect.11

The role of bronchodilators in bronchiolitis is controversial. It is reasonable to try an inhaled bronchodilator in infants with this disease, but if you observe no beneficial response, discontinue the drug.12,13 Routine use of antibiotics has not been shown to influence the course of bronchiolitis.14,15

Asthma

A significant number of children with asthma have symptoms of wheezing and airway obstructive disease early in life (30% before 1 year and 50% to 55% before 2 years of age).16 Therefore, consider the diagnosis of asthma in infants with recurrent wheezing, particularly if it is bronchodilator-responsive, and symptom-free intervals between attacks.2

Airway obstruction is the major abnormality in asthma, resulting from a combination of bronchospasm, mucosal edema, and increased secretions. This obstruction is not uniform, so there is ventilation-perfusion mismatch, with the development of hypoxemia.17

An asthma exacerbation in a previously healthy infant or young child usually begins with coryza, rapidly followed by cough, tachypnea, and wheezing. Physical examination and chest film findings may be indistinguishable from those of bronchiolitis.

The infant who has atopy as well as asthma may have physical signs of eczema, peripheral blood eosinophilia (more than 700/µL), or a high serum immunoglobulin E

Figure 2 – Hyperexpanded lungs are evident in these chest films of a child with asthma. Note the small pneumomediastinum around the heart in the frontal view. In addition to hyperinflation, the lateral view shows air trapping; there is a large retrosternal air space in front of the heart. Asthma and bronchiolitis may be indistinguishable on infants’ chest films.
concentration. In the infant with asthma, the history or pulmonary function test results should demonstrate improved airflow in the small airways after treatment with a bronchodilator.

Generally, when an infant has frequent or severe exacerbations of asthma, prescribe regular use of a topical anti-inflammatory agent (such as cromolyn, nedocromil, or a corticosteroid) plus a β₂-adrenergic agonist (such as albuterol or terbutaline). Recommend avoidance of substances that precipitate the infant’s asthma, such as cigarette smoke, dust, and pollen.

Cromolyn is usually prescribed for infants as a nebulized solution, 20 mg every 6 to 8 hours. Nedocromil and topical corticosteroids are available in metered-dose inhalers. These agents are given to infants by means of a spacer device attached to an appropriate-size face mask. Albuterol is given as a nebulized solution, 0.05 to 0.15 mg/kg, every 4 to 6 hours or as needed, depending on the frequency and severity of the infant’s asthma exacerbations.

Foreign-body aspiration

This is one of the most common causes of accidental death in children in the United States. It occurs most often in toddlers but may occur in late infancy if small objects are within the infant’s grasp.

Suspect foreign-body aspiration in any infant with a history of sudden onset of choking, coughing, wheezing, or respiratory distress, particularly if the aspiration was witnessed. However, the episode may not have been noted by the infant’s caregiver. Maintain a high index of suspicion for the presence of a retained foreign body in the airway if an infant exhibits unexplained wheezing or pneumonia that resolves poorly with therapy.

On auscultation of the chest, the classic findings are unilateral decreased breath sounds as a result of decreased aeration of the lung, as well as unilateral wheezes resulting from partial occlusion of a bronchus. Up to 20% of chest films are normal in infants who have aspirated foreign bodies. The remaining 80% of chest films demonstrate obstructive emphysema or atelectasis.

If the history, physical examination, and chest film indicate the presence of a bronchial foreign body, promptly remove it using a rigid bronchoscope. If the evidence is equivocal, flexible fiberoptic bronchoscopy can be performed to search for a foreign body. If one is found, it can subsequently be removed with a rigid bronchoscope.

Aspiration syndromes

A variety of functional and anatomic disorders result in periodic aspiration of a foreign substance. They form three main categories:

• Esophageal disorders, especially GER.
• Swallowing disorders.
• Fistulas occurring between the larynx, trachea, or bronchus and the esophagus.

Each can be associated with recurrent wheezing and pneumonia in the infant.

• GER: Although many infants have minor degrees of GER, about 1:300 to 1:1,000 have significant GER and associated complications. After retrograde movement, gastric contents may remain in the esophagus for prolonged periods or enter the pharynx, where they may be regurgitated, reswallowed, or—if the airway protection mechanisms fail—aspirated.

GER may cause respiratory symptoms by three mechanisms: direct aspiration into the lungs; induction of laryngospasm and apnea; and stimulation of vagal nerve endings, possibly inducing reflex bronchoconstriction. Thus, infants with pathologic GER may have respiratory manifestations of varying severity, such as cough, dyspnea, acute respiratory failure with aspiration pneumonia, tracheobronchitis, laryngospasm with obstructive apnea, or chronic or recurrent wheezing. In some infants, wheezing may be the sole symptom of GER.

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and aspiration, chest film findings may vary from normal to diffuse severe involvement. The most common findings are hyperinflation of the lungs and bronchial wall thickening. In other infants, infiltrates involving the dependent areas of the lungs are found. Microaspiration of refluxed gastric contents frequently occurs and often does not produce changes on the chest film. Pulmonary function tests frequently demonstrate air flow obstruction in the small airways that is not reversed by administration of bronchodilators.

Consider testing for GER when clinical signs or symptoms suggest its presence; also consider testing when you suspect that GER is causing wheezing although no clinical signs or symptoms of GER are present. The most reliable study to detect GER is placement of a pH monitor in the lower esophagus. Continuous recording of pH is performed during a 24-hour period to include sleep and several feedings. The presence of reflux is defined as an esophageal pH less than 4. Therefore, pH monitoring detects only acid reflux; it does not detect alkaline or neutral reflux.

Another frequently used method to detect GER is the barium esophagogram. This is probably the easiest study for the primary care physician to obtain to document the presence of GER in a wheezing infant. The test outlines the esophageal anatomy and reveals massive reflux occurring during the 5- to 10-minute testing period. However, GER cannot be ruled out by a normal barium esophagogram, and the false-positive result rate is also significant. Documented GER in a wheezing infant warrants a trial of medical therapy.

Aspiration of refluxed gastric contents can also be detected with technetium scintigraphy. Radioactive technetium-labeled formula is fed to the infant, and the emitted radiation is recorded continuously over the following 4 hours to determine whether any of the isotope is aspirated into the lungs. The sensitivity of scintigraphy is limited by variability in gastric emptying, reflux, and aspiration.

Numerous lipid-laden macrophages in a bronchoalveolar lavage fluid sample obtained by bronchoscopy is another sign of gastric reflux and aspiration. In a study of children, Nussbaum and colleagues described the association of lipid-laden alveolar macrophages on the one hand and GER with aspiration on the other. Colombo and Hallberg described a semiquantitative lipid-laden macrophage index to determine the likelihood of recurrent food aspiration in children.

Treatment of GER consists of:

- A conservative approach using small, thickened feedings; upright positioning during and after feedings; and elevation of the head and chest during sleep, on the assumption that GER often spontaneously improves after infancy.
- Use of a prokinetic medication, such as cisapride (0.1 to 0.3 mg/kg orally three or four times a day) or metoclopramide (0.1 mg/kg up to four times a day orally, intramuscularly, or intravenously), and an acid-suppressing H₂ blocker, such as cimetidine (10 to 20 mg/kg/d in divided doses given orally, intramuscularly, or intravenously every 6 hours) or ranitidine (2 to 5 mg/kg/d in divided doses given orally every 8 to 12 hours or 2 mg/kg/d in divided doses given intravenously every 6 to 8 hours).
- Surgical correction by fundoplexy if more conservative approaches have failed and GER is causing repetitive injury to the respiratory tract.

Bronchodilators can be useful as adjunctive therapy in some infants with GER-related wheezing. However, the primary therapy is control of GER, either medically or surgically. Refer wheezing infants with presumed or documented GER to a pediatric pulmonologist if they do not respond to appropriate therapy.

- Swallowing disorders: Neuro muscular diseases that suppress the level of consciousness, decrease cough effectiveness, or directly interfere with the swallowing or gag mechanism may make the infant more likely to aspirate during swallowing. Infants with such conditions often choke or cough when swallowing, and wheezing, cyanosis, or apnea may also develop.

Swallowing disorders in infants are best evaluated with an esophagogram under fluoroscopy using contrast mixtures of differing textures. The airway of an infant with a significant swallowing disorder must be protected by the use of a tube for feeding. Continue protective measures until the child can be taught by a speech pathologist to swallow without aspiration or until the cause of the swallowing disorder can be corrected.

- Fistulas: Passages between the trachea and esophagus or between the larynx and esophagus (laryngotracheal cleft) can cause recurrent cough, recurrent pneumonia, recurrent wheezing, and choking with swallowing in infants. A tracheoesophageal fistula (TEF) occurs in 1 in 4,000 to 1 in 5,000 live births. Many are diagnosed shortly after the patient is born, since most TEFs are associated with some form of esophageal atresia.

However, about 3% of TEFs occur in patients without associated esophageal atresia (H-type TEF). This type of TEF can go unrecognized during the immediate neo-
tal period. Suspect it in any infant who has recurrent wheezing asso-
ciated with the other manifestations of recurrent aspiration.

The diagnosis of an H-type TEF is best made by contrast esophago-
gram and fluoroscopy, with the use of soluble contrast media. If the H-
type TEF cannot be demonstrated radiographically, endoscopy can
be helpful. Surgical repair of the
TEF is the definitive therapy.

Laryngotracheal clefts are de-
velopmental failures involving sepa-
ration of the larynx and upper tra-
chea from the esophagus for a vari-
able distance. Symptoms in
infants with this condition are sim-
ilar to those in infants with a TEF.
Some may have symptoms of strid-
dor or aphonia. Radiographic
demonstration of a laryngotra-
cheal cleft by contrast esophago-
gram and fluoroscopy may be diffi-
cult; usually, endoscopy is re-
quired. Surgical repair is the
definitive therapy.

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