Respiratory distress syndrome

Although a neonate with RDS may breathe normally at first, he usually develops rapid, shallow respirations within minutes or hours of birth, with intercostal, subcostal, or sternal retractions; nasal flaring; and audible expiratory grunting. This grunting is a natural compensatory mechanism designed to produce positive end-expiratory pressure (PEEP) and prevent further alveolar collapse. Severe disease is marked by apnea, bradycardia, and cyanosis (from hypoxemia, left-to-right shunting through the foramen ovale, or right-to□left intrapulmonary shunting through atelectatic regions of the lung). Other clinical features include pallor, frothy sputum, and low body temperature as a result of an immature nervous system and the absence of subcutaneous fat.

Sudden infant death syndrome

Although parents find some victims wedged in crib corners or with blankets wrapped around their heads, autopsies rule out suffocation as the cause of death. Autopsy shows a patent airway, so aspiration of vomitus isn't the cause of death. Typically, SIDS babies don't cry out and show no signs of having been disturbed in their sleep. However, their positions or tangled blankets may suggest movement just before death, perhaps due to terminal spasm. Depending on how long the infant has been dead, a SIDS baby may have a mottled complexion with extreme cyanosis of the lips and fingertips or pooling of blood in the legs and feet that may be mistaken for bruises. Pulse and respirations are absent, and the diaper is wet and full of stool.

Croup

The onset of croup usually follows an upper respiratory tract infection. Clinical features include inspiratory stridor, hoarse or muffled vocal sounds, varying degrees of laryngeal obstruction and respiratory distress, and a characteristic sharp, barking, seal-like cough. These symptoms may last only a few hours or persist for a day or two. As it progresses, croup causes inflammatory edema and, possibly, spasm, which can obstruct the upper airway and severely compromise ventilation. (See How croup affects the upper airway.) Each form of croup has additional characteristics: In laryngotracheobronchitis, the symptoms seem to worsen at night. Inflammation causes edema of the bronchi and bronchioles as well as increasingly difficult expiration that frightens the child. Other characteristic features include fever, diffusely decreased breath sounds, expiratory rhonchi, and scattered crackles. Laryngitis, which results from vocal cord edema, is usually mild and produces no respiratory distress except in infants. Early signs include a sore throat and cough, which, rarely, may progress to marked hoarseness, suprasternal and intercostal retractions, inspiratory stridor, dyspnea, P diminished breath sounds, restlessness and, in later stages, severe dyspnea and exhaustion. Acute spasmodic laryngitis affects a child between ages 1 and 3, particularly one with allergies and a family history of croup. It typically begins with mild to moderate hoarseness and nasal discharge, followed by the characteristic cough and noisy inspiration (that usually awaken the child at night), labored breathing with retractions, rapid pulse, and clammy skin. The child understandably becomes anxious, which may lead to increasing dyspnea and transient cyanosis. These severe symptoms diminish after several hours but reappear in a milder form on the next one or two nights.

Epiglottiditis

Sometimes preceded by an upper respiratory infection, epiglottiditis may rapidly progress to complete upper airway obstruction within 2 to 5 hours. Laryngeal obstruction results from inflammation and edema of the epiglottis. Accompanying symptoms include high fever, stridor, sore throat, dysphagia, irritability, restlessness, and drooling. To relieve severe respiratory distress, the child with epiglottiditis may hyperextend his neck, sit up, and lean forward with his mouth open, tongue protruding, and nostrils flaring as he tries to breathe. He may develop inspiratory retractions and rhonchi.

Acute respiratory distress syndrome

ARDS initially produces rapid, shallow breathing and dyspnea within hours to days of the initial injury (sometimes after the patient's condition appears to have stabilized). Hypoxemia develops, causing an increased drive for ventilation. Because of the effort required to expand the stiff lung, intercostal and suprasternal retractions result. Fluid accumulation produces crackles and rhonchi; worsening hypoxemia causes restlessness, apprehension, mental sluggishness, motor dysfunction, and tachycardia (possibly with transient increased arterial blood pressure). Severe ARDS causes overwhelming hypoxemia. If uncorrected, this results in hypotension, decreasing urine output, respiratory and metabolic acidosis, and eventually ventricular fibrillation or standstill. ELDER TIP The older patient may appear to do well following an initial episode of ARDS. Symptoms commonly appear 2 to 3 days later.

Acute respiratory failure in COPD

In patients who have COPD with ARF, increased ventilation-perfusion mismatch and reduced alveolar ventilation decrease PaO_2 (hypoxemia) and increase $PaCO_2$ (hypercapnia). This rise in carbon dioxide (CO_2) lowers the pH. The resulting hypoxemia and acidemia affect all body organs, especially the CNS and the respiratory and cardiovascular systems. Specific symptoms vary with the underlying cause of ARF but may include these systems: Respiratory—Rate may be increased, decreased, or normal depending on the cause; respirations may be shallow, deep, or alternate between the two; and air hunger may occur. Cyanosis may or may not be present, depending on the hemoglobin (Hb) level and arterial oxygenation. Auscultation of the chest may reveal crackles, rhonchi, wheezing, or diminished breath sounds. CNS—When hypoxemia and hypercapnia occur, the patient may show evidence of restlessness, confusion, loss of concentration, irritability, tremulousness, diminished tendon reflexes, and papilledema; he may slip into a coma. Cardiovascular—Tachycardia, with increased cardiac output and mildly elevated blood pressure secondary to adrenal release of catecholamine, occurs early in response to low PaO_2 . With myocardial hypoxia, arrhythmias may develop. Pulmonary hypertension, secondary to pulmonary capillary vasoconstriction, may cause increased pressures on the right side of the heart, elevated jugular veins, an enlarged liver, and peripheral edema. Stresses on the heart may precipitate cardiac failure.

Pulmonary edema

The early symptoms of pulmonary edema reflect interstitial fluid accumulation and diminished lung compliance: dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea, and coughing. Clinical features include tachycardia, tachypnea, dependent crackles, jugular vein distention, and a diastolic (S_3) gallop. With severe pulmonary edema, the alveoli and bronchioles may fill with fluid and intensify the early symptoms. Respiration becomes labored and rapid, with more diffuse crackles and coughing that produces frothy, bloody sputum. Tachycardia increases, and arrhythmias may

occur. Skin becomes cold, clammy, diaphoretic, and cyanotic. Blood pressure falls and the pulse becomes thready as cardiac output falls. Symptoms of severe heart failure with pulmonary edema may also include signs of hypoxemia, such as anxiety, restlessness, and changes in the patient's level of consciousness.

Cor pulmonale

As long as the heart can compensate for the increased pulmonary vascular resistance, clinical features reflect the underlying disorder and occur mostly in the respiratory system. They include chronic productive cough, exertional dyspnea, wheezing respirations, fatigue, and weakness. Progression of cor pulmonale is associated with dyspnea (even at rest) that worsens on exertion, tachypnea, orthopnea, edema, weakness, and right upper quadrant discomfort. Chest examination reveals findings characteristic of the underlying lung disease. Signs of cor pulmonale and right-sided heart failure include dependent edema; distended jugular veins; prominent parasternal or epigastric cardiac impulse; hepatojugular reflux; an enlarged, tender liver; ascites; and tachycardia. Decreased cardiac output may cause a weak pulse and hypotension. Chest examination yields various findings, depending on the underlying cause of cor pulmonale. In COPD, auscultation reveals wheezing, rhonchi, and diminished breath sounds. When the disease is secondary to upper airway obstruction or damage to central nervous system respiratory centers, chest findings may be normal, except for a right ventricular lift, gallop rhythm, and loud pulmonic component of S2. Tricuspid insufficiency produces a pansystolic murmur heard at the lower left sternal border; its intensity increases on inspiration, distinguishing it from a murmur due to mitral valve disease. A right ventricular early murmur that increases on inspiration can be heard at the left sternal border or over the epigastrium. A systolic pulmonic ejection click may also be heard. Alterations in the patient's level of consciousness may occur.

Legionnaires' disease

The multisystem clinical features of Legionnaires' disease follow a predictable sequence, although the onset of the disease may be gradual or sudden. After a 2- to 10-day incubation period, nonspecific, prodromal signs and symptoms appear, including diarrhea, anorexia, malaise, diffuse myalgias and generalized weakness, headache, and recurrent chills. An unremitting fever develops within 12 to 48 hours with a temperature that may reach 105° F (40.6° C). A cough then develops that's nonproductive initially but eventually may produce grayish, nonpurulent, and occasionally blood-streaked sputum. Other characteristic features include nausea, vomiting, disorientation, mental sluggishness, confusion, mild temporary amnesia, pleuritic chest pain, tachypnea, dyspnea, and fine crackles. Patients who develop pneumonia may also experience hypoxia. Other complications include hypotension, delirium, heart failure, arrhythmias, acute respiratory failure, renal failure, and shock (usually fatal)

Atelectasis

Clinical effects vary with the cause of collapse, the degree of hypoxemia, and any underlying disease but generally include some degree of dyspnea. Atelectasis of a small area of the lung may produce only minimal symptoms that subside without specific treatment. However, massive collapse can produce severe dyspnea, anxiety, cyanosis, diaphoresis, peripheral circulatory collapse, tachycardia, and substernal or intercostal retraction. Also, atelectasis may result in compensatory hyperinflation

of unaffected areas of the lung, mediastinal shift to the affected side, and elevation of the ipsilateral hemidiaphragm.

Respiratory acidosis

Acute respiratory acidosis produces CNS disturbances that reflect changes in the pH of cerebrospinal fluid rather than increased CO2 levels in cerebral circulation. Effects range from restlessness, confusion, and apprehension to somnolence, with a fine or flapping tremor (asterixis), or coma. The patient may complain of headaches as well as exhibit dyspnea and tachypnea with papilledema and depressed reflexes. Unless the patient is P receiving O2 , hypoxemia accompanies respiratory acidosis. This disorder may also cause cardiovascular abnormalities, such as tachycardia, hypertension, atrial and ventricular arrhythmias and, in severe acidosis, hypotension with vasodilation (bounding pulses and warm periphery).

Respiratory alkalosis

The cardinal sign of respiratory alkalosis is deep, rapid breathing, possibly exceeding 40 breaths/minute. This pattern of breathing is similar to Kussmaul's respirations that characterize diabetic acidosis. Such hyperventilation usually leads to CNS and neuromuscular disturbances, such as light-headedness or dizziness (due to below-normal CO2 levels that decrease cerebral blood flow), agitation, circumoral and peripheral paresthesias, carpopedal spasms, twitching (possibly progressing to tetany), and muscle weakness. Severe respiratory alkalosis may cause cardiac arrhythmias (that may fail to respond to conventional treatment), seizures, or both.

Pneumothorax

The cardinal features of pneumothorax are sudden, sharp, pleuritic pain (exacerbated by movement of the chest, breathing, and coughing); asymmetrical chest wall movement; and shortness of breath. Additional signs of tension pneumothorax are weak and rapid pulse, pallor, jugular vein distention, and anxiety. Tracheal deviations may be present with mediastinal shift. Tension pneumothorax produces the most severe respiratory symptoms; a spontaneous pneumothorax that releases only a small amount of air into the pleural space may cause no symptoms. In a nontension pneumothorax, the severity of symptoms is usually related to the size of the pneumothorax and the degree of preexisting respiratory disease.

Pneumonia

The main symptoms of pneumonia are coughing, sputum production, pleuritic chest pain, shaking chills, shortness of breath, rapid shallow breathing, and fever. Physical signs vary widely, ranging from diffuse, fine crackles to signs of localized or extensive consolidation and pleural effusion. There may also be associated symptoms of headache, sweating, loss of appetite, excess fatigue, and confusion (in older people). Complications include hypoxemia, respiratory failure, pleural effusion, empyema, lung abscess, and bacteremia, with spread of infection to other parts of the body, resulting in meningitis, endocarditis, and pericarditis.

Idiopathic bronchiolitis obliterans with organizing pneumonia

The presenting symptoms of BOOP are usually subacute, with a flulike syndrome of fever, persistent and nonproductive cough, dyspnea (especially with exertion), malaise, anorexia, and weight loss

lasting for several weeks to several months. Physical assessment findings may reveal dry crackles as the only abnormality. Less common symptoms include a productive cough, hemoptysis, chest pain, generalized aching, and night sweats.

Pulmonary embolism

Total occlusion of the main pulmonary artery is rapidly fatal; smaller or fragmented emboli produce symptoms that vary with the size, number, and location of the emboli. Usually, the first symptom of pulmonary embolism is dyspnea, which may be accompanied by anginal or pleuritic chest pain. Other clinical features include tachycardia, productive cough (sputum may be blood-tinged), low-grade fever, and pleural effusion. Less common signs include massive hemoptysis, chest splinting, leg edema and, with a large embolus, cyanosis, syncope, and distended jugular veins. In addition, pulmonary embolism may cause pleural friction rub and signs of circulatory collapse (weak, rapid pulse and hypotension) and hypoxia P (restlessness and anxiety).

Sarcoidosis

Initial symptoms of sarcoidosis include arthralgia (in the wrists, ankles, and elbows), fatigue, malaise, and weight loss. Other clinical features vary according to the extent and location of the fibrosis: Respiratory—breathlessness, cough (usually nonproductive), substernal pain; complications in advanced pulmonary disease include pulmonary hypertension and cor pulmonale Cutaneous—erythema nodosum, subcutaneous skin nodules with maculopapular eruptions, and extensive nasal mucosal lesions Ophthalmic—anterior uveitis (common), glaucoma, and blindness (rare) Lymphatic—bilateral hilar and right paratracheal lymphadenopathy and splenomegaly P Musculoskeletal—muscle weakness, polyarthralgia, pain, and punched-out lesions on phalanges Hepatic—granulomatous hepatitis, usually asymptomatic Genitourinary—hypercalciuria Cardiovascular—arrhythmias (premature beats, bundle-branch or complete heart block) and, rarely, cardiomyopathy Central nervous system—cranial or peripheral nerve palsies, basilar meningitis, seizures, and pituitary and hypothalamic lesions producing diabetes insipidus.

Severe acute respiratory syndrome

The incubation period for SARS is typically 3 to 5 days but may last as long as 14 days. Initial signs and symptoms include fever, shortness of breath and other minor respiratory symptoms, general discomfort, headache, rigors, chills, myalgia, sore throat, and dry cough. Some individuals may develop diarrhea or a rash. Later complications include respiratory failure, liver failure, heart failure, myelodysplastic syndromes, and death

Lung abscess

The clinical effects of lung abscess include a cough that may produce bloody, purulent, or foul-smelling sputum, pleuritic chest pain, dyspnea, excessive sweating, chills, fever, headache, malaise, diaphoresis, and weight loss. Chronic lung abscess may cause localized bronchiectasis. Failure of an abscess to improve with antibiotic treatment suggests possible underlying neoplasm or other causes of obstruction.

Hemothorax

The patient with hemothorax may experience chest pain, tachypnea, and mild to severe dyspnea, depending on the amount of blood in the pleural cavity and associated pathologic conditions. If respiratory failure results, the patient may appear anxious, restless, possibly stuporous, and

cyanotic; marked blood loss produces hypotension and shock. The affected side of the chest expands and stiffens, whereas the unaffected side rises and falls with the patient's breaths.

Pulmonary hypertension

Most patients complain of increasing dyspnea on exertion, weakness, syncope, and fatigability. Many also show signs of right-sided heart failure, including peripheral edema, ascites, jugular vein distention, and hepatomegaly. Other clinical effects vary with the underlying disorder.

Pleural effusion and empyema

Patients with pleural effusion characteristically display symptoms relating to the underlying pathologic condition. Most patients with large effusions, particularly those with underlying pulmonary disease, complain of dyspnea. Those with effusions associated with pleurisy complain of pleuritic chest pain. Other clinical features depend on the cause of the effusion. Patients with empyema also develop fever and malaise.

Pleurisy

Sharp, stabbing pain that increases with deep breathing may be so severe that it limits movement on the affected side. Dyspnea also occurs. Other symptoms vary according to the underlying pathologic process.

Chronic obstructive pulmonary disease

The typical patient, a long-term cigarette smoker, has no symptoms until middle age. His ability to exercise or do strenuous work gradually starts to decline, and he begins to develop a productive cough. These signs are subtle at first but become more pronounced as the patient gets older and the disease progresses. Eventually the patient may develop dyspnea on minimal exertion, frequent respiratory infections, intermittent or continuous hypoxemia, and grossly abnormal pulmonary function studies. Advanced COPD may cause severe dyspnea, overwhelming disability, cor pulmonale, severe respiratory failure, and death.

Bronchiectasis

Initially, bronchiectasis may be asymptomatic. When symptoms do arise, they're commonly attributed to other illnesses. The patient usually P complains of frequent bouts of pneumonia or hemoptysis. The classic symptom, however, is a chronic cough that produces foul-smelling, mucopurulent secretions in amounts ranging from less than 10 ml/day to more than 150 ml/day. Cough and sputum production are observed in greater than 90% of bronchiectasis patients. Characteristic findings include coarse crackles during inspiration over involved lobes or segments, occasional wheezing, dyspnea, sinusitis, weight loss, anemia, malaise, clubbing, recurrent fever, chills, and other signs of infection.

Idiopathic pulmonary fibrosis

The usual presenting symptoms of IPF are dyspnea and a dry, hacking, and typically paroxysmal cough. Most patients have had these symptoms for several months to 2 years before seeking medical help. End-expiratory crackles, especially in the bases of the lungs, are usually heard early in the disease. Bronchial breath sounds appear later, when airway consolidation develops. Rapid, shallow breathing occurs, especially with exertion, and clubbing has been noted in more than 40% of patients. Late in the disease, cyanosis and evidence of pulmonary hypertension (augmented S2 and

S3 gallop) commonly occur. As the disease progresses, profound hypoxemia and severe, debilitating dyspnea are the hallmark signs.

Tuberculosis

After an incubation period of 4 to 8 weeks, TB is usually asymptomatic in primary infection but may produce nonspecific symptoms, such as fatigue, weakness, anorexia, weight loss, night sweats, and low-grade fever. ELDER TIP Fever and night sweats, the typical hallmarks of TB, may not be present in elderly patients, who instead may exhibit a change in activity or weight. Assess older patients carefully. In reactivation, symptoms may include a cough that produces mucopurulent sputum, occasional hemoptysis, and chest pains.

Silicosis

Initially, silicosis may be asymptomatic or may produce dyspnea on exertion, usually attributed to being “out of shape” or “slowing down.” If the disease progresses to the chronic and complicated stage, dyspnea on exertion worsens, and other symptoms—usually tachypnea and an insidious dry cough that's most pronounced in the morning—appear. Progression to the advanced stage causes dyspnea on minimal exertion, worsening cough, and pulmonary hypertension, which in turn leads to right-sided heart failure and cor pulmonale. Patients with silicosis have a high incidence of active TB, which should be considered when evaluating patients with this disease. Central nervous system changes—confusion, lethargy, and a decrease in the rate and depth of respiration as the partial pressure of arterial carbon dioxide increases—also occur in advanced silicosis. Other clinical features include malaise, disturbed sleep, and hoarseness. The severity of these symptoms may not correlate with chest X-ray findings or the results of pulmonary function tests.

Asbestosis

Clinical features may appear before chest X-ray changes. The first symptom is usually dyspnea on exertion, typically after 10 years' exposure. As fibrosis extends, dyspnea on exertion increases until, eventually, dyspnea occurs even at rest. Advanced disease also causes a dry cough (may be productive in smokers), chest pain (commonly pleuritic), recurrent respiratory infections, and tachypnea. Cardiovascular complications include pulmonary hypertension, right ventricular hypertrophy, and cor pulmonale. Finger clubbing commonly occurs.

Coal worker's pneumoconiosis

Simple CWP produces no symptoms, especially in nonsmokers. Symptoms of complicated CWP include exertional dyspnea and a cough that occasionally produces inky-black sputum (when fibrotic changes undergo avascular necrosis and their centers cavitate). Other clinical features of CWP include increasing dyspnea and a cough that produces milky, gray, clear, or coal-flecked sputum. Recurrent bronchial and pulmonary infections produce yellow, green, or thick sputum. Complications include pulmonary hypertension, right ventricular hypertrophy and cor pulmonale, and pulmonary tuberculosis (TB). In cigarette smokers, chronic bronchitis and emphysema may also complicate the disease.