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Respiratory disorders

Introduction

The respiratory system distributes air to the alveoli, where gas exchange—the addition of oxygen (O_2) and the removal of carbon dioxide (CO_2) from pulmonary capillary blood—takes place. Certain specialized structures within this system play a vital role in preparing air for use by the body. The nose, for example, contains vestibular hairs that filter the air and an extensive vascular network that warms it. The nose also contains a layer of goblet cells and a moist mucosal surface; water vapor enters the airstream from this mucosal surface to saturate inspired air as it's warmed in the upper airways. Ciliated mucosa in the posterior portion of the nose and nasopharynx as well as major portions of the tracheobronchial tree propel particles deposited by impaction or gravity to the oropharynx, where the particles are swallowed.

External respiration

The external component of respiration— ventilation or breathing—delivers inspired air to the lower respiratory tract and alveoli. Contraction and relaxation of the respiratory muscles move air into and out of the lungs. Ventilation begins with the contraction of the inspiratory muscles: the diaphragm (the major muscle of respiration) descends, while external intercostal muscles move the rib cage upward and outward.

Air then enters the lungs in response to the pressure gradient between the atmosphere and the lungs. The lungs adhere to the chest wall and diaphragm because of the vacuum created within the pleural space. As the thorax expands, negative pressure is created in the intrapleural space, causing the lungs to also expand and draw in the warmed, humidified air.

The accessory muscles of inspiration, which include the scalene and sternocleidomastoid muscles, raise the clavicles, upper ribs, and sternum. The accessory muscles aren't used in normal inspiration but may be used in some pathologic conditions.

Normal expiration is passive; the inspiratory muscles cease to contract, the diaphragm rises, and the elastic recoil of the lungs causes the lungs to contract. These actions raise the pressure within the lungs above atmospheric pressure, moving air from the lungs to the atmosphere. Active expiration causes pleural pressure to become less negative. (See *What happens in ventilation*.)

An adult lung contains an estimated 300 million alveoli; each alveolus is supplied by many capillaries. To reach the capillary lumen, O_2 must cross the alveolocapillary membrane, which consists of an alveolar epithelial cell, a thin interstitial space, the capillary basement membrane, and the capillary endothelial cell membrane. The O_2 tension of air entering the respiratory tract is approximately 150 mm Hg. In the alveoli, inspired air mixes with CO_2 and water vapor, lowering the O_2 pressure to approximately 100 mm Hg. Because alveolar partial pressure of O_2 is higher than that present in mixed venous blood entering the pulmonary capillaries (approximately 40 mm Hg), O_2 diffuses across the alveolocapillary membrane into the blood.

O_2 and CO_2 transport and internal respiration

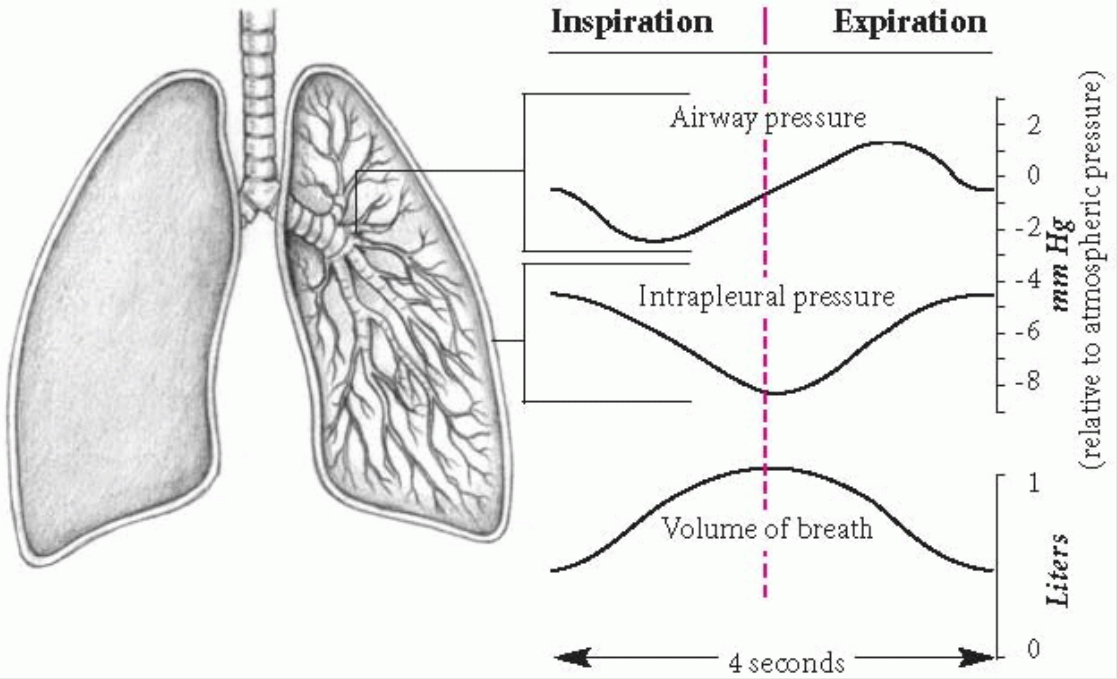
Circulating blood delivers O_2 to the cells of the body for metabolism and transports metabolic wastes and CO_2 from the tissues back to the lungs. When oxygenated arterial blood reaches tissue capillaries, O_2 diffuses from the blood into the cells because of the O_2 tension gradient. The amount of O_2 available is determined by the concentration of hemoglobin (Hb; the principal carrier of O_2), the percentage of O_2 saturation of the Hb, regional blood flow, arterial O_2 content, and cardiac output.

Internal (cellular) respiration occurs as a part of cellular metabolism, which can take place with O_2 (aerobic) or without O_2 (anaerobic). The most efficient method for providing fuel (high-energy compounds such as adenosine triphosphate [ATP]) for cellular reactions is aerobic metabolism, which produces CO_2 and water in addition to ATP. Anaerobic metabolism is

less efficient because a cell produces only a limited amount of ATP and yields lactic acid as well as CO_2 as a metabolic by-product.

WHAT HAPPENS IN VENTILATION

This illustration shows changes in the intrapleural and intrapulmonary (airway) pressures during inspiration and expiration.



Because circulation is continuous, CO_2 doesn't normally accumulate in tissues. CO_2 produced during cellular respiration diffuses from tissues into regional capillaries and is transported by systemic venous circulation. When CO_2 reaches the alveolar capillaries, it diffuses into the alveoli, where the partial pressure of CO_2 is lower; CO_2 is removed from the alveoli during exhalation.

Mechanisms of control

The central nervous system's (CNS's) control of respiration lies in the respiratory center, located in the lateral medulla oblongata of the brain stem. Impulses travel down the phrenic nerves to the diaphragm, and the intercostal nerves to the intercostal muscles, where the impulses change

the rate and depth of respiration. The inspiratory and expiratory centers, located in the posterior medulla, establish the involuntary rhythm of the breathing pattern.

Apneustic and pneumotaxic centers in the pons influence the pattern of breathing. Stimulation of the lower pontine apneustic center (by trauma, tumor, or stroke, for example) produces forceful inspiratory gasps alternating with weak expiration. The apneustic center continually excites the medullary inspiratory center and thus facilitates inspiration. Signals from the pneumotaxic center as well as afferent impulses from the vagus nerve inhibit the apneustic center and “turn off” inspiration. The apneustic pattern doesn't occur if vagi are intact.

Partial pressure of arterial oxygen (PaO_2), pH, and pH of cerebrospinal fluid (CSF) influence output from the respiratory center. When CO_2 enters the CSF, the pH of CSF falls, stimulating central chemoreceptors to increase ventilation.

The respiratory center also receives information from peripheral chemoreceptors in the carotid and aortic bodies. These chemoreceptors respond primarily to decreased PaO_2 but also to decreased pH. The peripheral chemoreceptors have little control

over respirations until the PaO_2 is less than 60 mm Hg.

During exercise, stretch receptors in lung tissue and the diaphragm prevent overdistention of the lungs. During swallowing, the cortex can interrupt automatic control of ventilation. During sleep, respiratory drive may fluctuate, producing hypoventilation and periods of apnea. External sensations, drugs, chronic hypercapnia, and changes in body temperature can also alter the respiratory pattern.

Diagnostic tests

Diagnostic tests evaluate physiologic characteristics and pathologic states within the respiratory tract.

Noninvasive tests include:

- Chest X-ray shows such conditions as atelectasis, pleural effusion, infiltrates, pneumothorax, lesions, mediastinal shifts, pulmonary edema, and chronic obstructive pulmonary disease (COPD).

- Computed tomography (or CT) scan provides a three-dimensional picture that's 100 times more sensitive than a chest X-ray.
- Magnetic resonance imaging (or MRI) identifies obstructed arteries and tissue perfusion, but movement of the heart and lungs reduces the image's clarity.
- Sputum specimen analysis assesses sputum quantity, color, viscosity, and odor; microbiological stains and culture of sputum can identify infectious organisms; and cytologic preparations can detect respiratory tract neoplasms. Sensitivity tests determine antibiotic sensitivity and resistance.
- Pulmonary function tests (or PFT) measure lung volume, flow rates, and compliance. Normal values, individualized by body stature and age, are reported in percentage of the normal predicted value. Static measurements are volume measurements that include tidal volume, volume of air contained in a normal breath; functional residual capacity, volume of air remaining in the lungs after normal expiration; vital capacity, volume of air that can be exhaled after maximal inspiration; residual volume, air remaining in the lungs after maximal expiration; and total lung capacity (TLC), volume of air in the lungs after maximal inspiration. Dynamic measurements characterize the movement of air into and out of the lungs and show changes in lung mechanics. They include measurement of forced expiratory volume in 1 second, maximum volume of air that can be expired in 1 second from total lung capacity; maximal voluntary ventilation, volume of air that can be expired in 1 minute with the patient's maximum voluntary effort; and forced vital capacity, maximal volume of air that the patient can exhale from TLC. (Peak flow rate, which can be obtained at the bedside, is also a dynamic measurement of pulmonary function.)
- Exercise stress test evaluates the ability to transport O_2 and remove CO_2 with increasing metabolic demands.
- Polysomnography can diagnose sleep disorders.
- Lung scan (ventilation-perfusion or scintiphotography scan) demonstrates ventilation and perfusion patterns. It's used primarily to evaluate pulmonary embolus.
- Arterial blood gas (ABG) analysis assesses gas exchange. Decreased PaO_2 may indicate hypoventilation, ventilation-perfusion mismatch, or shunting of blood away from gas exchange sites. Increased partial

pressure of arterial carbon dioxide ($PaCO_2$) reflects marked ventilation-perfusion mismatch or hypoventilation; decreased $PaCO_2$ reflects increased alveolar ventilation. Changes in pH may reflect metabolic or respiratory dysfunction.

- Pulse oximetry is a noninvasive assessment of arterial oxygen saturation.
- Capnography may be used either transcutaneously or in ventilator circuit to determine $PaCO_2$ trends.

Invasive tests include:

- Bronchoscopy permits direct visualization of the trachea and mainstem, lobar, segmental, and subsegmental bronchi. It may be used to localize the site of lung hemorrhage, visualize masses in these airways, and collect respiratory tract secretions. Brush biopsy may be used to obtain specimens from the lungs for microbiological stains, culture, and cytology. Lesion biopsies may be performed by using small forceps under direct visualization (when present in the proximal airways) or with the aid of fluoroscopy (when present distal to regions of direct visualization). Bronchoscopy can also be used to clear secretions and remove foreign bodies.
- Thoracentesis permits removal of pleural fluid for analysis.
- Pleural biopsy obtains pleural tissue for histologic examination and culture.
- Pulmonary artery angiography, the injection of dye into the pulmonary artery, can locate pulmonary embolism. This is considered the gold standard for diagnosing pulmonary emboli.
- Positron emission tomography (or PET) scan uses a short-life radionuclide. Increased uptake of the substance is seen in malignant cells.

Assessment

Assessment of the respiratory system begins with a thorough patient history. Ask the patient to describe his respiratory problem. How long has he had it? How long does each attack last? Does one attack differ from another? Does any activity in particular bring on an attack or make it worse? What relieves the symptoms? Always ask whether the patient was or

is a smoker, what and how often he smoked or smokes, and how long he smoked or has been smoking. Record this information in “pack years”—the number of packs of cigarettes per day multiplied by the number of smoking years. Remember to ask about the patient's occupation, hobbies, and travel; some of these activities may involve exposure to toxic or allergenic substances.

If the patient has dyspnea, ask if it occurs during activity or at rest. What position is the patient in when dyspnea occurs? How far can he walk? How many flights of stairs can he climb? Has his exercise tolerance been decreasing? Can he relate dyspnea to allergies or environmental conditions? Does it occur only at night, during sleep? If the patient has a cough, ask about its severity, persistence, and duration; ask if it produces sputum and, if so, how much and what kind. Have the patient's cough habits and character of sputum changed recently?

Physical examination

Use inspection skills to check for clues to respiratory disease, beginning with the patient's general appearance. If he's frail or cachectic, he may have a chronic disease that has impaired his appetite. If he's diaphoretic, restless, or irritable or protective of a painful body part, he may be in acute distress. Also, look for behavior changes that may indicate hypoxemia or hypercapnia. Confusion, lethargy, bizarre behavior, or quiet sleep from which he can't be aroused may point to hypercapnia. Watch for marked cyanosis, indicated by bluish or ashen skin (usually best seen on the lips, tongue, earlobes, and nail beds), which may be due to hypoxemia or poor tissue perfusion.

Assess chest shape and symmetry at rest and during ventilation. Increased anteroposterior diameter (“barrel chest”) characterizes emphysema. Kyphoscoliosis also alters chest configuration, which in turn restricts breathing. Assess respiratory excursion and observe for accessory muscle use during breathing. The use of upper chest and neck muscles is normal only during physical stress.

Observe the rate and pattern of breathing because certain disorders produce characteristic changes in breathing patterns. For example, an acute respiratory disorder can produce tachypnea (rapid, shallow breathing) or hyperpnea (increased rate and depth of breathing); intracranial lesions can produce Cheyne-Stokes and Biot's respirations; increased intracranial pressure can result in central hyperventilation and

apneustic or ataxic breathing; metabolic disorders can cause Kussmaul's respirations; and airway obstruction can lead to prolonged forceful expiration and pursed-lip breathing.

Also observe posture and carriage. A patient with COPD, for example, usually supports rib cage movement by placing his arms on the sides of a chair to increase expansion and leans forward during exhalation to help expel air.

Palpation of the chest wall detects areas of tenderness, masses, changes in fremitus (palpable vocal vibrations), or crepitus (air in subcutaneous tissues). To assess chest excursion and symmetry, place your hands in a horizontal position, bilaterally on the posterior chest, with your thumbs pressed lightly against the spine, creating folds in the skin. As the patient takes a deep breath,

your thumbs should move quickly and equally away from the spine. Repeat this with your hands placed anteriorly, at the costal margins (lower lobes) and clavicles (apices). Unequal movement indicates differences in expansion, seen in atelectasis, diaphragm or chest wall muscle disease, or splinting due to pain.

CHARACTERIZING AND INTERPRETING PERCUSSION SOUNDS

Percussion may produce several kinds of sounds. Known as flat, dull, resonant, hyperresonant, or tympanic, these sounds indicate the location and density of various structures. During percussion, determining other tonal characteristics, such as pitch, intensity, and quality, also will help identify respiratory structure. Use this chart as a guide to interpreting percussion sounds.

Characteristic

Sound	Pitch	Intensity	Quality	Implications
Flatness	High	Soft	Extremely dull	These sounds are normal over the sternum. Over the lung, they may indicate atelectasis or pleural effusion.

Dullness	Medium	Medium	Thudlike	Normal over the liver, heart, and diaphragm, these sounds over the lung may point to pneumonia, tumor, atelectasis, or pleural effusion.
Resonance	Low	Moderate to loud	Hollow	When percussed over the lung, these sounds are normal.
Hyperresonance	Lower than resonance	Very loud	Booming	These are normal findings with percussion over a child's lung. Over an adult's lung, these findings may indicate emphysema, chronic bronchitis, asthma, or pneumothorax.
Tympany	High	Loud	Musical, drumlike	Over the stomach, these are normal findings; over the lung, they suggest tension pneumothorax.

Percussion should detect resonance over lung fields that aren't covered by bony structures or the heart. A dull sound on percussion may mean consolidation or pleural disease. (See *Characterizing and interpreting percussion sounds*.)

Auscultation normally detects soft, vesicular breath sounds throughout most of the lung fields. Absent or adventitious breath sounds may indicate fluid in small airways or interstitial lung disease (crackles), secretions in moderate and large airways (rhonchi), and airflow obstruction (wheezes).

Special respiratory care

The hospitalized patient with respiratory disease may require an artificial upper airway, chest tubes, chest physiotherapy, and supervision of mechanical ventilation. In cardiopulmonary arrest, establishing an airway always takes precedence. In a patient with this condition, airway obstruction usually results when the tongue slides back and blocks the posterior pharynx. The head-tilt method or, in suspected or confirmed cervical fracture or arthritis, the jaw-thrust maneuver can immediately push the tongue forward, relieving such obstruction. Endotracheal (ET) intubation

and, sometimes, a tracheotomy may be necessary.

Chest tubes

An important procedure in patients with respiratory disease is chest tube drainage, which removes air or fluid from the pleural space. This allows the collapsed lung to reexpand to fill the evacuated pleural space. Chest drainage also allows removal of pleural fluid for culture. Chest tubes are commonly used after thoracic surgery, penetrating chest wounds, pleural effusion, and empyema. They're also used for evacuation of pneumothorax, hydrothorax, or hemothorax. Sometimes chest tubes are used to instill sclerosing drugs into the pleural space to prevent recurrent malignant pleural effusions.

Commonly, the chest tube is placed in the sixth or seventh intercostal space, in the axillary region. Occasionally, in pneumothorax, the tube is placed in the second or third intercostal space, in the midclavicular region.

Follow these guidelines when caring for a patient with a chest tube:

- Watch for air bubbling in the water-seal compartment of the drainage system. Bubbling should be intermittent; continuous bubbling may indicate an air leak.
- Monitor changes in suction pressure.
- Make sure that fluid in the drainage tube at the water-seal level fluctuates with breathing when the tube is patent.
- Make sure that all connections in the system are tight and secured with tape.
- Never clamp the chest tube unless checking for air leaks or changing the drainage system.
- Add water to the suction system when necessary.
- Record the amount, color, and consistency of drainage. Watch for signs of shock, such as tachycardia and hypotension, if drainage is excessive.
- Encourage the patient to cough and breathe deeply every hour to enhance lung expansion.

Ventilator methods

Mechanical ventilators are typically used for CNS problems, hypoxemia, or failure of the normal bellows action provided by the diaphragm and rib

cage. Volume-cycled ventilators deliver a preset volume of gas. The tidal volume is set at 10 to 15 ml/kg of ideal body weight. Lower volumes are now being advocated to reduce the risk of barotrauma. Positive end-expiratory pressure (PEEP) is used to retain a certain amount of pressure in the lungs at the end of expiration. By keeping small airways and alveoli open with this method, functional residual capacity is increased and oxygenation is improved.

When weaning is an option, several methods can be used to discontinue ventilation. The patient may be taken off the ventilator and supplied with a T-piece (ET tube O_2 adapter) that provides O_2 and humidification. The patient then breathes

spontaneously without the ventilator for gradually increasing periods.

With intermittent mandatory ventilation, the ventilator provides a specific number of breaths, and the patient is able to breathe spontaneously between ventilator breaths. The frequency of ventilator breaths is gradually decreased until the patient can breathe on his own. Recently, pressure support ventilation, in which the patient receives a preset pressure boost with each spontaneous breath, has proved effective. Vital signs, ABG levels, physical findings, and subjective symptoms should be monitored periodically during weaning to assess respiratory status.

Chest physiotherapy

In respiratory conditions marked by excessive accumulation of secretions in the lungs, chest physiotherapy may enhance removal of secretions. Chest physiotherapy includes chest assessment, effective breathing and coughing exercises, postural drainage, percussion, vibration, and evaluation of the therapy's effectiveness. Before initiating treatment, review X-rays and physical assessment findings to locate areas of secretions.

- Deep breathing maintains diaphragm use, increases negative intrathoracic pressure, and promotes venous return; it's especially important when pain or dressings restrict chest movement. An incentive spirometer can provide positive visual reinforcement to promote deep breathing.
- Pursed-lip breathing is used primarily in obstructive disease to slow expiration and prevent small airway collapse. Such breathing slows air

through smaller bronchi, maintaining positive pressure and preventing collapse of small airways and resultant air trapping.

- Segmental breathing or lateral costal breathing is used after lung resection and for localized disorders. Place your hand over the lung area on the affected side. Instruct the patient to try to push that portion of his chest against your hand on deep inspiration. You should be able to feel this with your hand.
- Coughing that's controlled and staged gradually increases intrathoracic pressure, reducing pain and bronchospasm of explosive coughing. When wound pain prevents effective coughing, splint the wound with a pillow, towel, or your hand during coughing exercises.
- Postural drainage uses gravity to drain secretions into larger airways, where they can be expectorated. This technique is used in the patient with copious or tenacious secretions. Before performing postural drainage, auscultate the patient's chest and review chest X-rays to determine the best position for maximum drainage. To prevent vomiting, schedule postural drainage at least 1 hour after meals.
- Percussion moves air against the chest wall, enhancing the effectiveness of postural drainage by loosening lung secretions. Percussion is contraindicated in severe pain, extreme obesity, cancer that has metastasized to the ribs, crushing chest injuries, bleeding disorders, spontaneous pneumothorax, spinal compression fractures, and in patients with temporary pacemakers.
- Vibration can be used with percussion or alone when percussion is contraindicated.
- PEEP therapy maintains positive pressure in airways, preventing small airway collapse.

Before and after chest physiotherapy, auscultate the patient's lung fields and assess for sputum production to evaluate the effectiveness of therapy.

CONGENITAL AND PEDIATRIC DISORDERS

Respiratory distress syndrome

Respiratory distress syndrome (RDS), also called *hyaline membrane disease*, is the most common cause of neonatal mortality. In the United States alone, it kills 40,000 neonates every year. RDS occurs in premature

neonates and, if untreated, is fatal within 72 hours of birth in up to 14% of neonates weighing less than 5½ lb (2.5 kg).

Aggressive management using mechanical ventilation can improve the prognosis, but some surviving neonates may develop some degree of bronchopulmonary dysplasia.

Causes and incidence

Although airways and alveoli of a neonate's respiratory system are present by 27 weeks' gestation, the intercostal muscles are weak and the alveolar capillary system is immature. The preterm neonate with RDS develops widespread alveolar collapse due to a lack of surfactant, a lipoprotein present in alveoli and respiratory bronchioles. Surfactant lowers surface tension and helps prevent alveolar collapse. This surfactant deficiency results in widespread atelectasis, which leads to inadequate alveolar ventilation with shunting of blood through collapsed areas of lung, causing hypoxemia and acidosis.

RDS occurs almost exclusively in neonates born before 37 weeks' gestation (in 60% of those born before the 28th week). The incidence is greatest in those with birth weights of 1,000 to 1,500 grams. Infants of diabetic mothers, those born by cesarean delivery, second-born twins, infants with perinatal asphyxia, and those delivered suddenly after antepartum hemorrhage are more commonly affected.

Complications

- Pneumothorax
- Pneumomediastinum
- Pneumopericardium
- Bronchopulmonary dysplasia (or BPD)
- Intraventricular bleed
- Hemorrhage into lungs after surfactant use
- Retinopathy of prematurity (or ROP)
- Delayed mental development or mental retardation

Signs and symptoms

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.

Diagnosis

CONFIRMING DIAGNOSIS

Signs of respiratory distress in a premature neonate during the first few hours of life strongly suggest RDS, but a chest X-ray and arterial blood gas (ABG) analysis are needed to confirm the diagnosis.

- Chest X-ray may be normal for the first 6 to 12 hours (in 50% of neonates with RDS), but 24 hours after birth it will show the characteristic ground-glass appearance and air bronchograms.
- ABG analysis shows decreased partial pressure of arterial oxygen; normal, decreased, or increased partial pressure of arterial carbon dioxide; and decreased pH (from respiratory or metabolic acidosis or both).
- Chest auscultation reveals normal or diminished air entry and crackles (rare in early stages).

When a cesarean birth is necessary before 36 weeks' gestation, amniocentesis enables the determination of the lecithin/sphingomyelin (L/S) ratio and the presence of phosphatidylglycerol. An L/S ratio of more than 2:1 and the presence of phosphatidylglycerol decrease the likelihood of RDS.

Treatment

Treatment of an infant with RDS requires vigorous respiratory support. Warm, humidified, oxygen-enriched gases are administered by oxygen hood or, if such treatment fails, by mechanical ventilation. Severe cases may require mechanical ventilation with PEEP or continuous positive airway pressure (CPAP), administered by nasal prongs or, when necessary, endotracheal (ET) intubation. Special ventilation

techniques are now used on the patients refractory to conventional mechanical ventilation. These include high-frequency jet ventilation and high-frequency oscillatory ventilation. Extracorporeal membrane oxygenation is the last choice for ventilation and is only available in certain specialized facilities. Treatment of RDS also includes:

- a radiant warmer or isolette for thermoregulation
- I.V. fluids and sodium bicarbonate to control acidosis and maintain fluid and electrolyte balance
- tube feedings or total parenteral nutrition if the neonate is too weak to eat
- administration of surfactant by an ET tube. (Studies show that this treatment can prevent or improve the course of RDS as well as reduce mortality.)

Special considerations

- Neonates with RDS require continual assessment and monitoring in an intensive care nursery.
- Closely monitor blood gases as well as fluid intake and output. If the neonate has an umbilical catheter (arterial or venous), check for arterial hypotension or abnormal central venous pressure. Watch for complications, such as infection, thrombosis, or decreased circulation to the legs. If the neonate has a transcutaneous oxygen monitor, change the site of the lead placement every 2 to 4 hours.
- To evaluate his progress, assess skin color, rate and depth of respirations, severity of retractions, nostril flaring, frequency of expiratory grunting, frothing at the lips, and restlessness.

- Regularly assess the effectiveness of oxygen or ventilator therapy. Evaluate every change in fraction of inspired oxygen and PEEP or CPAP by monitoring arterial oxygen saturation or ABG levels. Adjust the PEEP or CPAP as indicated, based on findings.
- Mechanical ventilation in neonates is usually done in a pressure-limited mode rather than the volume-limited mode used in adults.
- When the neonate is on mechanical ventilation, watch carefully for signs of barotrauma (an increase in respiratory distress and subcutaneous emphysema) and accidental disconnection from the ventilator. Check ventilator settings frequently. Be alert for signs of complications of PEEP or CPAP therapy, such as decreased cardiac output, pneumothorax, and pneumomediastinum. Mechanical ventilation increases the risk of infection in the preterm neonate, so preventive measures are essential.
- As needed, arrange for follow-up care with a neonatal ophthalmologist to check for retinal damage. Preterm neonates in an oxygen-rich environment are at increased risk for developing retinopathy of prematurity.
- Teach the parents about their neonate's condition and, if possible, let them participate in his care (using sterile technique), to encourage normal parent-infant bonding. Advise parents that full recovery may take up to 12 months. When the prognosis is poor, prepare the parents for the neonate's impending death and offer emotional support.
- Help reduce mortality in the neonate with RDS by detecting respiratory distress early. Recognize intercostal retractions and grunting, especially in a premature neonate, as signs of RDS; make sure the neonate receives immediate treatment.

PREVENTION

- *Prenatal care can help prevent prematurity.*
- *Give corticosteroids to the mother 2 to 3 days before delivery to help the infant's lungs mature in preterm deliveries.*

Sudden infant death syndrome

A medical mystery of early infancy, sudden infant death syndrome (SIDS), also called *crib death,* is the unexpected, sudden death of an infant or

child younger than age 1 year. Reasons for the death remain unexplained even after an autopsy. Typically, parents put the infant to bed and later find him dead, commonly with no indications of a struggle or distress of any kind. Incidence has decreased with the practice of teaching parents to place an infant on his back to sleep.

Causes and incidence

SIDS is the third leading cause of death in infants between age 1 month and 1 year. It occurs more commonly in winter months. The incidence is higher in males, preterm neonates, and those who sleep on their stomachs or in cribs with soft bedding. Incidence is also higher among neonates born in conditions of poverty and to those who were one of a single multiple birth, such as twins and triplets, and to mothers who smoke, take drugs, or failed to seek prenatal care until late in the pregnancy. SIDS may also result from an abnormality in the control of ventilation that allows carbon dioxide to build up in the blood, thereby causing prolonged apneic periods with profound hypoxemia and serious cardiac arrhythmias. It's also thought to be associated with problems in sleep arousal.

Signs and symptoms

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.

Diagnosis

Diagnosis of SIDS requires an autopsy to rule out other causes of death. Characteristic histologic findings on autopsy include small or normal adrenal glands and petechiae over the visceral surfaces of the pleura,

within the thymus, and in the epicardium. Autopsy also reveals extremely well-preserved lymphoid structures and certain pathologic characteristics that suggest chronic hypoxemia such as increased pulmonary artery smooth muscle. Examination also shows edematous, congestive lungs fully expanded in the pleural cavities, liquid (not clotted) blood in the heart, and curd from the stomach inside the trachea.

Treatment

If the parents bring the infant to the emergency department (ED), the physician will decide whether to try to resuscitate him. An “aborted SIDS” infant is one who's found apneic and is successfully resuscitated. Such an infant, or any infant who had a sibling stricken by SIDS, should be tested for infantile apnea. If tests are positive, a home apnea monitor may be recommended. Because the infant usually can't be resuscitated, however, treatment focuses on providing emotional support for the family.

Special considerations

- Make sure that parents are present when the child's death is announced. They may lash out at ED personnel, the babysitter, or anyone else involved in the child's care—even each other. Stay calm and let them express their feelings. Reassure them that they weren't to blame.
- Let the parents see the baby in a private room. Allow them to express their grief in their own way. Stay in the room with them if appropriate. Offer to call clergy, friends, or relatives.
- After the parents and family have recovered from their initial shock, explain the necessity for an autopsy to confirm the diagnosis of SIDS (in some states, this is mandatory). At this time, provide the family with some basic facts about SIDS and encourage them to give their consent for the autopsy. Make sure that they receive the autopsy report promptly.
- Find out whether your community has a local counseling and information program for SIDS parents. Participants in such a program will contact the parents, ensure that they receive the autopsy report promptly, put them in touch with a professional counselor, and maintain supportive telephone contact. Also, find out whether there's a local SIDS parent group; such a group can provide significant emotional support. Contact the National Sudden Infant Death Foundation for information about such local groups.

- If your facility's policy is to assign a public health nurse to the family, she will provide the continuing reassurance and assistance the parents will need.
- If the parents decide to have another child, they'll need information and counseling to help them through the pregnancy and the first year of the new infant's life.
- Infants at high risk for SIDS may be placed on apnea monitoring at home.
- All new parents should be informed of the American Academy of Pediatrics' recommendation that infants be positioned on their back, not on their stomach or side, for sleeping.

PREVENTION

- *Tell parents to place infants on their backs to sleep.*
- *Tell parents infants should sleep on a firm mattress and shouldn't have soft objects in the crib; like stuffed toys and blankets.*
- *Tell parents infants shouldn't sleep in the same bed as their parents.*
- *Tell parents to give infants pacifiers at bedtime.*
- *Tell parents infants shouldn't be exposed to secondhand smoke.*

Croup

Croup is a severe inflammation and obstruction of the upper airway, occurring as acute laryngotracheobronchitis (most common), laryngitis, and acute spasmodic laryngitis; it must always be distinguished from epiglottiditis. It's derived from an old German word for “voice box” and refers to swelling around the larynx or vocal cords. Recovery is usually complete.

Causes and incidence

Croup usually results from a viral infection but can also be caused by bacteria, allergies, and inhaled irritants. Parainfluenza viruses cause 75% of

such infections; adenoviruses, respiratory syncytial virus (RSV), influenza, and measles viruses account for the rest.

Croup is a childhood disease affecting more boys than girls (typically between ages 3 months and 5 years) that usually occurs during the winter. Up to 15% of patients have a strong family history of croup.

Complications

- Respiratory distress
- Respiratory arrest
- Epiglottis
- Bacterial tracheitis
- Atelectasis
- Dehydration

Signs and symptoms

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,

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.

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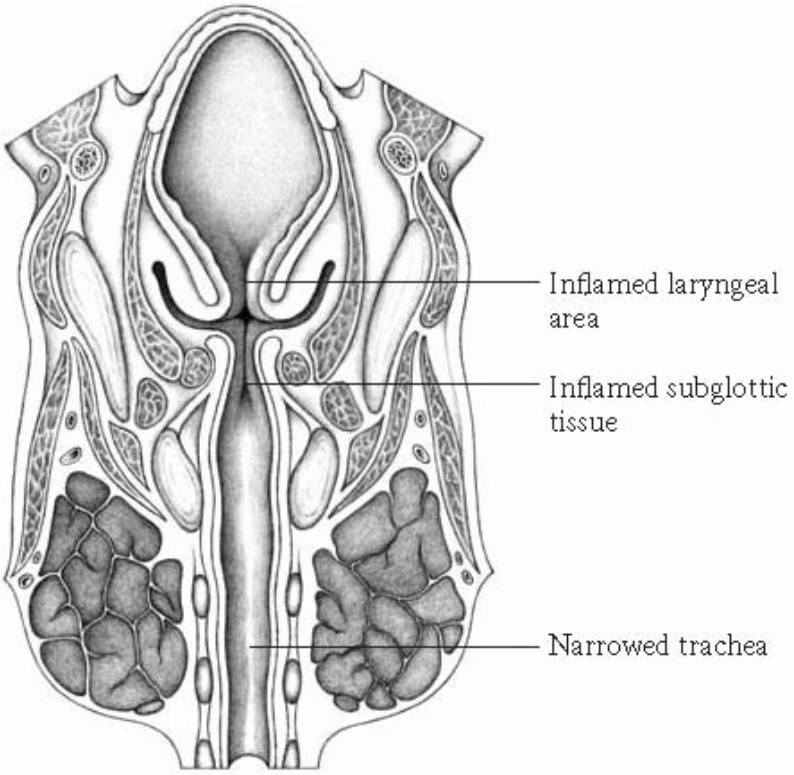
HOW CROUP AFFECTS THE UPPER AIRWAY

In croup, inflammatory swelling and spasms constrict the larynx, thereby reducing airflow. This cross-sectional drawing (from chin to chest) shows the upper airway changes caused by croup. Inflammatory changes almost completely obstruct the larynx (which includes the epiglottis) and significantly narrow the trachea.



Diagnosis

The clinical picture is very characteristic, so the diagnosis should be suspected immediately. When bacterial infection is the cause, throat cultures may identify the organisms and their sensitivity to antibiotics and rule out diphtheria. On a posterioranterior X-ray of the chest, narrowing of the upper airway (“steeple sign”) may be apparent. Laryngoscopy may reveal inflammation and obstruction in epiglottal and laryngeal areas. In evaluating the patient, assess for foreign body obstruction (a common cause of crouplike cough in a young child) as well as masses and cysts.

Treatment

For most children with croup, home care with rest, cool mist humidification during sleep, and antipyretics, such as acetaminophen, relieve symptoms. However, respiratory distress that's severe or interferes with oral hydration requires hospitalization and parenteral fluid replacement to prevent dehydration. If bacterial infection is the cause, antibiotic therapy is necessary. Oxygen therapy may also be required. Increasing obstruction of the airway requires intubation and mechanical ventilation.

Inhaled racemic epinephrine and corticosteroids may be used to alleviate respiratory distress.

Special considerations

Monitor and support respiration, and control fever. Because croup is so frightening to the child and his family, you must also provide support and reassurance.

- Carefully monitor cough and breath sounds, hoarseness, severity of retractions, inspiratory stridor, cyanosis, respiratory rate and character (especially prolonged and labored respirations), restlessness, fever, and cardiac rate.

- Keep the child as quiet as possible. However, avoid sedation because it may depress respiration. If the patient is an infant, position him in an infant seat or prop him up with a pillow; place an older child in Fowler's position. If an older child requires a cool mist tent to help him breathe, explain why it's needed.
- Isolate patients suspected of having RSV and parainfluenza infections if possible. Wash your hands carefully before leaving the room, to avoid transmission to other children, particularly infants. Instruct parents and others involved in the care of these children to take similar precautions.
- Control fever with sponge baths and antipyretics. Keep a hypothermia blanket on hand for temperatures above 102° F (38.9° C). Watch for seizures in infants and young children with high fevers. Give I.V. antibiotics as ordered.
- Relieve sore throat with soothing, water-based ices, such as fruit sherbet and popsicles. Avoid thicker, milk-based fluids if the child is producing heavy mucus or has great difficulty in swallowing. Apply petroleum jelly or another ointment around the nose and lips to soothe irritation from nasal discharge and mouth breathing.
- Maintain a calm, quiet environment and offer reassurance. Explain all procedures and answer any questions.

When croup doesn't require hospitalization:

- Teach the parents effective home care. Suggest the use of a cool humidifier (vaporizer). To relieve croupy spells, tell parents to carry the

child into the bathroom, shut the door, and turn on the hot water. Breathing in warm, moist air quickly eases an acute spell of croup.

- Warn parents that ear infections and pneumonia are complications of croup, which may appear about 5 days after recovery. Stress the importance of immediately reporting earache, productive cough, high fever, or increased shortness of breath.

PREVENTION

- *Wash hands frequently to prevent a respiratory infection.*
- *Give diphtheria, tetanus, and pertussis (DpT);* Haemophilus influenzae *B (Hib); and measles, mumps, and rubella (MMR) vaccines to children.*

Epiglottiditis

Acute epiglottiditis, also known as *epiglottitis,* is an acute inflammation of the epiglottis that tends to cause airway obstruction. A critical emergency, epiglottiditis can prove fatal unless it's recognized and treated promptly.

Causes and incidence

Epiglottiditis usually results from infection with *Haemophilus influenzae* type B (Hib) and, occasionally, pneumococci and group A streptococci. It typically strikes children between ages 2 and 6 years. (However, immunosuppression can predispose adults to epiglottiditis.) Since the advent of the Hib vaccine, epiglottiditis is becoming more rare.

Signs and symptoms

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.

Diagnosis

In acute epiglottiditis, throat examination reveals a large, edematous, bright red epiglottis. Such examination should follow lateral neck X-rays and, generally, *shouldn't* be performed if the suspected obstruction is great. Special equipment (laryngoscope and endotracheal [ET] tubes) should be available because a tongue blade can cause sudden complete airway obstruction. Trained personnel (such as an anesthesiologist) should be on hand during the throat examination to secure an emergency airway. On the lateral soft tissue X-ray of the neck, a large, thick but indistinct ("thumbprint") epiglottis will be seen.

Blood or throat culture may show *H. influenzae* or other bacteria.

Treatment

A child with acute epiglottiditis and airway obstruction requires emergency hospitalization; he may need emergency ET intubation or a tracheotomy with subsequent monitoring in an intensive care unit. Respiratory distress that interferes with swallowing necessitates parenteral fluid administration to prevent dehydration. A patient with acute epiglottiditis should always receive a complete course of parenteral antibiotics—usually a second- or third-generation cephalosporin. (If the child is allergic to penicillin, a quinolone or sulfa drug may be substituted.) Corticosteroids should be used to decrease swelling of the throat.

Special considerations

- Keep equipment available in case of sudden complete airway obstruction to secure an airway. Be prepared to assist with intubation or tracheotomy, as necessary.

ALERT

Watch for increasing restlessness, rising cardiac rate, fever, dyspnea, and retractions, which may indicate the need for an emergency tracheotomy. Monitor blood gases for hypoxemia and hypercapnia.

- After a tracheotomy, anticipate the patient's needs because he won't be able to cry or call out; provide emotional support. Reassure the patient

and his family that the tracheotomy is a short-term intervention (usually from 4 to 7 days). Monitor the patient for rising temperature and pulse rate and hypotension—signs of secondary infection.

- The bacterial infection causing epiglottiditis is contagious and airborne or droplet precautions should be followed. Family members should be screened.

PREVENTION

- *Wash hands frequently to prevent infections.*
- *Administer the vaccine to children.*

ACUTE DISORDERS

Acute respiratory distress syndrome

A form of noncardiogenic pulmonary edema that causes acute respiratory failure, acute respiratory distress syndrome (ARDS), also called *shock lung* or *adult respiratory distress syndrome*, results from increased permeability of the alveolocapillary membrane. Fluid accumulates in the lung interstitium, alveolar spaces, and small airways, causing the lung to stiffen. Effective ventilation is thus impaired, prohibiting adequate oxygenation of pulmonary capillary blood. Severe ARDS can cause intractable and fatal hypoxemia. However, patients who recover may have little or no permanent lung damage. (See *What happens in ARDS*, page 114.)

Causes and incidence

ARDS results from many respiratory and nonrespiratory insults, such as:

- aspiration of gastric contents
- sepsis (primarily gram-negative), trauma, or oxygen toxicity
- shock
- viral, bacterial, or fungal pneumonia or microemboli (fat or air emboli or disseminated intravascular coagulation)
- drug overdose (barbiturates, glutethimide, or opioids) or blood transfusion
- smoke or chemical inhalation (nitrous oxide, chlorine, or ammonia)

- hydrocarbon and paraquat ingestion
- pancreatitis, uremia, or miliary tuberculosis (rare)
- near drowning
- severe traumatic injuries, such as head injury or pulmonary contusions.

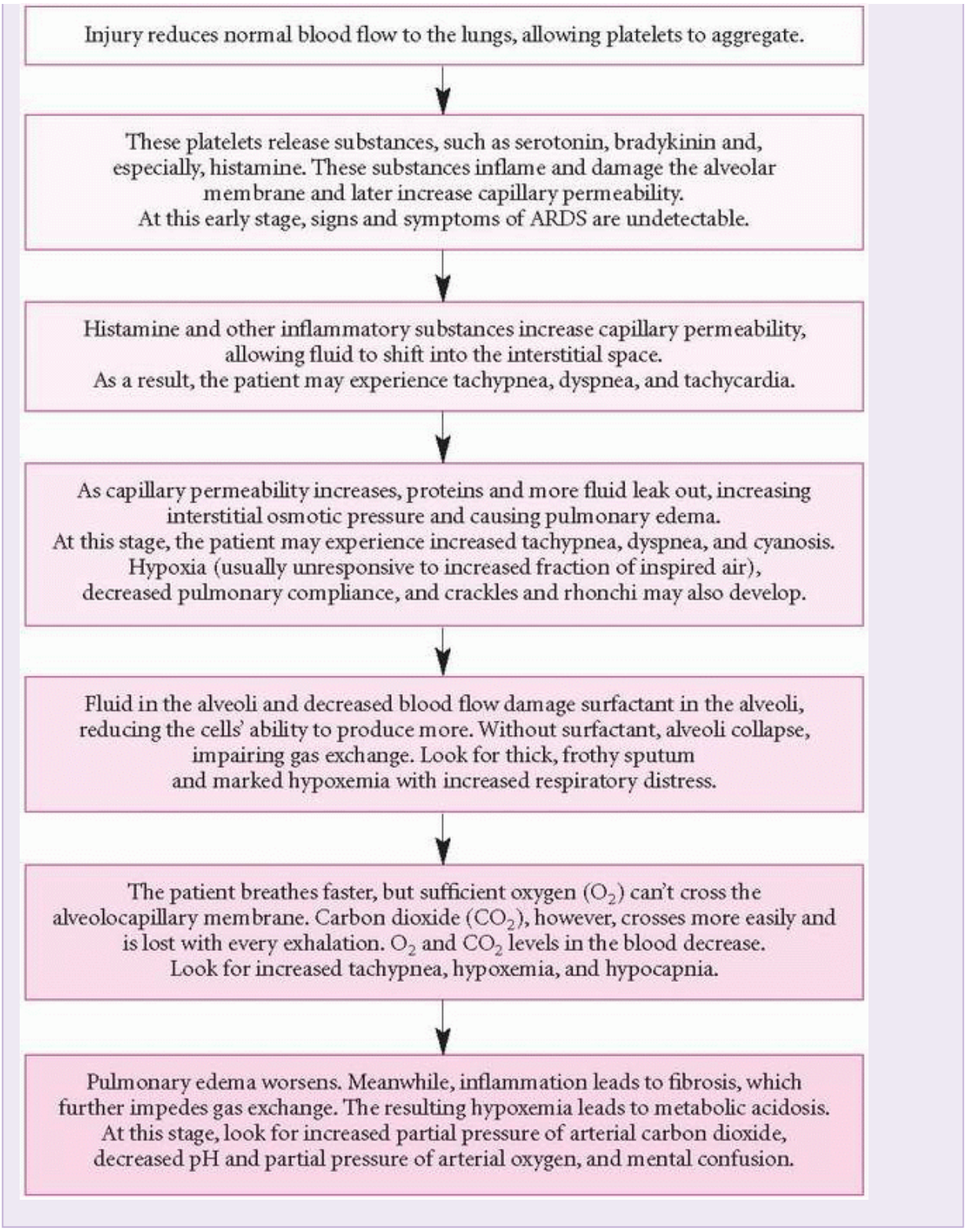
Altered permeability of the alveolocapillary membrane causes fluid to accumulate in the interstitial space. If the pulmonary lymphatic glands can't remove this fluid, interstitial edema develops. The fluid collects in the peribronchial and peribronchiolar spaces, producing bronchiolar narrowing. Hypoxemia occurs as a result of fluid accumulation in alveoli and subsequent alveolar collapse, causing the shunting of

blood through nonventilated lung regions. In addition, alveolar collapse causes a dramatic increase in lung compliance, which makes it more difficult to achieve adequate ventilation.

PATHOPHYSIOLOGY

WHAT HAPPENS IN ARDS

This flowchart shows the process and progress of acute respiratory distress syndrome (ARDS).



ARDS affects 10 to 14 people per 100,000, with a mortality rate of 36% to 52%.

Complications

- Multisystem failure
- Pulmonary fibrosis
- Pneumothorax

Signs and symptoms

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).

ELDER TIP

The older patient may appear to do well following an initial episode of ARDS. Symptoms commonly appear 2 to 3 days later.

Severe ARDS causes overwhelming hypoxemia. If uncorrected, this results in hypotension, decreasing urine output, respiratory and metabolic acidosis, and eventually ventricular fibrillation or standstill.

Diagnosis

On room air, arterial blood gas (ABG) analysis initially shows decreased partial pressure of arterial oxygen (PaO_2; less than 60 mm Hg) and partial pressure of arterial carbon dioxide ($PaCO_2$; less than 35 mm Hg). The resulting pH usually reflects respiratory alkalosis. As ARDS becomes more severe, ABG analysis shows respiratory acidosis (increasing $PaCO_2$ [more than 45 mm Hg]), metabolic acidosis (decreasing bicarbonate [less than 22 mEq/L]), and a decreasing PaO_2 despite oxygen therapy.

Other diagnostic tests include the following:

- Pulmonary artery catheterization helps identify the cause of pulmonary edema (cardiac versus noncardiac) by evaluating pulmonary artery wedge

pressure; allows collection of pulmonary artery blood, which shows decreased oxygen saturation, reflecting tissue hypoxia; measures pulmonary artery pressure; measures cardiac output by thermodilution techniques; and provides information to allow calculation of the percentage of blood shunted through the lungs.

- Serial chest X-rays initially show bilateral infiltrates. In later stages, a ground-glass appearance and eventually (as hypoxemia becomes irreversible), “whiteouts” of both lung fields are apparent. Medical personnel can differentiate ARDS from heart failure by noting the following on serial chest X-rays:

  – normal cardiac silhouette

  – diffuse bilateral infiltrates that tend to be more peripheral and patchy, as opposed to the usual perihilar “bat wing” appearance of cardiogenic pulmonary edema

  – fewer pleural effusions.

Differential diagnosis must rule out cardiogenic pulmonary edema, pulmonary vasculitis, and diffuse pulmonary hemorrhage. To establish the etiology, laboratory work should include sputum Gram stain, culture and sensitivity tests, and blood cultures to detect infections; a toxicology screen for drug ingestion; and, when pancreatitis is a consideration, a serum amylase determination.

Treatment

When possible, treatment is designed to correct the underlying cause of ARDS as well as to prevent progression and the potentially fatal complications of hypoxemia and respiratory acidosis. Supportive medical care consists of administering humidified oxygen with continuous positive airway pressure. Hypoxemia that doesn't respond adequately to these measures requires ventilatory support with intubation, volume ventilation, and positive end-expiratory pressure (PEEP). Other supportive measures include fluid restriction, diuretics, and correction of electrolyte and acid-base abnormalities.

When ARDS requires mechanical ventilation, sedatives, opioids, or neuromuscular blocking agents may be ordered to optimize ventilation. Treatment to reverse severe metabolic acidosis with sodium bicarbonate

may be necessary, although in severe cases this may worsen the acidosis if carbon dioxide can't be cleared adequately. Use of fluids and vasopressors may be required to maintain blood pressure. Infections require appropriate anti-infective therapy.

Special considerations

ARDS requires careful monitoring and supportive care.

- Frequently assess the patient's respiratory status. Be alert for retractions on inspiration. Note the rate, rhythm, and depth of respirations; watch for dyspnea and the use of accessory muscles of respiration. On auscultation, listen for adventitious or diminished breath sounds. Check for clear, frothy sputum, which may indicate pulmonary edema.
- Observe and document the hypoxemic patient's neurologic status (level of consciousness and mental status).
- Maintain a patent airway by suctioning, using sterile, nontraumatic technique. Ensure adequate humidification to help liquefy tenacious secretions.
- Closely monitor heart rate and blood pressure. Watch for arrhythmias that may result from hypoxemia, acid-base disturbances, or electrolyte imbalance. With pulmonary artery catheterization, know the desired pressure levels. Check readings often and watch for decreasing mixed venous oxygen saturation.
- Monitor serum electrolytes and correct imbalances. Measure intake and output; weigh the patient daily.
- Check ventilator settings frequently, and empty condensate from tubing promptly to ensure maximum oxygen delivery. Monitor ABG studies and pulse oximetry. The patient with severe hypoxemia may need controlled mechanical ventilation with positive pressure. Give sedatives, as needed, to reduce restlessness.
- Because PEEP may decrease cardiac output, check for hypotension, tachycardia, and decreased urine output. Suction only as needed to maintain PEEP or use an in-line suctioning apparatus. Reposition the patient often and record an increase in secretions, temperature, or hypotension that may indicate a deteriorating condition. Monitor peak pressures during ventilation. Because of stiff, noncompliant lungs, the patient is at high risk for barotrauma (pneumothorax), evidenced by

increased peak pressures, decreased breath sounds on one side, and restlessness.

- Monitor nutrition, maintain joint mobility, and prevent skin breakdown. Accurately record calorie intake. Give tube feedings and parenteral nutrition, as ordered. Perform passive range-of-motion exercises or help the patient perform active exercises, if possible. Provide meticulous skin care. Plan patient care to allow periods of uninterrupted sleep.
- Provide emotional support. Warn the patient who's recovering from ARDS that recovery will take some time and that he will feel weak for a while.
- Watch for and immediately report all respiratory changes in the patient with injuries that may adversely affect the lungs (especially during the 2- to 3-day period after the injury, when the patient may appear to be improving).

Acute respiratory failure in COPD

In patients with essentially normal lung tissue, acute respiratory failure (ARF) usually means partial pressure of arterial carbon dioxide ($PaCO_2$) above 50 mm Hg and partial pressure of arterial oxygen (PaO_2) below 50 mm Hg. These limits, however, don't apply to patients with chronic obstructive pulmonary disease (COPD), who usually have a consistently high $PaCO_2$ and low PaO_2. In patients with COPD, only acute deterioration in arterial blood gas (ABG) values, with corresponding clinical deterioration, indicates ARF.

Causes and incidence

ARF may develop in patients with COPD as a result of any condition that increases the work of breathing and decreases the respiratory drive. Such conditions include

respiratory tract infection (such as bronchitis or pneumonia). The most common precipitating factor is bronchospasm, or accumulating secretions secondary to cough suppression. Other causes of ARF in COPD include the following:

- Central nervous system (CNS) depression —head trauma or injudicious use of sedatives, opioids, tranquilizers, or oxygen (O_2)

- Cardiovascular disorders—myocardial infarction, heart failure, or pulmonary emboli
- Airway irritants—smoke or fumes
- Endocrine and metabolic disorders—myxedema or metabolic alkalosis
- Thoracic abnormalities—chest trauma, pneumothorax, or thoracic or abdominal surgery.

The incidence of ARF increases markedly with age and is especially high among people age 65 and older.

Signs and symptoms

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.

Diagnosis

Progressive deterioration in ABG levels and pH, when compared with the patient's "normal" values, strongly suggests ARF in COPD. (In patients with essentially normal lung tissue, pH below 7.35 usually indicates ARF, but patients with COPD display an even greater deviation from this normal value, as they do with $PaCO_2$ and PaO_2.)

Other supporting findings include:

- Bicarbonate—Increased levels indicate metabolic alkalosis or reflect metabolic compensation for chronic respiratory acidosis.
- Hematocrit (HCT) and Hb—Abnormally low levels may be due to blood loss, indicating decreased oxygen-carrying capacity. Elevated levels may occur with chronic hypoxemia.
- Serum electrolytes—Hypokalemia and hypochloremia may result from diuretic and corticosteroid therapies used to treat ARF.
- White blood cell count—Count is elevated if ARF is due to bacterial infection; Gram stain and sputum culture can identify pathogens.
- Chest X-ray—Findings identify pulmonary pathologic conditions, such as emphysema, atelectasis, lesions, pneumothorax, infiltrates, or effusions.
- Electrocardiogram—Arrhythmias commonly suggest cor pulmonale and myocardial hypoxia.

Treatment

ARF in patients with COPD is an emergency that requires cautious O_2 therapy (using nasal prongs or Venturi mask) to raise the PaO_2. In patients with chronic hypercapnia, O_2 therapy can cause hypoventilation by increasing $PaCO_2$ and decreasing the respiratory drive, necessitating mechanical

ventilation. The minimum fraction of inspired air (FIO_2) required to maintain ventilation or O_2 saturation greater than 85% to 90% should be used. If significant uncompensated respiratory acidosis or unrefractory hypoxemia exists, mechanical ventilation (through an endotracheal [ET] or a tracheostomy tube) or noninvasive ventilation (with a face or nose mask)

may be necessary. Treatment routinely includes antibiotics for infection, bronchodilators, and possibly steroids.

Special considerations

- Because most patients with ARF are treated in an intensive care unit, orient them to the environment, procedures, and routines to minimize their anxiety.
- To reverse hypoxemia, administer O_2 at appropriate concentrations to maintain PaO_2 at a minimum of 50 to 60 mm Hg. Patients with COPD usually require only small amounts of supplemental O_2. Watch for a positive response—such as improvement in the patient's breathing, color, and ABG levels.
- Maintain a patent airway. If the patient is retaining CO_2, encourage him to cough and to breathe deeply. Teach him to use pursed-lip and diaphragmatic breathing to control dyspnea. If the patient is alert, have him use an incentive spirometer; if he's intubated and lethargic, turn him every 1 to 2 hours. Use postural drainage and chest physiotherapy to help clear secretions.
- In an intubated patient, suction the trachea as needed after hyperoxygenation. Observe for a change in quantity, consistency, and color of sputum. Provide humidification to liquefy secretions.
- Observe the patient closely for respiratory arrest. Auscultate for chest sounds. Monitor ABG levels and report any changes immediately.
- Check the cardiac monitor for arrhythmias.

If the patient requires mechanical ventilation:

- Check ventilator settings, cuff pressures, and ABG values often because the FIO_2 setting depends on ABG levels. Draw specimens for ABG analysis 20 to 30 minutes after every FIO_2 change or oximetry check.
- Prevent infection by using sterile technique while suctioning.
- Stress ulcers are common in the intubated patient. Check gastric secretions for evidence of bleeding if the patient has a nasogastric tube or if he complains of epigastric tenderness, nausea, or vomiting. Monitor Hb level and HCT; check all stools for occult blood. Administer antacids, histamine-2 receptor antagonists, or sucralfate, as ordered.

PREVENTION

- *Prevent tracheal erosion, which can result from artificial airway cuff overinflation. Use the minimal leak technique and a cuffed tube with high residual volume (low-pressure cuff), a foam cuff, or a pressure-regulating valve on the cuff.*
- *To prevent oral or vocal cord trauma, make sure that the ET tube is positioned midline or moved carefully from side to side every 8 hours.*
- *To prevent nasal necrosis, keep the nasotracheal tube midline within the patient's nostrils and provide good hygiene. Loosen the tape periodically to prevent skin breakdown. Avoid excessive movement of any tubes; make sure the ventilator tubing is adequately supported.*

Pulmonary edema

Pulmonary edema is the accumulation of fluid in the extravascular spaces of the lung. In cardiogenic pulmonary edema, fluid accumulation results from elevations in pulmonary venous and capillary hydrostatic pressures. A common complication of cardiac disorders, pulmonary edema can occur as a chronic condition or it can develop quickly to cause death.

Causes and incidence

Pulmonary edema usually results from left-sided heart failure due to arteriosclerotic, hypertensive, cardiomyopathic, or valvular cardiac disease. In such disorders, the compromised left ventricle in unable to maintain adequate cardiac output; increased pressures are transmitted to the left atrium, pulmonary veins, and pulmonary capillary bed. This increased pulmonary capillary hydrostatic force promotes transudation of intravascular fluids into the pulmonary interstitium, decreasing lung compliance and interfering with gas exchange. Other factors that may predispose the patient to pulmonary edema include:

- excessive infusion of I.V. fluids

- decreased serum colloid osmotic pressure as a result of nephrosis, protein-losing enteropathy, extensive burns, hepatic disease, or nutritional deficiency
- impaired lung lymphatic drainage from Hodgkin's lymphoma or obliterative lymphangitis after radiation
- mitral stenosis, which impairs left atrial emptying
- pulmonary veno-occlusive disease
- lung damage from a severe infection or exposure to poisonous gas
- kidney failure.

Signs and symptoms

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.

Diagnosis

Clinical features of pulmonary edema permit a working diagnosis. Arterial blood gas (ABG) analysis usually shows hypoxia; the partial pressure of arterial carbon dioxide is variable. Profound respiratory alkalosis and acidosis may occur. Chest X-ray shows diffuse haziness of the lung fields and, commonly, cardiomegaly and pleural effusions. Ultrasound (echocardiogram) may show weak heart muscle, leaking or narrow heart valves, and fluid surrounding the heart. Pulmonary artery catheterization helps identify left-sided heart failure by showing elevated pulmonary

wedge pressures. This helps to rule out acute respiratory distress syndrome—in which pulmonary wedge pressure is usually normal.

Treatment

Treatment measures for pulmonary edema are designed to reduce extravascular fluid, improve gas exchange and myocardial function and, if possible, correct any underlying pathologic conditions.

Administration of high concentrations of oxygen by a cannula, a face mask and, if the patient fails to maintain an acceptable partial pressure of arterial oxygen level, assisted ventilation improves oxygen delivery to the tissues and usually improves acid-base disturbances. Diuretics—furosemide and bumetanide, for example—promote diuresis, which reduces extravascular fluid.

Treatment of heart failure includes angiotensin-converting enzyme inhibitors, diuretics, inotropic drugs such as digoxin, antiarrhythmic agents, beta-adrenergic blockers, and human B-type natriuretic peptide. Vasodilator drugs, such as nitroprusside, may be used to reduce preload and afterload in acute episodes of pulmonary edema.

Morphine is used to reduce anxiety and dyspnea as well as dilate the systemic venous bed, promoting blood flow from pulmonary circulation to the periphery.

Special considerations

- Carefully monitor the vulnerable patient for early signs of pulmonary edema, especially tachypnea, tachycardia, and abnormal breath sounds. Report any abnormalities. Assess for peripheral edema and weight gain, which may also indicate that fluid is accumulating in tissue.
- Administer oxygen as ordered.
- Monitor the patient's vital signs every 15 to 30 minutes while administering nitroprusside in dextrose 5% in water by I.V. drip. Protect the nitroprusside solution from light by wrapping the bottle or bag with aluminum foil, and discard unused

  solution after 4 hours. Watch for arrhythmias in the patient receiving cardiac glycosides and for marked respiratory depression in the patient receiving morphine.

- Assess the patient's condition frequently, and record response to treatment. Monitor ABG levels, oral and I.V. fluid intake, urine output and, in the patient with a pulmonary artery catheter, pulmonary enddiastolic and wedge pressures. Check the cardiac monitor often. Report changes immediately.
- Carefully record the time and amount of morphine given.
- Reassure the patient, who will have be anxious due to hypoxia and respiratory distress. Explain all procedures. Provide emotional support to his family as well.

Cor pulmonale

The World Health Organization defines chronic cor pulmonale as "hypertrophy of the right ventricle resulting from diseases affecting the function or the structure of the lungs, except when these pulmonary alterations are the result of diseases that primarily affect the left side of the heart or of congenital heart disease." Invariably, cor pulmonale follows some disorder of the lungs, pulmonary vessels, chest wall, or respiratory control center. For instance, chronic obstructive pulmonary disease (COPD) produces pulmonary hypertension, which leads to right ventricular hypertrophy and right-sided heart failure. Because cor pulmonale generally occurs late during the course of COPD and other irreversible diseases, the prognosis is generally poor.

Causes and incidence

Approximately 85% of patients with cor pulmonale have COPD, and 25% of patients with COPD eventually develop cor pulmonale.

Other respiratory disorders that produce cor pulmonale include:

- obstructive lung diseases—for example, bronchiectasis and cystic fibrosis
- restrictive lung diseases—for example, pneumoconiosis, interstitial pneumonitis, scleroderma, and sarcoidosis
- loss of lung tissue after extensive lung surgery
- congenital cardiac shunts—such as a ventricular septal defect
- pulmonary vascular diseases—for example, recurrent thromboembolism, primary pulmonary hypertension, schistosomiasis, and pulmonary vasculitis

- respiratory insufficiency without pulmonary disease—for example, in chest wall disorders such as kyphoscoliosis, neuromuscular incompetence due to muscular dystrophy and amyotrophic lateral sclerosis, polymyositis, and spinal cord lesions above C6
- obesity hypoventilation syndrome (pickwickian syndrome) and upper airway obstruction
- living at high altitudes (chronic mountain sickness).

Pulmonary capillary destruction and pulmonary vasoconstriction (usually secondary to hypoxia) reduce the area of the pulmonary vascular bed. Thus, pulmonary vascular resistance is increased, causing pulmonary hypertension. To compensate for the extra work needed to force blood through the lungs, the right ventricle dilates and hypertrophies. In response to low oxygen content, the bone marrow produces more red blood cells (RBCs), causing erythrocytosis. When the hematocrit (HCT) exceeds 55%, blood viscosity increases, which further aggravates pulmonary hypertension and increases the hemodynamic load on the right ventricle. Right-sided heart failure is the result.

Cor pulmonale accounts for about 25% of all types of heart failure. It's most common in areas of the world where the incidence of cigarette smoking and COPD is high; cor pulmonale affects middle-age to elderly men more often than women, but incidence in women is increasing. In children, cor pulmonale may be a complication of cystic fibrosis, hemosiderosis, upper airway obstruction, scleroderma, extensive bronchiectasis, neurologic diseases affecting respiratory muscles, or abnormalities of the respiratory control center.

Complications

- Right- and left-sided heart failure
- Hepatomegaly
- Edema
- Ascites
- Pleural effusions
- Thromboembolism

Signs and symptoms

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.

Diagnosis

- Pulmonary artery pressure measurements show increased right ventricular and pulmonary artery pressures, stemming from increased pulmonary vascular resistance. Right ventricular systolic and pulmonary artery systolic pressures will exceed 30 mm Hg. Pulmonary artery diastolic pressure will exceed 15 mm Hg.
- Echocardiography or angiography indicates right ventricular enlargement; echocardiography can estimate pulmonary artery pressure while also ruling out structural and congenital lesions.

- Chest X-ray shows large central pulmonary arteries and suggests right ventricular enlargement by rightward enlargement of the heart's silhouette on an anterior chest film.
- Arterial blood gas (ABG) analysis shows decreased partial pressure of arterial oxygen (PaO_2; typically less than 70 mm Hg and usually no more than 90 mm Hg on room air).
- Electrocardiogram frequently shows arrhythmias, such as premature atrial and ventricular contractions and atrial fibrillation during severe hypoxia; it may also show right bundle-branch block, right axis deviation, prominent P waves and inverted T wave in right precordial leads, and right ventricular hypertrophy.
- Pulmonary function tests show results consistent with the underlying pulmonary disease.
- HCT is typically greater than 50%.

Treatment

Treatment of cor pulmonale is designed to reduce hypoxemia, increase the patient's exercise tolerance and, when possible, correct the underlying condition.

In addition to bed rest, treatment may include administration of:

- a cardiac glycoside (digoxin)
- antibiotics when respiratory infection is present; culture and sensitivity of a sputum specimen helps select an antibiotic
- potent pulmonary artery vasodilators (such as diazoxide, nitroprusside, hydralazine, angiotensin-converting enzyme inhibitors, calcium channel blockers, or prostaglandins) in primary pulmonary hypertension
- oxygen by mask or cannula in concentrations ranging from 24% to 40%, depending on PaO_2, as necessary; in acute cases, therapy may also include mechanical ventilation; patients with underlying COPD generally shouldn't receive high

  concentrations of oxygen because of possible subsequent respiratory depression

- a low-sodium diet, restricted fluid intake, and diuretics, such as furosemide, to reduce edema
- phlebotomy to reduce the RBC count
- anticoagulants to reduce the risk of thromboembolism.

Depending on the underlying cause, some variations in treatment may be indicated. For example, a tracheotomy may be necessary if the patient has an upper airway obstruction. Steroids may be used in the patient with a vasculitis autoimmune phenomenon or acute exacerbations of COPD.

Special considerations

- Plan diet carefully with the patient and staff dietitian. Because the patient may lack energy and tire easily when eating, provide small, frequent feedings rather than three heavy meals.
- Prevent fluid retention by limiting the patient's fluid intake to 1 to 2 qt (1 to 2 L)/day and providing a low-sodium diet.
- Monitor serum potassium levels closely if the patient is receiving diuretics. Low serum potassium levels can increase the risk of arrhythmias associated with cardiac glycosides.
- Watch the patient for signs of digoxin toxicity, such as complaints of anorexia, nausea, vomiting, and halos around visual images and color perception shifts. Monitor for cardiac arrhythmias. Teach the patient to check his radial pulse before taking digoxin or any cardiac glycoside. He should be instructed to notify the physician if he detects changes in pulse rate.
- Reposition bedridden patients often to prevent atelectasis.
- Provide meticulous respiratory care, including oxygen therapy and, for the patient with COPD, pursed-lip breathing exercises. Periodically measure ABG levels and watch for signs of respiratory failure: changes in pulse rate, labored respirations, changes in mental status, and increased fatigue after exertion.

Before discharge, maintain the following protocol:

- Make sure that the patient understands the importance of maintaining a low-sodium diet, weighing himself daily, and watching for increased edema. Teach him to detect edema by pressing the skin over a shin with

one finger, holding it for a second or two, then checking for a finger impression. Increased weight, increased edema, or respiratory difficulty should be reported to the health care provider.

- Instruct the patient to plan for frequent rest periods and to do breathing exercises regularly.
- If the patient needs supplemental oxygen therapy at home, refer him to an agency that can help obtain the required equipment and, as necessary, arrange for follow-up examinations.
- If the patient has been placed on anticoagulant therapy, emphasize the need to watch for bleeding (epistaxis, hematuria, bruising) and to report signs to the physician. Also encourage him to return for periodic laboratory tests to monitor partial thromboplastin time, fibrinogen level, platelet count, HCT, hemoglobin level, and prothrombin time.
- Because pulmonary infection commonly exacerbates COPD and cor pulmonale, tell the patient to watch for and immediately report early signs of infection, such as increased sputum production, change in sputum color, increased coughing or wheezing, chest pain, fever, and tightness in the chest. Tell the patient to avoid crowds and persons known to have pulmonary infections, especially during the flu season. The patient should receive pneumovax and annual influenza vaccines.
- Warn the patient to avoid substances that may depress the ventilatory drive, such as sedatives and alcohol.

Legionnaires' disease

Legionnaires' disease is an acute bronchopneumonia produced by a gram-negative bacillus, *Legionella pneumophila*. It derives its name and notoriety from the peculiar, highly publicized disease that struck 182 people (29 of whom died) at an American Legion convention in Philadelphia in July 1976. This disease may occur epidemically or sporadically, usually in late summer or early fall. Its severity ranges from a mild illness,

with or without pneumonitis, to multilobar pneumonia, with a mortality as high as 15%. A milder, self-limiting form (Pontiac syndrome) subsides within a few days but leaves the patient fatigued for several weeks. This form mimics Legionnaires' disease but produces few or no respiratory symptoms, no pneumonia, and no fatalities.

Causes and incidence

Legionella pneumophila is an aerobic, gram-negative bacillus that's probably transmitted by an airborne route. In past epidemics, it has spread through cooling towers or evaporation condensers in airconditioning systems. However, *Legionella* bacilli also flourish in soil and excavation sites. The disease doesn't spread from person to person.

Legionnaires' disease is most likely to affect:

- middle-age and elderly people
- immunocompromised patients (particularly those receiving corticosteroids, for example, after a transplant) or those with lymphoma or other disorders associated with delayed hypersensitivity
- patients with a chronic underlying disease, such as diabetes, chronic renal failure, or chronic obstructive pulmonary disease
- those with alcoholism
- cigarette smokers
- those on a ventilator for extended periods.

Signs and symptoms

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).

Diagnosis

The patient history focuses on possible sources of infection and predisposing conditions. Additional tests reveal the following:

- Chest X-ray shows patchy, localized infiltration, which progresses to multilobar consolidation (usually involving the lower lobes), pleural effusion and, in fulminant disease, opacification of the entire lung.
- Auscultation reveals fine crackles, progressing to coarse crackles as the disease advances.
- Abnormal findings include leukocytosis, increased erythrocyte sedimentation rate, an increase in liver enzyme levels (alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase), hyponatremia, decreased partial pressure of arterial oxygen and, initially, decreased partial pressure of arterial carbon dioxide. Bronchial washings and blood, pleural fluid, and sputum tests rule out other infections.

CONFIRMING DIAGNOSIS

Definitive tests include direct immuno-fluorescence of respiratory tract secretions and tissue, culture of L. pneumophila*, and indirect fluorescent antibody testing of serum comparing acute samples with convalescent samples drawn at least 3 weeks later. A convalescent serum showing a fourfold or greater rise in antibody titer for* Legionella *confirms the diagnosis.*

Treatment

Antibiotic treatment begins as soon as Legionnaires' disease is suspected and diagnostic material is collected; it shouldn't await laboratory confirmation. Quinolone (ciprofloxacin, levofloxacin, moxifloxacin, or gatifloxacin) is commonly used, although a macrolide (azithromycin, clarithromycin, or erythromycin) may be prescribed for some patients. Supportive therapy includes administration of antipyretics, fluid replacement, circulatory support with pressor drugs, if necessary, and oxygen administration by mask, cannula, or mechanical ventilation.

Special considerations

- Closely monitor the patient's respiratory status. Evaluate chest wall expansion, depth and pattern of respirations, cough, and chest pain. Watch for restlessness as a sign of hypoxemia, which requires suctioning, repositioning, or more aggressive oxygen therapy.
- Continually monitor the patient's vital signs, oximetry or arterial blood gas values, level of consciousness, and dryness and color of lips and mucous membranes. Watch for signs of shock (decreased blood pressure, thready pulse, diaphoresis, and clammy skin).
- Keep the patient comfortable. Provide mouth care frequently. If necessary, apply soothing cream to the nostrils.
- Replace fluid and electrolytes, as needed. The patient with renal failure may require dialysis.
- Provide mechanical ventilation and other respiratory therapy, as needed. Teach the patient how to cough effectively, and encourage deep-breathing exercises. Stress the need to continue these until recovery is complete.
- Give antibiotic therapy as indicated, and observe carefully for adverse effects.

Atelectasis

Atelectasis is incomplete expansion of lobules (clusters of alveoli) or lung segments, which may result in partial or complete lung collapse. Because parts of the lung are unavailable for gas exchange, unoxygenated blood passes through these areas unchanged, resulting in hypoxemia. Atelectasis may be chronic or acute. Many patients undergoing upper abdominal or thoracic surgery experience atelectasis to some degree. The prognosis depends on prompt removal of any airway obstruction, relief of hypoxemia, and reexpansion of the collapsed lung.

Causes

Atelectasis commonly results from bronchial occlusion by mucus plugs. It's a problem in many patients with chronic obstructive pulmonary disease, bronchiectasis, or cystic fibrosis and in those who smoke heavily. (Smoking increases mucus production and damages cilia.) Atelectasis may also result

from occlusion by foreign bodies, bronchogenic carcinoma, and inflammatory lung disease.

Other causes include respiratory distress syndrome of the neonate (hyaline membrane disease), oxygen toxicity, and pulmonary edema, in which alveolar surfactant changes increase surface tension and permit complete alveolar deflation.

External compression, which inhibits full lung expansion, or any condition that makes deep breathing painful, may also cause atelectasis. Such compression or pain may result from abdominal surgical incisions, rib fractures, pleuritic chest pain, tight dressings around the chest, stab wounds, impalement accidents, car accidents in which the driver slams into the steering column, or obesity (which elevates the diaphragm and reduces tidal volume).

Prolonged immobility may also cause atelectasis by producing preferential ventilation of one area of the lung over another. Mechanical ventilation using constant small tidal volumes without intermittent deep breaths may also result in atelectasis. Central nervous system depression (as in drug overdose) eliminates periodic sighing and is a predisposing factor of progressive atelectasis.

Signs and symptoms

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.

Diagnosis

Diagnosis requires an accurate patient history, a physical examination, and a chest X-ray. Auscultation reveals diminished or bronchial breath sounds. When much of the lung is collapsed, percussion reveals dullness. However, extensive areas of “microatelectasis” may exist without abnormalities on

the chest X-ray. In widespread atelectasis, the chest X-ray shows characteristic horizontal lines in the lower lung zones. With segmental or lobar collapse, characteristic dense shadows commonly associated with hyperinflation of neighboring lung zones are also apparent. If the cause is unknown, diagnostic procedures may include bronchoscopy to rule out an obstructing neoplasm or a foreign body.

Treatment

Treatment includes incentive spirometry, frequent coughing, and deep-breathing exercises. If atelectasis is secondary to mucus plugging, mucolytics, chest percussion, and postural drainage may be used. If these measures fail, bronchoscopy may be helpful in removing secretions. Humidity and bronchodilators can improve mucociliary clearance and dilate airways.

Atelectasis secondary to an obstructing neoplasm may require surgery or radiation therapy. Postoperative thoracic and abdominal surgery patients require analgesics to facilitate deep breathing, which minimizes the risk of atelectasis.

Special considerations

- If mechanical ventilation is used, tidal volume should be maintained at appropriate levels to ensure adequate expansion of the lungs. Use the sigh mechanism on the ventilator, if appropriate, to intermittently increase tidal volume at the rate of 10 to 15 sighs/hour.
- Use an incentive spirometer to encourage deep inspiration through positive reinforcement. Teach the patient how to use the spirometer, and encourage him to use it every 1 to 2 hours.
- Humidify inspired air and encourage adequate fluid intake to mobilize secretions. To promote loosening and clearance of secretions, encourage deep-breathing and coughing exercises and use postural drainage and chest percussion.
- If the patient is intubated or uncooperative, provide suctioning, as needed. Use sedatives with discretion because they depress respirations and the cough reflex as well as suppress sighing. However, remember that the patient won't cooperate with treatment if he's in pain.

- Assess breath sounds and ventilatory status frequently; report changes at once.
- Teach the patient about respiratory care, including postural drainage, coughing, and deep breathing.
- Encourage the patient to stop smoking and lose weight, as needed. Refer him to appropriate support groups for help.
- Provide reassurance and emotional support; the patient may be anxious due to hypoxia or respiratory distress.

PREVENTION

- *In a patient who is bedridden, encourage movement and deep breathing.*
- *Administer adequate analgesics.*
- *To prevent atelectasis, encourage the postoperative or other high-risk patient to cough and deep-breathe every 1 to 2 hours. To minimize pain during coughing exercises, splint the incision; teach the patient this technique as well. Gently reposition the patient often and encourage ambulation as soon as possible.*
- *Teach patients to keep small objects out of reach of children.*

Respiratory acidosis

An acid-base disturbance characterized by reduced alveolar ventilation and manifested by hypercapnia (partial pressure of arterial carbon dioxide [$PaCO_2$] greater than 45 mm Hg), respiratory acidosis can be acute (due to a sudden failure in ventilation) or chronic (as in long-term pulmonary disease). The prognosis depends on the severity of the underlying disturbance as well as the patient's general clinical condition.

Causes and incidence

Some predisposing factors in respiratory acidosis include:

- Drugs—Opioids, anesthetics, hypnotics, and sedatives, including some of the new designer drugs, such as Ecstasy, decrease the sensitivity of the respiratory center.
- Central nervous system (CNS) trauma —Medullary injury may impair ventilatory drive.
- Chronic metabolic alkalosis—Respiratory compensatory mechanisms attempt to normalize pH by decreasing alveolar ventilation.
- Ventilation therapy—Use of high-flow oxygen (O_2) in chronic respiratory disorders suppresses the patient's hypoxic drive to breathe.
- Neuromuscular diseases (such as myasthenia gravis, Guillain-Barré syndrome, and poliomyelitis)—Failure of the respiratory muscles to respond properly to respiratory drive decreases alveolar ventilation.
- In addition, respiratory acidosis can result from airway obstruction or parenchymal lung disease, which interferes with alveolar ventilation; chronic obstructive pulmonary disease (COPD); asthma; severe acute respiratory distress syndrome; chronic bronchitis; large pneumothorax; extensive pneumonia; and pulmonary edema.

Hypoventilation compromises elimination of carbon dioxide (CO_2) produced through metabolism. The retained CO_2 then combines with water to form an excess of carbonic acid, decreasing the blood pH. As a result, concentration of hydrogen ions in body fluids, which directly reflects acidity, increases.

Complications

- Shock
- Cardiac arrest

Signs and symptoms

Acute respiratory acidosis produces CNS disturbances that reflect changes in the pH of cerebrospinal fluid rather than increased CO_2 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

receiving O_2, 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).

Diagnosis

CONFIRMING DIAGNOSIS

Arterial blood gas (ABG) analysis confirms the diagnosis: $PaCO_2$ exceeds the normal 45 mm Hg; pH is below the normal range of 7.35 to 7.45 unless compensation has occurred; and bicarbonate is normal in the acute stage but elevated in the chronic stage.

Chest X-ray, computed tomography scan, and pulmonary function tests can help determine the cause.

Treatment

Effective treatment of respiratory acidosis requires correction of the underlying source of alveolar hypoventilation.

Significantly reduced alveolar ventilation may require mechanical ventilation until the underlying condition can be treated. In COPD, this includes bronchodilators, O_2, corticosteroids, and antibiotics for infectious conditions; drug therapy for conditions such as myasthenia gravis; removal of foreign bodies from the airway; antibiotics for pneumonia; dialysis or charcoal to remove toxic drugs; and correcting metabolic alkalosis.

Dangerously low blood pH (less than 7.15) can produce profound CNS and cardiovascular deterioration; careful administration of I.V. sodium bicarbonate may be required. In chronic lung disease, elevated CO_2 may persist despite optimal treatment.

Special considerations

- Be alert for critical changes in the patient's respiratory, CNS, and cardiovascular functions. Report such changes as well as any variations in ABG values or electrolyte

  status immediately. Also, maintain adequate hydration.

- Maintain a patent airway and provide adequate humidification if acidosis requires mechanical ventilation. Perform tracheal suctioning regularly and vigorous chest physiotherapy, if ordered. Continuously monitor ventilator settings and respiratory status.
- To prevent respiratory acidosis, closely monitor patients with COPD and chronic CO_2 retention for signs of acidosis. Also, administer O_2 at low flow rates; closely monitor all patients who receive opioids and sedatives. Instruct patients who have received general anesthesia to turn, cough, and perform deep-breathing exercises frequently to prevent the onset of respiratory acidosis.

Respiratory alkalosis

Respiratory alkalosis is an acid-base disturbance characterized by a decrease in the partial pressure of arterial carbon dioxide ($PaCO_2$) to less than 35 mm Hg, which is due to alveolar hyperventilation. Uncomplicated respiratory alkalosis leads to a decrease in hydrogen ion concentration, which results in elevated blood pH. Hypocapnia occurs when the elimination of carbon dioxide (CO_2) by the lungs exceeds the production of CO_2 at the cellular level.

Causes

Causes of respiratory alkalosis fall into two categories:

- pulmonary—severe hypoxemia, pneumonia, interstitial lung disease, pulmonary vascular disease, and acute asthma
- nonpulmonary—anxiety, fever, aspirin toxicity, metabolic acidosis, central nervous system (CNS) disease (inflammation or tumor), sepsis, hepatic failure, and pregnancy.

Complications

- Cardiac arrhythmias
- Seizures

Signs and symptoms

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 CO_2 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.

Diagnosis

CONFIRMING DIAGNOSIS

Arterial blood gas (ABG) analysis confirms respiratory alkalosis and rules out respiratory compensation for metabolic acidosis: $PaCO_2$ less than 35 mm Hg; pH elevated in proportion to the fall in $PaCO_2$ in the acute stage, but falling toward normal in the chronic stage; and bicarbonate normal in the acute stage, but below normal in the chronic stage.

Chest X-ray or pulmonary function tests may aid in diagnosing possible lung disease.

Treatment

Treatment is designed to eradicate the underlying condition—for example, removal of ingested toxins, treatment of fever or sepsis, providing oxygen for acute hypoxemia, and treatment of CNS disease. When hyperventilation is caused by severe anxiety, the patient may be instructed to breathe into a paper bag, which increases CO_2 levels and helps relieve anxiety.

Prevention of hyperventilation in patients receiving mechanical ventilation requires monitoring ABG levels and adjusting tidal volume and minute ventilation.

Special considerations

- Watch for and report any changes in neurologic, neuromuscular, or cardiovascular functions.
- Remember that twitching and cardiac arrhythmias may be associated with alkalemia and electrolyte imbalances. Monitor ABG and serum electrolyte levels closely, reporting any variations immediately.

- Explain all diagnostic tests and procedures to reduce anxiety.

Pneumothorax

Pneumothorax is an accumulation of air or gas between the parietal and visceral pleurae. The amount of air or gas trapped in the intrapleural space determines the degree of lung collapse. In tension pneumothorax, the air in the pleural space is under higher pressure than air in adjacent lung and vascular structures. Without prompt treatment, tension or large pneumothorax results in fatal pulmonary and circulatory impairment.

Causes and incidence

Spontaneous pneumothorax usually occurs in otherwise healthy adults ages 20 to 40. It may be caused by air leakage from ruptured congenital blebs adjacent to the visceral pleural surface, near the apex of the lung. Secondary spontaneous pneumothorax is a complication of underlying lung disease, such as chronic obstructive pulmonary disease, asthma, cystic fibrosis, tuberculosis, and whooping cough. Spontaneous pneumothorax may also occur in interstitial lung disease, such as eosinophilic granuloma or lymphangiomyomatosis.

Traumatic pneumothorax may result from insertion of a central venous line, thoracic surgery, or a penetrating chest injury, such as a gunshot or knife wound. It may follow a transbronchial biopsy, or it may also occur during thoracentesis or a closed pleural biopsy. When traumatic pneumothorax follows a penetrating chest injury, it frequently coexists with hemothorax (blood in the pleural space).

In *tension pneumothorax,* positive pleural pressure develops as a result of traumatic pneumothorax. When air enters the pleural space through a tear in lung tissue and is unable to leave by the same vent, each inspiration traps air in the pleural space, resulting in positive pleural pressure. This in turn causes collapse of the ipsilateral lung and marked impairment of venous return, which can severely compromise cardiac output and may

cause a mediastinal shift. Decreased filling of the great veins of the chest results in diminished cardiac output and lowered blood pressure.

Pneumothorax can also be classified as open or closed. In *open pneumothorax* (usually the result of trauma), air flows between the pleural space and the outside of the body. In *closed pneumothorax,* air reaches the pleural space directly from the lung.

Complication

- Fatal pulmonary and circulatory impairment

Signs and symptoms

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.

Diagnosis

Sudden, sharp chest pain and shortness of breath suggest pneumothorax.

CONFIRMING DIAGNOSIS

Chest X-ray showing air in the pleural space and, possibly, mediastinal shift confirm this diagnosis.

In the absence of a definitive chest X-ray, the physical examination may reveal:

- on inspection—overexpansion and rigidity of the affected chest side; in tension pneumothorax, jugular vein distention with hypotension and tachycardia

- on palpation—crackling beneath the skin, indicating subcutaneous emphysema (air in tissue) and decreased vocal fremitus
- on percussion—hyperresonance on the affected side
- on auscultation—decreased or absent breath sounds over the collapsed lung.

If the pneumothorax is significant, arterial blood gas findings include pH less than 7.35, partial pressure of arterial oxygen less than 80 mm Hg, and partial pressure of arterial carbon dioxide above 45 mm Hg.

Treatment

Treatment is conservative for spontaneous pneumothorax in which no signs of increased pleural pressure (indicating tension pneumothorax) appear, lung collapse is less than 30%, and the patient shows no signs of dyspnea or other indications of physiologic compromise. Such treatment consists of bed rest, careful monitoring of blood pressure and pulse and respiratory rates, oxygen administration and, possibly, needle aspiration of air with a large-bore needle attached to a syringe. If more than 30% of the lung is collapsed, treatment to reexpand the lung includes placing a thoracostomy tube in the second or third intercostal space in the midclavicular line (or in the fifth or sixth intercostal space in the midaxillary line), connected to an underwater seal or low suction pressures.

Recurring spontaneous pneumothorax requires thoracotomy and pleurectomy; these procedures prevent recurrence by causing the lung to adhere to the parietal pleura. Traumatic and tension pneumothoraces require chest tube drainage; traumatic pneumothorax may also require surgery.

Special considerations

ALERT

Watch for pallor, gasping respirations, and sudden chest pain. Monitor patient's vital signs at least every hour for signs of shock, increasing respiratory distress, or mediastinal shift. Listen for breath sounds over both lungs. Falling blood pressure and rising pulse and respiratory rates

may indicate tension pneumothorax, which can be fatal without prompt treatment.

- Urge the patient to control coughing and gasping during thoracotomy. However, after the chest tube is in place, encourage him to cough and breathe deeply (at least once an hour) to facilitate lung expansion.
- If the patient is undergoing chest tube drainage, watch for continuing air leakage (bubbling), indicating the lung defect has failed to close; this may require surgery. Also watch for increasing subcutaneous emphysema by checking around the neck or at the tube insertion site for crackling beneath the skin. If the patient is on a ventilator, watch for difficulty in breathing in time with the ventilator as well as pressure changes on ventilator gauges.
- Change dressings around the chest tube insertion site according to your facility's policy. Don't reposition or dislodge the tube. If it dislodges, immediately place a petroleum gauze dressing over the opening to prevent rapid lung collapse.
- Secure the chest tube drainage apparatus appropriately. Tape connections securely.
- Monitor the patient's vital signs frequently after thoracotomy. Also, for the first 24 hours, assess respiratory status by checking breath sounds hourly. Observe the chest tube site for leakage, noting the amount and color of drainage. Help the patient walk, as ordered (usually on the first postoperative day), to facilitate deep inspiration and lung expansion.
- To reassure the patient, explain what pneumothorax is, what causes it, and all diagnostic tests and procedures. Make him as comfortable as possible. (The patient with pneumothorax is usually most comfortable sitting upright.)

Pneumonia

Pneumonia is an acute infection of the lung parenchyma that commonly impairs gas exchange. The prognosis is generally good for people who have normal lungs and adequate host defenses before the onset of pneumonia; however, pneumonia is the sixth leading cause of death in the United States.

Causes and incidence

Pneumonia can be classified in several ways:

- Microbiologic etiology—Pneumonia can be viral, bacterial, fungal, protozoan, mycobacterial, mycoplasmal, or rickettsial in origin. (See Diagnosing and treating the types of pneumonia, pages 130 to 133.)
- Location—Bronchopneumonia involves distal airways and alveoli; lobular pneumonia, part of a lobe; and lobar pneumonia, an entire lobe.
- Type—Primary pneumonia results from inhalation or aspiration of a pathogen; it includes pneumococcal and viral pneumonia. Secondary pneumonia may follow initial lung damage from a noxious chemical or other insult (superinfection), or may result from hematogenous spread of bacteria from a distant focus.

DIAGNOSING AND TREATING THE TYPES OF PNEUMONIA

Type	Signs and symptoms	Diagnosis	Treatment
Aspiration			
Results from vomiting and aspiration of gastric or oropharyngeal contents into trachea and lungs	▪ Noncardiogenic pulmonary edema that may follow damage to respiratory epithelium from contact with stomach acid ▪ Crackles, dyspnea, cyanosis, hypotension, and tachycardia ▪ May be subacute pneumonia with cavity formation; lung abscess may	▪ *Chest X-ray:* locates areas of infiltrates, which suggest diagnosis	▪ *Antimicrobial therapy:* penicillin G or clindamycin ▪ *Supportive:* oxygen therapy, suctioning, coughing, deep breathing, and adequate hydration

	occur if foreign body is present		
Bacterial			
Klebsiella	▪ Fever and recurrent chills; cough producing rusty, bloody, viscous sputum (currant jelly); cyanosis of lips and nail beds due to hypoxemia; and shallow, grunting respirations ▪ Common in patients with chronic alcoholism, pulmonary disease, diabetes, or those at risk for aspiration	▪ *Chest X-ray:* typically, but not always, consolidation in the upper lobe that causes bulging of fissures ▪ *White blood cell (WBC) count:* elevated ▪ *Sputum culture and Gram stain:* may show gram-negative *Klebsiella*	▪ *Antimicrobial therapy:* an aminoglycoside and a cephalosporin
Staphylococcus	▪ Temperature of 102° to 104° F (38.9° to 40° C), recurrent shaking chills, bloody sputum, dyspnea, tachypnea, and hypoxemia ▪ Should be suspected with viral illness, such as influenza or measles, and in patients with cystic fibrosis	▪ *Chest X-ray:* multiple abscesses and infiltrates; high incidence of empyema ▪ *WBC count:* elevated ▪ *Sputum culture and Gram stain:* may show gram-positive staphylococci	▪ *Antimicrobial therapy:* nafcillin or oxacillin for 14 days if staphylococci are penicillinase producing ▪ *Supportive:* chest tube drainage of empyema
Streptococcus *(Streptococcus pneumoniae)*	▪ Sudden onset of single, shaking chills and a sustained temperature of 102° to 104° F (38.9° to	▪ *Chest X-ray:* areas of consolidation, commonly lobar	▪ *Antimicrobial therapy:* penicillin G (or erythromycin, if patient is allergic to penicillin) for 7 to 10 days beginning after obtaining

	40° C); commonly preceded by upper respiratory tract infection	▪ *WBC count:* elevated ▪ *Sputum culture:* may show gram-positive *S. pneumoniae*; this organism not always recovered	culture specimen but without waiting for results (Resistance to penicillin is becoming much more common and, in the patient with risk factors for resistance [extreme age, day care attendance, or immunosuppression], treatment with vancomycin, imipenem, or levofloxacin should be considered.)
Protozoan			
Pneumocystis carinii (jiroveci)	▪ Occurs in immunocompromised persons ▪ Dyspnea and nonproductive cough ▪ Anorexia, weight loss, and fatigue ▪ Low-grade fever	▪ *Fiber-optic bronchoscopy:* obtains specimens for histologic studies ▪ *Chest X-ray:* nonspecific infiltrates, nodular lesions, or spontaneous pneumothorax	▪ *Antimicrobial therapy:* co-trimoxazole or pentamidine by I.V. administration or inhalation (Prophylactic pentamidine may be used for high-risk patients.) ▪ *Supportive:* oxygen, improved nutrition, and mechanical ventilation
Viral			
Adenovirus (insidious onset; generally affects young adults)	▪ Sore throat, fever, cough, chills, malaise, small amounts of mucoid sputum, retrosternal chest pain, anorexia, rhinitis, adenopathy, scattered crackles, and rhonchi	▪ *Chest X-ray:* patchy distribution of pneumonia, more severe than indicated by physical examination ▪ *WBC count:* normal to slightly elevated	▪ Treat symptoms only ▪ Mortality low; usually clears with no residual effects
Chicken pox (varicella) (uncommon in	▪ Cough, dyspnea, cyanosis, tachypnea, pleuritic chest pain,	▪ *Chest X-ray:* shows more extensive	▪ *Supportive:* adequate hydration, and oxygen therapy in critically ill patients

children, but present in 30% of adults with varicella)	hemoptysis, and rhonchi 1 to 6 days after onset of rash	pneumonia than indicated by physical examination and bilateral, patchy, diffuse, nodular infiltrates ▪ *Sputum analysis:* predominant mononuclear cells and characteristic intranuclear inclusion bodies, with characteristic skin rash, confirm diagnosis	▪ Therapy with I.V. acyclovir
Cytomegalovirus	▪ Difficult to distinguish from other nonbacterial pneumonias ▪ Fever, cough, shaking chills, dyspnea, cyanosis, weakness, and diffuse crackles ▪ Occurs in neonates as devastating multisystemic infection; in normal adults, resembles mononucleosis; in immunocompromised hosts, varies from clinically inapparent to devastating infection	▪ *Chest X-ray:* in early stages, variable patchy infiltrates; later, bilateral, nodular, and more predominant in lower lobes ▪ *Percutaneous aspiration of lung tissue, transbronchial biopsy, or open lung biopsy:* microscopic examination shows typical intranuclear and cytoplasmic inclusions; the virus can be cultured from lung tissue	▪ Generally, benign and self-limiting in mononucleosis-like form ▪ *Supportive:* adequate hydration and nutrition, oxygen therapy, and bed rest ▪ In immunosuppressed patients, disease is more severe and may be fatal; ganciclovir or foscarnet treatment warranted
Influenza	▪ Cough (initially	▪ *Chest X-ray:*	▪ *Supportive:* for respiratory

(prognosis poor even with treatment; 30% mortality)	nonproductive; later, purulent sputum), marked cyanosis, dyspnea, high fever, chills, substernal pain and discomfort, moist crackles, frontal headache, and myalgia ▪ Death results from cardiopulmonary collapse	diffuse bilateral bronchopneumonia radiating from hilus ▪ *WBC count:* normal to slightly elevated ▪ *Sputum smears:* no specific organisms	failure, endotracheal intubation and ventilator assistance; for fever, hypothermia blanket or antipyretics; and for influenza A, amantadine or rimantadine
Measles (rubeola)	▪ Fever, dyspnea, cough, small amounts of sputum, coryza, rash, and cervical adenopathy	▪ *Chest X-ray:* reticular infiltrates, sometimes with hilar lymph node enlargement ▪ *Lung tissue specimen:* characteristic giant cells	▪ *Supportive:* bed rest, adequate hydration, and antimicrobials; assisted ventilation if necessary
Respiratory syncytial virus (most prevalent in infants and children)	▪ Listlessness, irritability, tachypnea with retraction of intercostal muscles, wheezing, slight sputum production, fine moist crackles, fever, severe malaise, and cough	▪ *Chest X-ray:* patchy bilateral consolidation ▪ *WBC count:* normal to slightly elevated	▪ *Supportive:* humidified air, oxygen, antimicrobials (commonly given until viral etiology confirmed), and aerosolized ribavirin ▪ Usually complete recovery

Predisposing factors for bacterial and viral pneumonia include chronic illness and debilitation, cancer (particularly lung cancer), abdominal and thoracic surgery, atelectasis, common colds or other viral respiratory infections, such as acquired immunodeficiency syndrome, chronic respiratory disease (chronic obstructive pulmonary disease [COPD], asthma, bronchiectasis, and cystic fibrosis), influenza, smoking, malnutrition,

alcoholism, sickle cell disease, tracheostomy, exposure to noxious gases, aspiration, and immunosuppressive therapy.

Predisposing factors for aspiration pneumonia include old age, debilitation, artificial airway use, nasogastric (NG) tube feedings, impaired gag reflex, poor oral hygiene, and decreased level of consciousness.

In elderly patients and patients who are debilitated, bacterial pneumonia may follow influenza or a common cold. Respiratory viruses are the most common cause of pneumonia in children ages 2 to 3. In school-age children, mycoplasma pneumonia is more common.

Complications

- Septic shock
- Hypoxemia
- Respiratory failure
- Empyema
- Lung abscess
- Bacteremia
- Endocarditis
- Pericarditis
- Meningitis

Signs and symptoms

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.

Diagnosis

Clinical features, chest X-ray showing infiltrates, and sputum smear demonstrating acute inflammatory cells support the diagnosis. Gram stain and sputum culture may identify the organism. Positive blood cultures in the patient with pulmonary infiltrates strongly suggest pneumonia produced by the organisms isolated from the blood cultures. Pleural effusions, if present, should be tapped and fluid analyzed for evidence of infection in the pleural space. Occasionally, a transtracheal aspirate of tracheobronchial secretions or bronchoscopy with brushings or washings may be done to obtain material for smear and culture. The patient's response to antimicrobial therapy also provides important evidence of the presence of pneumonia.

Treatment

Antimicrobial therapy varies with the causative agent. Therapy should be reevaluated early in the course of treatment. Supportive measures include humidified oxygen therapy for hypoxemia, mechanical ventilation for respiratory failure, a high-calorie diet and adequate fluid intake, bed rest, and an analgesic to relieve pleuritic chest pain. Patients with severe pneumonia on mechanical ventilation may require positive end-expiratory pressure to facilitate adequate oxygenation.

Special considerations

Correct supportive care can increase patient comfort, avoid complications, and speed recovery.

The following protocol should be observed throughout the illness:

- Maintain a patent airway and adequate oxygenation. Monitor pulse oximetry. Measure arterial blood gas levels, especially in hypoxemic patients. Administer supplemental oxygen if the partial pressure of arterial oxygen is less than 55 to 60 mm Hg. Patients with underlying chronic lung disease should be given oxygen cautiously.
- Teach the patient how to cough and perform deep-breathing exercises to clear secretions; encourage him to do so often. In severe pneumonia that requires endotracheal intubation or tracheostomy (with or without mechanical ventilation), provide thorough respiratory care. Suction often, using sterile technique, to remove secretions.

- Obtain sputum specimens as needed, by suction if the patient can't produce specimens independently. Collect specimens in a sterile container and deliver them promptly to the microbiology laboratory.
- Administer antibiotics as ordered and pain medication as needed; record the patient's response to medications. Fever and dehydration may require I.V. fluids and electrolyte replacement.
- Maintain adequate nutrition to offset hypermetabolic state secondary to infection. Ask the dietary department to provide a high-calorie, high-protein diet consisting of soft, easy-to-eat foods. Encourage the patient to eat. As necessary, supplement oral feedings with NG tube feedings or parenteral nutrition. Monitor fluid intake and output. Consider limiting the use of milk products as they may increase sputum production.
- Provide a quiet, calm environment for the patient, with frequent rest periods.
- Give emotional support by explaining all procedures (especially intubation and suctioning) to the patient and his family. Encourage family visits. Provide diversionary activities appropriate to the patient's age.
- To control the spread of infection, dispose of secretions properly. Tell the patient to sneeze and cough into a disposable tissue; tape a lined bag to the side of the bed for used tissues.

Pneumonia can be prevented as follows:

- Advise the patient to avoid using antibiotics indiscriminately during minor viral

  infections because this may result in upper airway colonization with antibioticresistant bacteria. If the patient then develops pneumonia, the organisms producing the pneumonia may require treatment with more toxic antibiotics.
- Encourage pneumovax and annual influenza vaccination for high-risk patients, such as those with COPD, chronic heart disease, or sickle cell disease.
- Urge all bedridden and postoperative patients to perform deep-breathing and coughing exercises frequently. Reposition such patients often to promote full aeration and drainage of secretions. Encourage early ambulation in postoperative patients.

- To prevent aspiration during NG tube feedings, elevate the patient's head, check the tube's position, and administer the formula slowly. Don't give large volumes at one time; this could cause vomiting. Keep the patient's head elevated for at least 30 minutes after the feeding. Check for residual formula at 4- to 6-hour intervals.

PREVENTION

- *Wash hands frequently to prevent infections.*
- *Advise patients to avoid taking antibiotics indiscriminately during viral infections.*

Idiopathic bronchiolitis obliterans with organizing pneumonia

Idiopathic bronchiolitis obliterans with organizing pneumonia (BOOP), also known as *cryptogenic organizing pneumonia,* is one of several types of bronchiolitis obliterans. *Organizing pneumonia* refers to unresolved pneumonia, in which inflammatory alveolar exudate persists and eventually undergoes fibrosis. *Bronchiolitis obliterans* is a generic term used to describe an inflammatory disease of the small airways.

Causes and incidence

BOOP has no known cause. However, other forms of bronchiolitis obliterans and organizing pneumonia may be associated with specific diseases or situations, such as bone marrow, heart, or heart-lung transplantation; collagen vascular diseases, such as rheumatoid arthritis and systemic lupus erythematosus; inflammatory diseases, such as Crohn's disease, ulcerative colitis, and polyarteritis nodosa; bacterial, viral, or mycoplasmal respiratory infections; inhalation of toxic gases; and drug therapy with amiodarone, bleomycin, penicillamine, or lomustine.

Much debate still exists about the various pathologies and classifications of bronchiolitis obliterans. Most patients with BOOP are between ages 50 and 60. Incidence is equally divided between men and women. A smoking history doesn't seem to increase the risk of developing BOOP.

Signs and symptoms

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.

Diagnosis

Diagnosis begins with a thorough patient history meant to exclude any known cause of bronchiolitis obliterans or diseases with a pathology that includes an organizing pneumonia pattern.

- Chest X-ray usually shows patchy, diffuse airspace opacities with a ground-glass appearance that may migrate from one location to another. High-resolution computed tomography scans show areas of consolidation. Except for the migrating opacities, these findings are nonspecific and present in many other respiratory disorders.
- Pulmonary function tests may be normal or show reduced capacities. The diffusing capacity for carbon monoxide is generally low.
- Arterial blood gas analysis usually shows mild to moderate hypoxemia at rest, which worsens with exercise.
- Blood tests reveal an increased erythrocyte sedimentation rate, an increased C-reactive protein level, an increased white blood cell count with a somewhat an increased proportion of neutrophils, and a minor rise in eosinophils. Immunoglobulin (Ig) G and IgM levels are normal or slightly increased, and the IgE level is normal.
- Bronchoscopy reveals normal or slightly inflamed airways. Bronchoalveolar lavage fluid obtained during bronchoscopy shows a moderate elevation in lymphocytes and, sometimes, elevated neutrophil and eosinophil levels. Foamy-looking alveolar macrophages may also be found.

CONFIRMING DIAGNOSIS

Lung biopsy, thoracoscopy, or bronchoscopy is required to confirm the diagnosis of BOOP. Pathologic changes in lung

tissue include plugs of connective tissue in the lumen of the bronchioles, alveolar ducts, and alveolar spaces.

These changes may occur in other types of bronchiolitis and in other diseases that cause organizing pneumonia. They also differentiate BOOP from constrictive bronchiolitis (characterized by inflammation and fibrosis that surrounds and may narrow or completely obliterate the bronchiolar airways). Although the pathologic findings in proliferative and constrictive bronchiolitis are different, the causes and presentations may overlap. Any known cause of bronchiolitis obliterans or organizing pneumonia must be ruled out before the diagnosis of BOOP is made.

Treatment

Corticosteroids are the current treatment for BOOP, although the ideal dosage and duration of treatment remain topics of discussion. Relapse is common when steroids are tapered off or stopped. This usually can be reversed when steroids are increased or resumed. Occasionally, a patient may need to continue corticosteroids indefinitely.

Immunosuppressive-cytotoxic drugs, such as cyclophosphamide, have been used in the few cases of intolerance or unresponsiveness.

Oxygen is used to correct hypoxemia. The patient may need either no oxygen or a small amount of oxygen at rest and a greater amount when he exercises.

Other treatments vary, depending on the patient's symptoms, and may include inhaled bronchodilators, cough suppressants, and bronchial hygiene therapies.

BOOP is very responsive to treatment and usually can be completely reversed with corticosteroid therapy. However, a few deaths have been reported, particularly in patients who had more widespread pathologic changes in the lung or patients who developed opportunistic infections or other complications related to steroid therapy.

Special considerations

- Explain all diagnostic tests. The patient may experience anxiety and frustration because of the length of time and number of tests needed to establish the diagnosis.

- Explain the diagnosis to the patient and his family. This uncommon diagnosis may cause confusion and anxiety.
- Monitor the patient for adverse effects of corticosteroid therapy: weight gain, “moon face,” glucose intolerance, fluid and electrolyte imbalance, mood swings, cataracts, peptic ulcer disease, opportunistic infections, and osteoporosis leading to bone fractures. In many cases, these effects leave the patient unable to tolerate the treatment. Teach the patient and his family about these adverse effects, emphasizing which reactions should be reported to the physician.
- Teach measures that may help prevent complications related to treatment, such as infection control and improved nutrition.
- Teach breathing, relaxation, and energy conservation techniques to help the patient manage symptoms.
- Monitor oxygenation, both at rest and with exertion. The physician will probably prescribe an oxygen flow rate for use when the patient is at rest and a higher one for exertion. Teach the patient how to increase the oxygen flow rate to the appropriate level for exercise.
- If the patient needs oxygen at home, ensure continuity of care by making appropriate referrals to discharge planners, respiratory care practitioners, and home equipment vendors.

Pulmonary embolism

The most common pulmonary complication in hospitalized patients, pulmonary embolism is an obstruction of the pulmonary arterial bed by a dislodged thrombus, heart valve vegetation, or foreign substance. Although pulmonary infarction that results from embolism may be so mild as to be asymptomatic, massive embolism (more than 50% obstruction of pulmonary arterial circulation) and the accompanying infarction can be rapidly fatal.

Causes and incidence

Pulmonary embolism generally results from dislodged thrombi originating in the leg veins. More than half of such thrombi arise in the deep veins of the legs. Other less common sources of thrombi are the pelvic veins, renal veins, hepatic vein, right side of the heart, and upper extremities. Such thrombus formation results directly from vascular wall damage, venostasis,

or hypercoagulability of the blood. Trauma, clot dissolution, sudden muscle spasm, intravascular pressure changes, or a change in peripheral blood flow can cause the thrombus to loosen or fragment. Then the thrombus—now called an *embolus*—floats to the heart's right side and enters the lung through the pulmonary artery. There, the embolus may dissolve, continue to fragment, or grow.

By occluding the pulmonary artery, the embolus prevents alveoli from producing enough surfactant to maintain alveolar integrity. As a result, alveoli collapse and atelectasis develops. If the embolus enlarges, it may clog most or all of the pulmonary vessels and cause death.

Rarely, the emboli contain air, fat, bacteria, amniotic fluid, talc (from drugs intended for oral administration, which are injected intravenously by addicts), or tumor cells.

Predisposing factors for pulmonary embolism include long-term immobility, chronic pulmonary disease, heart failure or atrial fibrillation, thrombophlebitis, polycythemia vera, thrombocytosis, autoimmune hemolytic anemia, sickle cell disease, varicose veins, recent surgery, advanced age, pregnancy, lower-extremity fractures or surgery, burns, obesity, vascular injury, cancer, I.V. drug abuse, or hormonal contraceptives.

Complications

- Pulmonary infarction
- Death

Signs and symptoms

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

(restlessness and anxiety).

Diagnosis

The patient history should reveal predisposing conditions for pulmonary embolism. A triad of deep vein thrombosis (DVT) formation is stasis, endothelial injury, and hypercoagulability. Risk factors include long car or plane trips, cancer, pregnancy, hypercoagulability, prior DVT, and pulmonary emboli.

- Chest X-ray helps to rule out other pulmonary diseases; areas of atelectasis, an elevated diaphragm and pleural effusion, aprominent pulmonary artery and, occasionally, the characteristic wedge-shaped infiltrate suggestive of pulmonary infarction, or focal oligemia of blood vessels, are apparent.
- Lung scan shows perfusion defects in areas beyond occluded vessels; however, it doesn't rule out microemboli.
- Pulmonary angiography is the most definitive test but requires a skilled angiographer and radiologic equipment; it also poses some risk to the patient. Its use depends

  on the uncertainty of the diagnosis and the need to avoid unnecessary anticoagulant therapy in a high-risk patient.
- Electrocardiography may show right axis deviation; right bundle-branch block; tall, peaked P waves; depression of ST segments and T-wave inversions (indicative of right-sided heart strain); and supraventricular tachyarrhythmias in extensive pulmonary embolism. A pattern sometimes observed is S_1, Q_3, and T_3 (S wave in lead I, Q wave in lead III, and inverted T wave in lead III).
- Auscultation occasionally reveals a right ventricular S_3 gallop and an increased intensity of a pulmonic component of S_2. Also, crackles and a pleural rub may be heard at the embolism site.
- Arterial blood gas (ABG) analysis showing a decreased partial pressure of arterial oxygen and partial pressure of arterial carbon dioxide are characteristic but don't always occur.

If pleural effusion is present, thoracentesis may rule out empyema, which indicates pneumonia.

Treatment

Treatment is designed to maintain adequate cardiovascular and pulmonary function during resolution of the obstruction and to prevent recurrence of embolic episodes. Because most emboli resolve within 10 to 14 days, treatment consists of oxygen therapy as needed and anticoagulation with heparin to inhibit new thrombus formation, followed by oral warfarin. Heparin therapy is monitored by daily coagulation studies (partial thromboplastin time [PTT]).

Patients with massive pulmonary embolism and shock may need fibrinolytic therapy with thrombolytic therapy (streptokinase, urokinase, or tissue plasminogen activator) to enhance fibrinolysis of the pulmonary emboli and remaining thrombi. Emboli that cause hypotension may require the use of vasopressors. Treatment of septic emboli requires antibiotics—not anticoagulants—and evaluation for the infection's source, particularly endocarditis.

Surgery is performed on patients who can't take anticoagulants, who have recurrent emboli during anticoagulant therapy, or who have been treated with thrombolytic agents or pulmonary thromboendarterectomy. This procedure (which shouldn't be performed without angiographic evidence of pulmonary embolism) consists of vena caval ligation, plication, or insertion of an inferior vena cava device to filter blood returning to the heart and lungs.

Special considerations

- Give oxygen by nasal cannula or mask. Check ABG levels if the patient develops fresh emboli or worsening dyspnea. Be prepared to provide endotracheal intubation with assisted ventilation if breathing is severely compromised.
- Administer heparin, as ordered, through I.V. push or continuous drip. Monitor coagulation studies daily. Effective heparin therapy raises the PTT to more than 1½ times normal. Watch closely for nosebleeds, petechiae, and other signs of abnormal bleeding; check stools for occult blood. Patients should be protected from trauma and injury; avoid I.M. injections and maintain pressure over venipuncture sites for 5 minutes, or until bleeding stops, to reduce hematoma.

- After the patient is stable, encourage him to move about often, and assist with isometric and range-of-motion exercises. Check pedal pulses, temperature, and color of feet to detect venostasis. *Never* massage the patient's legs. Offer diversional activities to promote rest and relieve restlessness.
- Help the patient walk as soon as possible after surgery to prevent venostasis.
- Maintain adequate nutrition and fluid balance to promote healing.
- Report frequent pleuritic chest pain, so that analgesics can be prescribed. Also, incentive spirometry can assist in deep breathing. Provide tissues and a bag for easy disposal of expectorations.
- Warn the patient not to cross his legs; this promotes thrombus formation.
- To relieve anxiety, explain procedures and treatments. Encourage the patient's family to participate in his care.
- Most patients need treatment with an oral anticoagulant (warfarin) for 3 to 6 months after a pulmonary embolism. Advise these patients to watch for signs of bleeding (bloody stools, blood in urine, and large ecchymoses), to take the prescribed

  medication exactly as ordered, not to change dosages without consulting their physician, and to avoid taking additional medication (including aspirin and vitamins). Stress the importance of follow-up laboratory tests (International Normalized Ratio) to monitor anticoagulant therapy.

PREVENTION

- *Encourage early ambulation in patients predisposed to this condition. With close medical supervision, low-dose heparin may be useful prophylactically.*
- *In high-risk patients, low-molecularweight heparin may be given.*

Sarcoidosis

Sarcoidosis is a multisystem, granulomatous disorder that characteristically produces lymphadenopathy, pulmonary infiltration, and skeletal, liver, eye, or skin lesions. Acute sarcoidosis usually resolves within 2 years. Chronic,

progressive sarcoidosis, which is uncommon, is associated with pulmonary fibrosis and progressive pulmonary disability.

Causes and incidence

The cause of sarcoidosis is unknown, but these factors may play a role:

- hypersensitivity response (possibly from T-cell imbalance) to such agents as atypical mycobacteria, fungi, and pine pollen
- genetic predisposition (suggested by a slightly higher incidence of sarcoidosis within the same family)
- extreme immune response to infection.

Sarcoidosis occurs most commonly in adults ages 30 to 50. In the United States, sarcoidosis occurs predominantly among blacks, affecting twice as many women as men.

Complications

- Pulmonary fibrosis
- Pulmonary hypertension
- Cor pulmonale

Signs and symptoms

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

- 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.

Diagnosis

Typical clinical features with appropriate laboratory data and X-ray findings suggest sarcoidosis. A positive skin lesion biopsy supports the diagnosis.

Other relevant findings include:

- Chest X-ray—bilateral hilar and right paratracheal adenopathy with or without diffuse interstitial infiltrates; occasionally large nodular lesions present in lung parenchyma
- Lymph node or lung biopsy—non-caseating granulomas with negative cultures for mycobacteria and fungi
- Other laboratory data—rarely, increased serum calcium, mild anemia, leukocytosis, and hyperglobulinemia
- Pulmonary function tests—decreased total lung capacity and compliance, and decreased diffusing capacity
- Arterial blood gas (ABG) analysis—decreased arterial oxygen tension.

Negative tuberculin skin test, fungal serologies, and sputum cultures for mycobacteria

and fungi as well as negative biopsy cultures help rule out infection.

Treatment

Sarcoidosis that produces no symptoms requires no treatment. However, those severely affected with sarcoidosis require treatment with corticosteroids. Such therapy is usually continued for 1 to 2 years, but some

patients may need lifelong therapy. Immunosuppressive agents, such as methotrexate, azathioprine, and cyclophosphamide, may also be used. If organ failure occurs (although this is rare), transplantation may be required. Other measures include a low-calcium diet and avoidance of direct exposure to sunlight in patients with hypercalcemia.

Special considerations

- Watch for and report any complications. Be aware of abnormal laboratory results (anemia, for example) that could alter patient care.
- For the patient with arthralgia, administer analgesics as ordered. Record signs of progressive muscle weakness.
- Provide a nutritious, high-calorie diet and plenty of fluids. If the patient has hypercalcemia, suggest a low-calcium diet. Weigh the patient regularly to detect weight loss.
- Monitor respiratory function. Check chest X-rays for the extent of lung involvement; note and record any bloody sputum or increase in sputum. If the patient has pulmonary hypertension or end-stage cor pulmonale, check ABG levels, observe for arrhythmias, and administer oxygen, as needed.
- Because steroids may induce or worsen diabetes mellitus, perform fingerstick glucose tests at least every 12 hours at the beginning of steroid therapy. Also, watch for other steroid adverse effects, such as fluid retention, electrolyte imbalance (especially hypokalemia), moon face, hypertension, and personality change. During or after steroid withdrawal (particularly in association with infection or other types of stress), watch for and report vomiting, orthostatic hypotension, hypoglycemia, restlessness, anorexia, malaise, and fatigue. Remember that the patient on long-term or high-dose steroid therapy is vulnerable to infection.
- When preparing the patient for discharge, stress the need for compliance with prescribed steroid therapy and regular, careful follow-up examinations and treatment. Refer the patient with failing vision to community support and resource groups and the American Foundation for the Blind, if necessary.

Severe acute respiratory syndrome

Severe acute respiratory syndrome (SARS) is a viral respiratory infection that can progress to pneumonia and, eventually, death. The disease was first recognized in 2003 with outbreaks in China, Canada, Singapore, Taiwan, and Vietnam, with other countries—including the United States—reporting smaller numbers of cases.

Causes and incidence

SARS is caused by the SARS-associated coronavirus (SARS-CoV). Coronaviruses are a common cause of mild respiratory illnesses in humans, but researchers believe that a virus may have mutated, allowing it to cause this potentially life-threatening disease.

Close contact with a person who's infected with SARS, including contact with infectious aerosolized droplets or body secretions, is the method of transmission. Most people who contracted the disease during the 2003 outbreak contracted it during travel to endemic areas. However, the virus has been found to live on hands, tissues, and other surfaces for up to 6 hours in its droplet form. It has also been found to live in the stool of people with SARS for up to 4 days. The virus may be able to live for months or years in below-freezing temperatures.

Complications

- Respiratory failure
- Liver failure
- Heart failure
- Myelodysplastic syndromes
- Death

Signs and symptoms

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.

Diagnosis

Diagnosis of severe respiratory illness is made when the patient has a fever greater than 100.4° F (38° C) or upon clinical findings of lower respiratory illness and a chest X-ray demonstrating pneumonia or acute respiratory distress syndrome.

Laboratory validation for the virus includes cell culture of SARS-CoV, detection of SARS-CoV ribonucleic acid by the reverse transcription polymerase chain reaction (PCR) test, or detection of serum antibodies to SARS-CoV. Detectable levels of antibodies may not be present until 21 days after the onset of illness, but some individuals develop antibodies within 14 days. A negative PCR, antibody test, or cell culture doesn't rule out the diagnosis.

Treatment

Treatment is symptomatic and supportive and includes maintenance of a patent airway and adequate nutrition. Other treatment measures include supplemental oxygen, chest physiotherapy, or mechanical ventilation. In addition to standard precautions, contact precautions requiring gowns and gloves for all patient contacts and airborne precautions utilizing a negative-pressure isolation room and properly fitted N-95 respirators are recommended for patients who are hospitalized. Quarantine may be used to prevent the spread of infection.

Antibiotics may be given to treat bacterial causes of atypical pneumonia. Antiviral medications have also been used. High doses of corticosteroids have been used to reduce lung inflammation. In some serious cases, serum from individuals who have already recovered from SARS (convalescent serum) has been given. The general benefit of these treatments hasn't been determined conclusively.

Special considerations

- Report suspected cases of SARS to local and national health organizations.
- Frequently monitor the patient's vital signs and respiratory status.
- Maintain isolation as recommended. The patient will need emotional support to deal with anxiety and fear related to the diagnosis of SARS

and as a result of isolation.

- Provide patient and family teaching, including the importance of frequent hand washing, covering the mouth and nose when coughing or sneezing, and avoiding close personal contact while infected or potentially infected. Instruct the patient and his family that such items as eating utensils, towels, and bedding shouldn't be shared until they have been washed with soap and hot water and that disposable gloves and household disinfectant should be used to clean any surface that may have been exposed to the patient's body fluids.
- Emphasize to the patient the importance of not going to work, school, or other public places, as recommended by the health care provider.

Lung abscess

Lung abscess is a lung infection accompanied by pus accumulation and tissue destruction. The abscess may be putrid (due to anaerobic bacteria) or nonputrid (due to anaerobes or aerobes) and usually has a well-defined border. The availability of effective antibiotics has made lung abscess much less common than it was in the past.

Causes and incidence

Lung abscess is a manifestation of necrotizing pneumonia, generally the result of aspiration of oropharyngeal contents. Poor oral hygiene with dental or gingival (gum) disease is strongly associated with putrid lung abscess. Septic pulmonary emboli commonly produce cavitary lesions. Infected cystic lung lesions and cavitating bronchial carcinoma must be distinguished from lung abscesses.

Complications

- Rupture into pleural space, resulting in empyema
- Massive hemorrhage

Signs and symptoms

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.

Diagnosis

- Auscultation of the chest may reveal crackles and decreased breath sounds.
- Chest X-ray shows a localized infiltrate with one or more clear spaces, usually containing air-fluid levels.
- Percutaneous aspiration or bronchoscopy may be used to obtain cultures to identify the causative organism. Bronchoscopy is only used if abscess resolution is eventful and the patient's condition permits it.
- Blood cultures, Gram stain, and sputum culture are also used to detect the causative organism; leukocytosis (white blood cell count greater than 10,000/µl) is commonly present.

Treatment

Treatment consists of prolonged antibiotic therapy, commonly lasting for months, until radiographic resolution or definite stability occurs. Symptoms usually disappear in a few weeks. Postural drainage may facilitate discharge of necrotic material into the upper airways where expectoration is possible; oxygen therapy may relieve hypoxemia. Poor response to therapy requires resection of the lesion or removal of the diseased section of the lung. All patients need rigorous follow-up and serial chest X-rays.

Special considerations

- Help the patient with chest physiotherapy (including coughing and deep breathing), increase fluid intake to loosen secretions, and provide a quiet, restful atmosphere.
- To prevent lung abscess in the unconscious patient and the patient with seizures, first prevent aspiration of secretions. Do this by suctioning the patient and by positioning him to promote drainage of secretions.

Hemothorax

In hemothorax, blood from damaged intercostal, pleural, mediastinal, and (infrequently) lung parenchymal vessels enters the pleural cavity. Depending on the amount of bleeding and the underlying cause, hemothorax may be associated with varying degrees of lung collapse and mediastinal shift. Pneumothorax—air in the pleural cavity—commonly accompanies hemothorax.

Causes and incidence

Hemothorax usually results from blunt or penetrating chest trauma; in fact, about 25% of patients with such trauma have hemothorax. In some cases, it results from thoracic surgery, pulmonary infarction, neoplasm, dissecting thoracic aneurysm, or a complication of tuberculosis or anticoagulant therapy.

Complications

- Mediastinal shift
- Ventilatory compromise
- Lung collapse
- Cardiopulmonary arrest

Signs and symptoms

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.

Diagnosis

Characteristic clinical signs and a history of trauma strongly suggest hemothorax. Percussion and auscultation reveal dullness and decreased to absent breath sounds over the affected side. Thoracentesis yields blood or serosanguineous fluid; chest X-rays show pleural fluid with or without mediastinal shift. Arterial blood gas (ABG) analysis may reveal respiratory

failure; hemoglobin may be decreased, depending on the amount of blood lost.

Treatment

Treatment is designed to stabilize the patient's condition, stop the bleeding, evacuate blood from the pleural space, and reexpand the underlying lung. Mild hemothorax usually clears in 10 to 14 days, requiring only observation for further bleeding. In severe hemothorax, thoracentesis not only serves as a diagnostic tool but also removes fluid from the pleural cavity.

After the diagnosis is confirmed, a chest tube is inserted into the sixth intercostal space at the posterior axillary line. Suction may be used; a large-bore tube is used to prevent clot blockage. If the chest tube doesn't improve the patient's condition, he may need a thoracotomy to evacuate blood and clots and to control bleeding.

Special considerations

- Give oxygen by face mask or nasal cannula.
- Give I.V. fluids and blood transfusions, as ordered, to treat shock. Monitor pulse oximetry and ABG levels often.
- Explain all procedures to the patient to allay his fears. Assist with thoracentesis. Warn the patient not to cough during this procedure.
- Carefully observe chest tube drainage and record the volume drained (at least every hour). Milk the chest tube (only if necessary and according to facility and physician protocols) to keep it open and free from clots. If the tube is warm and full of blood and the bloody fluid level in the water-seal bottle is rising rapidly, report this at once. The patient may need immediate surgery.
- Watch the patient closely for pallor and gasping respirations. Monitor his vital signs diligently. Falling blood pressure, rising pulse rate, and rising respiratory rate may indicate shock or massive bleeding.

Pulmonary hypertension

Pulmonary hypertension occurs when pulmonary artery pressure (PAP) rises above normal for reasons other than aging or altitude. No definitive set of

values is used to diagnose pulmonary hypertension, but the National Institutes of Health requires a mean PAP of 25 mm Hg or more. The prognosis depends on the cause of the underlying disorder, but the long-term prognosis is poor. Within 5 years of diagnosis, only 25% of patients are still alive.

Causes and incidence

Pulmonary hypertension begins as hypertrophy of the small pulmonary arteries. The medial and intimal muscle layers of these vessels thicken, decreasing distensibility and increasing resistance. This disorder then progresses to vascular sclerosis and obliteration of small vessels.

In most cases, pulmonary hypertension occurs secondary to an underlying disease process, including:

- *alveolar hypoventilation* from chronic obstructive pulmonary disease (most common cause in the United States), sarcoidosis, diffuse interstitial disease, pulmonary metastasis, and certain diseases such as scleroderma (In these disorders, pulmonary vascular resistance occurs secondary to hypoxemia and destruction of the alveolocapillary bed. Other disorders that cause alveolar hypoventilation without lung tissue damage include obesity, kyphoscoliosis, and obstructive sleep apnea.)
- *vascular obstruction* from pulmonary embolism, vasculitis, and disorders that cause obstruction of small or large pulmonary veins, such as left atrial myxoma, idiopathic veno-occlusive disease, fibrosing mediastinitis, and mediastinal neoplasm
- *primary cardiac disease,* which may be congenital or acquired. Congenital defects

  that cause left-to-right shunting of blood—such as patent ductus arteriosus or atrial or ventricular septal defect—increase blood flow into the lungs and, consequently, raise pulmonary vascular pressure. Acquired cardiac diseases, such as rheumatic valvular disease and mitral stenosis, increase pulmonary venous pressure by restricting blood flow returning to the heart.

Primary (or idiopathic) pulmonary hypertension is rare, occurring most commonly—and with no known cause—in women between ages 20 and 40. Secondary pulmonary hypertension results from existing cardiac,

pulmonary, thromboembolic, or collagen vascular diseases or from the use of certain drugs.

Complications

- Cor pulmonale
- Cardiac failure
- Cardiac arrest

Signs and symptoms

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.

Diagnosis

Characteristic diagnostic findings include:

- Auscultation reveals abnormalities associated with the underlying disorder.
- Arterial blood gas (ABG) analysis indicates hypoxemia (decreased partial pressure of arterial oxygen).
- Electrocardiography shows right axis deviation and tall or peaked P waves in inferior leads in the patient with right ventricular hypertrophy.
- Cardiac catheterization reveals pulmonary systolic pressure above 30 mm Hg as well as increased pulmonary artery wedge pressure (PAWP) if the underlying cause is left atrial myxoma, mitral stenosis, or left-sided heart failure (otherwise normal).
- Pulmonary angiography detects filling defects in pulmonary vasculature such as those that develop in patients with pulmonary emboli.
- Pulmonary function tests may show decreased flow rates and increased residual volume in underlying obstructive disease and decreased total lung capacity in underlying restrictive disease.

Treatment

Treatment usually includes oxygen therapy to decrease hypoxemia and resulting pulmonary vascular resistance. It may also include vasodilator therapy (nifedipine [Procardia], diltiazem [Cardizem], or prostaglandin E). For patients with right-sided heart failure, treatment also includes fluid restriction, cardiac glycosides to increase cardiac output, and diuretics to decrease intravascular volume and extravascular fluid accumulation. Treatment also aims to correct the underlying cause.

Some patients with pulmonary hypertension may be candidates for heart-lung transplantation to improve their chances of survival.

Special considerations

Pulmonary hypertension requires keen observation and careful monitoring as well as skilled supportive care.

- Administer oxygen therapy as ordered, and observe the patient's response. Report any signs of increasing dyspnea to the physician so he can adjust treatment accordingly.
- Monitor ABG levels for acidosis and hypoxemia. Report any change in the patient's level of consciousness at once.
- When caring for a patient with right-sided heart failure, especially one receiving diuretics, record his weight daily, carefully measure intake and output, and explain all medications and diet restrictions. Check for worsening jugular vein distention, which may indicate fluid overload.
- Monitor the patient's vital signs, especially blood pressure and heart rate. Watch for hypotension and tachycardia. If he has a pulmonary artery catheter, check PAP and PAWP, as indicated. Report any changes.
- Before discharge, help the patient adjust to the limitations imposed by this disorder. Advise against overexertion, and suggest frequent rest periods between activities. Refer the patient to the social services department

  if he'll need special equipment, such as oxygen equipment, for home use. Make sure that he understands the prescribed medications and diet and the need to weigh himself daily.

Pleural effusion and empyema

Pleural effusion is an excess of fluid in the pleural space. Normally, this space contains a small amount of extracellular fluid that lubricates the pleural surfaces. Increased production or inadequate removal of this fluid results in pleural effusion. Empyema is the accumulation of pus and necrotic tissue in the pleural space. Blood (hemothorax) and chyle (chylothorax) may also collect in this space.

Causes and incidence

The balance of osmotic and hydrostatic pressures in parietal pleural capillaries normally results in fluid movement into the pleural space. Balanced pressures in visceral pleural capillaries promote reabsorption of this fluid. Excessive hydrostatic pressure or decreased osmotic pressure can cause excessive amounts of fluid to pass across intact capillaries. The result is a transudative pleural effusion, an ultrafiltrate of plasma containing low concentrations of protein. Such effusions frequently result from heart failure, hepatic disease with ascites, peritoneal dialysis, hypoalbuminemia, and disorders resulting in overexpanded intravascular volume.

Exudative pleural effusions result when capillaries exhibit increased permeability with or without changes in hydrostatic and colloid osmotic pressures, allowing protein-rich fluid to leak into the pleural space. Exudative pleural effusions occur with tuberculosis (TB), subphrenic abscess, pancreatitis, bacterial or fungal pneumonitis or empyema, malignancy, pulmonary embolism with or without infarction, collagen disease (lupus erythematosus [LE] and rheumatoid arthritis), myxedema, and chest trauma.

Empyema is usually associated with infection in the pleural space. Such infection may be idiopathic or may be related to pneumonitis, carcinoma, perforation, or esophageal rupture.

Complications

- Atelectasis
- Infection
- Hypoxemia

Signs and symptoms

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.

Diagnosis

Auscultation of the chest reveals decreased breath sounds; percussion detects dullness over the effused area, which doesn't change with breathing. Chest X-ray shows fluid in dependent regions. However, diagnosis also requires other tests to distinguish transudative from exudative effusions and to help pinpoint the underlying disorder.

The most useful test is thoracentesis, in which pleural fluid is analyzed in the laboratory to show components. Acute inflammatory white blood cells and microorganisms may be evident in empyema.

In addition, if a pleural effusion results from esophageal rupture or pancreatitis, fluid amylase levels are usually higher than serum levels. Aspirated fluid may be tested for LE cells, antinuclear antibodies, and neoplastic cells. It may also be analyzed for color and consistency; acid-fast bacillus, fungal, and bacterial cultures; and triglycerides (in chylothorax). Cell analysis shows leukocytosis in empyema. A negative tuberculin skin test strongly rules against TB as the cause. In exudative pleural effusions in which thoracentesis isn't definitive, pleural biopsy may be done. This is particularly useful for confirming TB or malignancy.

Treatment

Depending on the amount of fluid present, symptomatic effusion may require thoracentesis to remove fluid or careful monitoring of the patient's own reabsorption of the fluid. Hemothorax requires drainage to prevent fibrothorax formation. Pleural effusions associated with lung cancer commonly reaccumulate quickly. If a chest tube is inserted to drain the fluid, a sclerosing agent, such as talc, may be injected through the tube to cause adhesions between the parietal and visceral pleura, thereby obliterating the potential space for fluid to recollect.

Treatment of empyema requires insertion of one or more chest tubes after thoracentesis, to allow drainage of purulent material, and possibly decortication (surgical removal of the thick coating over the lung) or rib resection to allow open drainage and lung expansion. Empyema also requires parenteral antibiotics. Associated hypoxia requires oxygen administration.

Special considerations

- Explain thoracentesis to the patient. Before the procedure, tell him to expect a stinging sensation from the local anesthetic and a feeling of pressure when the needle is inserted. Instruct him to tell you immediately if he feels uncomfortable or has difficulty breathing during the procedure.
- Reassure the patient during thoracentesis. Remind him to breathe normally and avoid sudden movements, such as coughing or sighing. Monitor his vital signs, and watch for syncope. If fluid is removed too quickly, the patient may suffer bradycardia, hypotension, pain, pulmonary edema, or even cardiac arrest. Watch for respiratory distress or pneumothorax (sudden onset of dyspnea and cyanosis) after thoracentesis.
- Administer oxygen and, in empyema, antibiotics, as ordered.
- Encourage the patient to perform deep-breathing exercises to promote lung expansion. Use an incentive spirometer to promote deep breathing.
- Provide meticulous chest tube care, and use sterile technique for changing dressings around the tube insertion site in empyema. Ensure tube patency by watching for fluctuations of fluid or air bubbling in the underwater seal chamber. Continuous bubbling may indicate an air leak. Record the amount, color, and consistency of any tube drainage.
- If the patient has open drainage through a rib resection or intercostal tube, use hand and dressing precautions. Because weeks of such drainage are usually necessary to obliterate the space, make visiting nurse referrals for the patient who will be discharged with the tube in place.
- If pleural effusion was a complication of pneumonia or influenza, advise prompt medical attention for upper respiratory infections.

Pleurisy

Pleurisy, also known as *pleuritis,* is an inflammation of the visceral and parietal pleurae that line the inside of the thoracic cage and envelop the lungs.

Causes and incidence

Pleurisy develops as a complication of pneumonia, tuberculosis, viruses, systemic lupus erythematosus, rheumatoid arthritis, uremia, Dressler's syndrome, certain cancers, pulmonary infarction, and chest trauma. Pleuritic pain is caused by the inflammation or irritation of sensory nerve endings in the parietal pleura. As the lungs inflate and deflate, the visceral pleura covering the lungs moves against the fixed parietal pleura lining the pleural space, causing pain. This disorder usually begins suddenly.

In the United States, pleural effusions develop in 36% to 66% of hospitalized patients with bacterial pneumonia.

Signs and symptoms

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.

Diagnosis

Auscultation of the chest reveals a characteristic pleural friction rub—a coarse, creaky sound heard during late inspiration and early expiration, directly over the area of pleural inflammation. Palpation over the affected area may reveal coarse vibration. Chest X-ray, ultrasound of the chest, and thoracentesis may aid in diagnosis.

Treatment

Treatment is directed at the underlying cause; bacterial infections are treated with appropriate antibiotics, tuberculosis requires special treatment, and viral infections may be permitted to run their course. Treatment also includes measures to relieve symptoms, such as anti-inflammatory agents, analgesics, and bed rest. Severe pain may require an intercostal nerve block of two or three intercostal nerves. Pleurisy with pleural effusion calls for thoracentesis as a therapeutic and diagnostic measure.

Special considerations

- Stress the importance of bed rest, and plan your care to allow the patient as much uninterrupted rest as possible.
- Administer antitussives and pain medication, as ordered, but be careful not to overmedicate. If the pain requires an opioid analgesic, warn the patient who's about to be discharged to avoid overuse because such medication depresses coughing and respiration.
- Encourage the patient to cough. Tell him to apply firm pressure at the site of the pain during coughing exercises to minimize pain.

CHRONIC DISORDERS

Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is chronic airway obstruction that results from emphysema, chronic bronchitis, asthma, or any combination of these disorders. (See *Types of chronic obstructive pulmonary disease,* pages 148 to 151. Also see *Three types of emphysema,* page 152.) Usually, more than one of these underlying conditions coexist; in most cases, bronchitis and emphysema occur together. It doesn't always produce symptoms and causes only minimal disability in many patients. However, COPD tends to worsen with time.

Causes and incidence

Predisposing factors include cigarette smoking, recurrent or chronic respiratory infections, air pollution, occupational exposure to chemicals, and allergies. Smoking is by far the most important of these factors—it impairs ciliary action and macrophage function, inflames airways, increases mucus production, destroys alveolar septae, and causes peribronchiolar fibrosis. Early inflammatory changes may reverse if the patient stops smoking before lung destruction is extensive. Familial and hereditary factors (such as deficiency of alpha1-antitrypsin) may also predispose a person to COPD.

The most common chronic lung disease, COPD (also known as chronic obstructive lung disease) affects an estimated 17 million Americans, and its incidence is rising. It affects more males than females, probably because

until recently men were more likely to smoke heavily. COPD occurs mostly in people older than age 40.

Complications

- Overwhelming disability
- Cor pulmonale
- Severe respiratory failure
- Death

Signs and symptoms

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.

TYPES OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Disease	Causes and pathophysiology	Clinical features	Confirming diagnostic measures	Management
Asthma				
▪ Increased bronchial reactivity to many stimuli, which produces	▪ Reversible airway inflammation usually occurs in response to an allergen. Swelling of membranes,	▪ History of intermittent attacks of dyspnea and wheezing	▪ *Physical examination:* usually normal between attacks; auscultation reveals rhonchi	▪ Aerosol containing beta-adrenergic agents, such as metaproterenol or albuterol;

episodic bronchospasm and airway obstruction in conjunction with airway inflammation ▪ Asthma with onset in adulthood: usually without distinct allergies; asthma with onset in childhood: commonly associated with definite allergens. Status asthmaticus is an acute asthma attack with severe bronchospasm that fails to clear with bronchodilator therapy. ▪ *Prognosis:* More than half of children with asthma become asymptomatic as adults; more than half of asthmatics with onset	bronchoconstriction, and production of mucus obstruct airways. Activated mast cells release chemical mediators, including histamine, bradykinin, prostaglandins, and leukotrienes, which perpetuates the inflammatory response. ▪ Upper airway infection, exercise, anxiety and, rarely, coughing or laughing can precipitate an asthma attack; nocturnal flare-ups are common. ▪ Paroxysmal airway obstruction associated with nasal polyps may be seen in response to aspirin or indomethacin ingestion. ▪ Airway obstruction from spasm of bronchial smooth muscle narrows airways; inflammatory edema of the bronchial wall and inspissation of tenacious mucoid secretions are also important, particularly in status asthmaticus.	▪ Mild wheezing progresses to severe dyspnea, audible wheezing, chest tightness (a feeling of not being able to breathe), and cough that produces thick mucus. ▪ *Other signs:* prolonged expiration, intercostal and supraclavicular retraction on inspiration, use of accessory muscles of respiration, flaring nostrils, tachypnea, tachycardia, perspiration, and flushing; patients usually have symptoms of eczema and allergic rhinitis (hay fever). ▪ Status asthmaticus, unless treated promptly, can progress to	and wheezing throughout lung fields on expiration and, at times, inspiration and absent or diminished breath sounds during severe obstruction. Loud bilateral wheezing may be grossly audible; chest is hyperinflated. ▪ *Chest X-ray:* hyperinflated lungs with air trapping during attack; normal during remission ▪ *Sputum:* presence of Curschmann's spirals (casts of airways), Charcot-Leyden crystals, and eosinophils ▪ *Pulmonary function tests (PFTs):* during attacks, decreased forced expiratory volumes that improve significantly after inhaled bronchodilator; increased residual volume and, occasionally, total lung capacity;	also oral beta-adrenergic agents (terbutaline) and oral methylxanthines (theophylline). Many patients require inhaled, oral, or I.V. corticosteroids. ▪ *Emergency treatment:* oxygen therapy, I.V. corticosteroids, and bronchodilators, such as subcutaneous epinephrine, I.V. theophylline, and inhaled agents (such as metaproterenol, albuterol, or ipratropium bromide). ▪ Monitor for deteriorating respiratory status and note sputum characteristics; provide adequate fluid intake and oxygen, as ordered. ▪ *Prevention:* Tell the patient

after age 15 have persistent disease, with occasional severe attacks.		respiratory failure.	may be normal between attacks ▪ *Arterial blood gas (ABG) analysis:* decreased partial pressure of arterial oxygen (PaO_2); decreased, normal, or increased partial pressure of arterial carbon dioxide ($PaCO_2$; in severe attack) ▪ *Electrocardiogram (ECG):* sinus tachycardia during an attack; severe attack may produce signs of cor pulmonale (right axis deviation, peaked P wave) that resolve after the attack ▪ *Skin tests:* may identify allergens	to avoid possible triggers and to use inhaled corticosteroids, antihistamines, decongestants, cromolyn powder by inhalation, leukotriene modifier, and oral or aerosol bronchodilators, as ordered. Explain the influence of stress and anxiety on asthma as well as its frequent association with exercise (particularly running), cold air, and nighttime flare-ups. ▪ Help the patient identify the triggers and teach him how to use a metered-dose inhaler and a peak flow meter.
Chronic bronchitis				
▪ Excessive	▪ Severity of disease	Insidious	▪ *Physical*	▪ Antibiotics for

mucus production with productive cough for at least 3 months per year for 2 successive years ▪ Only a minority of patients with the clinical syndrome of chronic bronchitis develop significant airway obstruction.	related to the amount and duration of smoking; respiratory infection exacerbates symptoms ▪ Hypertrophy and hyperplasia of bronchial mucous glands, increased goblet cells, damage to cilia, squamous metaplasia of columnar epithelium, and chronic leukocytic and lymphocytic infiltration of bronchial walls; widespread inflammation, distortion, narrowing of airways, and mucus within the airways produce resistance in small airways and cause severe ventilation-perfusion imbalance	onset, with productive cough and exertional dyspnea predominant symptoms ▪ *Other signs and symptoms:* upper respiratory infections associated with increased sputum production and worsening dyspnea, which take progressively longer to resolve; copious sputum (gray, white, or yellow); weight gain due to edema; cyanosis; tachypnea; wheezing; prolonged expiratory time; and use of accessory muscles of respiration ▪ Complications include recurrent respiratory	*examination:* barrel chest, rhonchi and wheezes on auscultation, prolonged expiration, jugular vein distention, and pedal edema ▪ *Chest X-ray:* may show hyperinflation and increased bronchovascular markings ▪ *PFTs:* increased residual volume, decreased vital capacity and forced expiratory volumes, and normal static compliance and diffusing capacity ▪ *ABG analysis:* decreased PaO_2, normal or increased $PaCO_2$ ▪ *ECG:* may show atrial arrhythmias; peaked P waves in leads II, III, and aV_F; and, occasionally, right ventricular hypertrophy	infections ▪ Avoidance of smoking and air pollutants ▪ Bronchodilators to relieve bronchospasm and facilitate mucociliary clearance ▪ Adequate fluid intake and chest physiotherapy to mobilize secretions ▪ Ultrasonic or mechanical nebulizer treatments to loosen secretions and aid in mobilization ▪ Occasionally, corticosteroids ▪ Diuretics for edema ▪ Oxygen for hypoxemia or cor pulmonale

		tract infections, cor pulmonale, and polycythemia.		
Emphysema				
▪ Abnormal, irreversible enlargement of air spaces distal to terminal bronchioles due to destruction of alveolar walls, resulting in decreased elastic recoil properties of lungs ▪ Most common cause of death from respiratory disease in the United States	▪ Cigarette smoking and congenital deficiency of alphaantitrypsin ▪ Recurrent inflammation associated with release of proteolytic enzymes from cells in lungs causes bronchiolar and alveolar wall damage and, ultimately, destruction. Loss of lung supporting structure results in decreased elastic recoil and airway collapse on expiration. Destruction of alveolar walls decreases surface area for gas exchange.	▪ Insidious onset, with dyspnea the predominant symptom ▪ *Other signs and symptoms of long-term disease:* anorexia, weight loss, malaise, barrel chest, use of accessory muscles of respiration, prolonged expiratory period with grunting, pursed-lip breathing, and tachypnea ▪ *Complications* include recurrent respiratory tract infections, cor pulmonale, and respiratory failure.	▪ *Physical examination:* barrel chest, hyperresonance on percussion, decreased breath sounds, expiratory prolongation, and quiet heart sounds ▪ *Chest X-ray:* in advanced disease, flattened diaphragm, reduced vascular markings at lung periphery, hyperexpansion of lungs, enlarged anteroposterior chest diameter, and large retrosternal air space ▪ *Computed tomography scan:* may show emphysema ▪ *PFTs:* increased residual volume, total lung capacity, and compliance; decreased vital capacity, diffusing	▪ Oxygen at low-flow settings to treat hypoxia ▪ Avoidance of smoking and air pollutants ▪ Breathing techniques to control dyspnea ▪ Treatment only slightly helpful for emphysema component of chronic obstructive pulmonary disease ▪ Lung volume reduction surgery for selected patients

		capacity, and expiratory volumes ▪ *ABG analysis:* reduced PaO2 with normal $PaCO_2$ until late in disease ▪ *ECG:* tall, symmetrical P waves in leads II, III, and aV_F; vertical QRS axis; signs of right ventricular hypertrophy late in disease ▪ *Red blood cell count:* increased hemoglobin late in disease when persistent severe hypoxia is present

Diagnosis

For specific diagnostic tests used to determine COPD, see *Types of chronic obstructive pulmonary disease*.

Treatment

Treatment is designed to relieve symptoms and prevent complications. Because most patients with COPD receive outpatient treatment, they need comprehensive teaching to help them comply with therapy and understand the nature of this chronic, progressive disease. If programs in pulmonary rehabilitation are available, encourage patients to enroll.

Urge the patient to stop smoking. Provide smoking cessation counseling or refer him to a program. Avoid other respiratory irritants, such as secondhand smoke, aerosol spray products, and outdoor air pollution. An air conditioner with an air filter in his home may be helpful.

The patient is usually treated with betaagonist bronchodilators (albuterol or salmeterol), anticholinergic bronchodilators (ipratropium), and corticosteroids (beclomethasone or triamcinolone). These are usually given by metered-dose inhaler, requiring that the patient be taught the correct administration technique.

Antibiotics are used to treat respiratory infections. Stress the need to complete the prescribed course of antibiotic therapy.

Special considerations

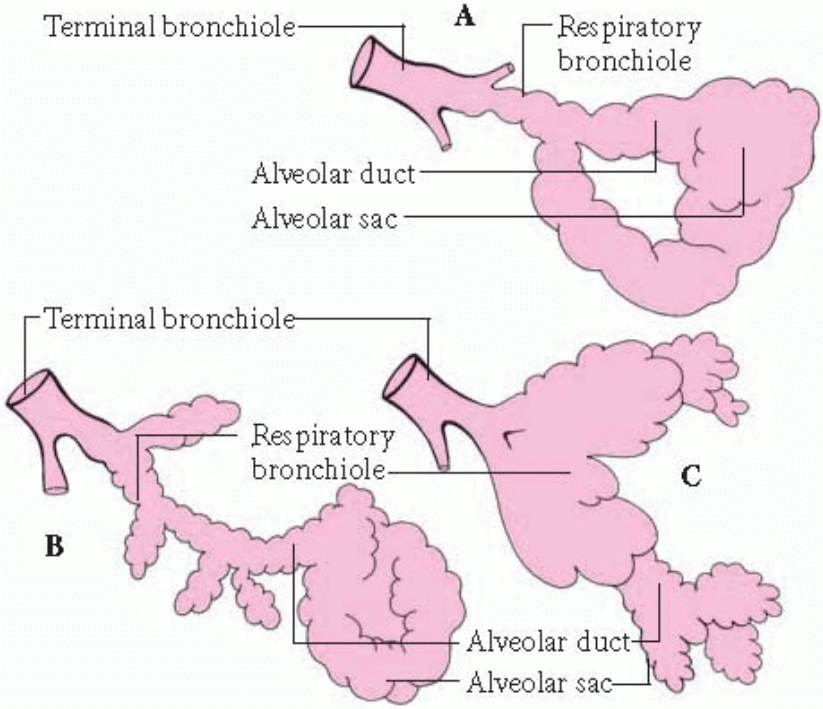
- Teach the patient and his family how to recognize early signs of infection; warn the patient to avoid contact with people with respiratory infections. Encourage good oral hygiene to help prevent infection. Pneumococcal vaccination and annual influenza vaccinations are important preventive measures.
- To promote ventilation and reduce air trapping, teach the patient to breathe slowly, prolong expirations to two to three times the duration of inspiration, and to exhale through pursed lips.
- To help mobilize secretions, teach the patient how to cough effectively. If the patient with copious secretions has difficulty mobilizing secretions, teach his family how to perform postural drainage and chest physiotherapy. If secretions are thick, urge

  the patient to drink 12 to 15 glasses of fluid per day. A home humidifier may be beneficial, particularly in the winter.
- Administer low concentrations of oxygen as ordered. Perform blood gas analysis to determine the patient's oxygen needs and to avoid carbon dioxide narcosis. If the patient is to continue oxygen therapy at home, teach him how to use the equipment correctly. The patient with COPD rarely requires more than 2 to 3 L/minute to maintain adequate oxygenation. Higher flow rates will further increase the partial pressure of arterial oxygen, but the patient whose ventilatory drive is largely based on hypoxemia commonly develops markedly increased partial pressure of arterial carbon dioxide tensions. In these cases, chemoreceptors in the brain are relatively insensitive to the increase in carbon dioxide. Teach the patient and his family that excessive oxygen therapy may eliminate the hypoxic respiratory drive, causing confusion and drowsiness, signs of carbon dioxide narcosis.

- Emphasize the importance of a balanced diet. Because the patient may tire easily when eating, suggest that he eat frequent, small meals and consider using oxygen, administered by nasal cannula, during meals.
- Help the patient and his family adjust their lifestyles to accommodate the limitations imposed by this debilitating chronic disease. Instruct the patient to allow for daily rest periods and to exercise daily as his physician directs.
- As COPD progresses, encourage the patient to discuss his fears.
- To help prevent COPD, advise all patients, especially those with a family history of COPD or those in its early stages, not to smoke.
- Assist in the early detection of COPD by urging persons to have periodic physical examinations, including spirometry and medical evaluation of a chronic cough, and to seek treatment for recurring respiratory infections promptly.
- Lung volume reduction surgery is a new procedure for carefully selected patients with primarily emphysema. Nonfunctional parts of the lung (tissue filled with disease and providing little ventilation or perfusion) are surgically removed. Removal allows more functional lung tissue to expand and the diaphragm to return to its normally elevated position.

THREE TYPES OF EMPHYSEMA

A Panacinar (panlobular): destroys alveoli and alveolar ducts; associated with aging and alpha$_1$-antitrypsin deficiency **B** Paraseptal (distal acinar): commonly causes spontaneous pneumothorax in young adults **C** Centriacinar (centrilobular): associated with chronic bronchitis and smoking; destroys respiratory bronchioles



Bronchiectasis

A condition marked by chronic abnormal dilation of bronchi and destruction of bronchial walls, bronchiectasis can occur throughout the tracheobronchial tree or can be confined to one segment or lobe. However, it's usually bilateral and involves the basilar segments of the lower lobes. This disease has three forms: cylindrical (fusiform), varicose, and saccular (cystic). Bronchiectasis is irreversible once established.

Causes and incidence

Because of the availability of antibiotics to treat acute respiratory tract infections, the incidence of bronchiectasis has dramatically decreased in the past 20 years. Incidence is highest among Eskimos and the Maoris of New Zealand. It affects people of both sexes and all ages.

The different forms of bronchiectasis may occur separately or simultaneously. In *cylindrical bronchiectasis,* the bronchi expand unevenly, with little change in diameter, and end suddenly in a squared-off fashion. In *varicose bronchiectasis,* abnormal, irregular dilation and narrowing of the bronchi give the appearance of varicose veins. In *saccular bronchiectasis,* many large dilations end in sacs. These sacs balloon into

pus-filled cavities as they approach the periphery and are then called saccules. (See *Forms of bronchial dilatation*.)

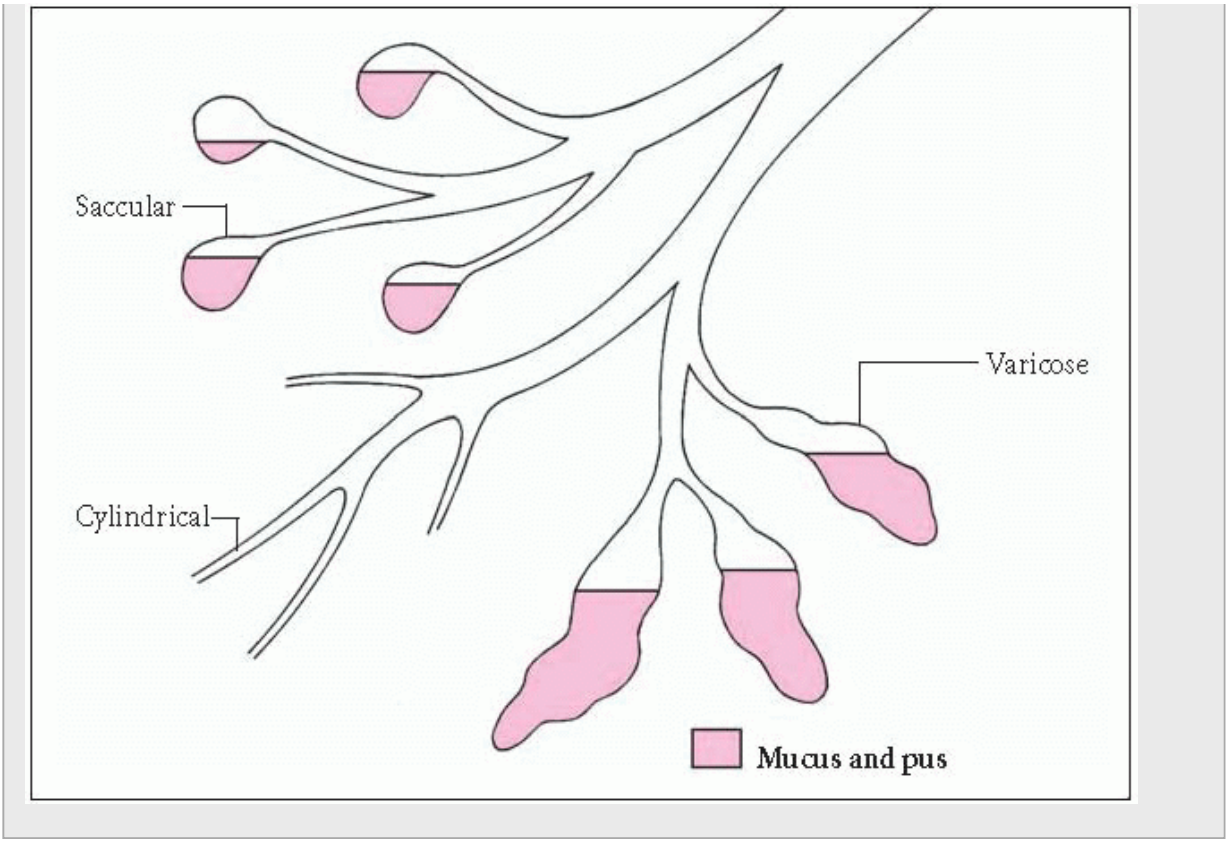
This disease results from conditions associated with repeated damage to bronchial walls and abnormal mucociliary clearance, which cause a breakdown of supporting tissue adjacent to airways. Such conditions include:

- cystic fibrosis
- immunologic disorders (agammaglobulinemia, for example)
- recurrent, inadequately treated bacterial respiratory tract infections, such as tuberculosis, and complications of measles, pneumonia, pertussis, or influenza

- obstruction (by a foreign body—most common in children, tumor, or stenosis) in association with recurrent infection
- inhalation of corrosive gas or repeated aspiration of gastric juices into the lungs
- congenital anomalies (uncommon), such as bronchomalacia, congenital bronchiectasis, immotile cilia syndrome, and Kartagener's syndrome, a variant of immotile cilia syndrome characterized by situs inversus, bronchiectasis, and either nasal polyps or sinusitis.

FORMS OF BRONCHIAL DILATATION

Dilatations of the air sacs occur due to bronchiectasis, as depicted below.



In bronchiectasis, hyperplastic squamous epithelium denuded of cilia replaces ulcerated columnar epithelium. Abscess formation involving all layers of the bronchial wall produces inflammatory cells and fibrous tissue, resulting in dilation and narrowing of the airways. Mucus plugs or fibrous tissue obliterates smaller bronchioles, whereas peribronchial lymphoid tissue becomes hyperplastic. Extensive vascular proliferation of bronchial circulation occurs and produces frequent hemoptysis.

Complications

- Chronic malnutrition
- Amyloidosis
- Right ventricular failure
- Cor pulmonale

Signs and symptoms

Initially, bronchiectasis may be asymptomatic. When symptoms do arise, they're commonly attributed to other illnesses. The patient usually

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.

Advanced bronchiectasis may produce chronic malnutrition as well as right-sided heart failure and cor pulmonale due to hypoxic pulmonary vasoconstriction.

Diagnosis

A history of recurrent bronchial infections, pneumonia, and hemoptysis in a patient whose chest X-rays show peribronchial thickening, areas of atelectasis, and scattered cystic changes suggest bronchiectasis.

In recent years, computed tomography scanning has supplanted bronchography as the most useful diagnostic test for bronchiectasis. It's sometimes used with high-resolution techniques to better determine anatomic changes. Bronchoscopy doesn't establish the diagnosis of bronchiectasis, but it does help to identify the source of secretions. Bronchoscopy can also be instrumental in pinpointing the site of bleeding in hemoptysis.

Other helpful laboratory tests include:

- sputum culture and Gram stain to identify predominant organisms
- complete blood count to detect anemia and leukocytosis
- pulmonary function tests to detect decreased vital capacity, expiratory flow rate, and hypoxemia. These tests also help determine the physiologic severity of the disease and the effects of therapy and help evaluate patients for surgery.

When cystic fibrosis is suspected as the underlying cause of bronchiectasis, a sweat electrolyte test is useful.

Treatment

Treatment includes antibiotics, given orally or I.V., for 7 to 10 days or until sputum production decreases. Bronchodilators, combined with postural drainage and chest percussion, help remove secretions if the patient has bronchospasm and thick, tenacious sputum. Bronchoscopy may be used to remove obstruction and secretions. Hypoxia requires oxygen therapy; severe hemoptysis commonly requires lobectomy, segmental resection, or bronchial artery embolization if pulmonary function is poor. Long-term antibiotic therapy isn't appropriate because it may predispose the patient to serious gram-negative infections and resistant organisms.

Special considerations

- Provide supportive care and help the patient adjust to the permanent changes in lifestyle that irreversible lung damage necessitates. Thorough teaching is vital.
- Administer antibiotics as ordered, and explain all diagnostic tests. Perform chest physiotherapy, including postural drainage and chest percussion designed for involved lobes, several times a day. The best times to do this are early morning and just before bedtime. Instruct the patient to maintain each position for 10 minutes, and then perform percussion and tell him to cough. Show family members how to perform postural drainage and percussion. Also teach the patient coughing and deep-breathing techniques to promote good ventilation and the removal of secretions.
- Advise the patient to stop smoking, if appropriate, to avoid stimulating secretions and irritating the airways. Refer him to a local self-help group.
- Provide a warm, quiet, comfortable environment, and urge the patient to rest as much as possible. Encourage balanced, high-protein meals to promote good health and tissue healing and plenty of fluids (2 to 3 qt [2 to 3 L]) per day to hydrate and thin bronchial secretions). Give frequent mouth care to remove foul-smelling sputum. Teach the patient to dispose of all secretions properly. Instruct him to seek prompt attention for respiratory infections.
- Tell the patient to avoid air pollutants and people with upper respiratory tract infections. Instruct him to take medications (especially antibiotics) exactly as prescribed.

PREVENTION

- *Treat bacterial pneumonia vigorously.*
- *Stress the need for immunization to prevent childhood diseases.*

Idiopathic pulmonary fibrosis

Idiopathic pulmonary fibrosis (IPF) is a chronic and usually fatal interstitial pulmonary disease. About 50% of patients with IPF die within 5 years of diagnosis. Once thought to be a rare condition, it's now diagnosed with much greater frequency. IPF has been known by several other names over the years, including cryptogenic fibrosing alveolitis, diffuse interstitial fibrosis, idiopathic interstitial pneumonitis, and Hamman-Rich syndrome.

Causes and incidence

IPF is the result of a cascade of events that involve inflammatory, immune, and fibrotic processes in the lung. However, despite many studies and hypotheses, the stimulus that begins the progression remains unknown. Speculation has revolved around viral and genetic causes, but no good evidence has been found to support either theory. However, it's clear that chronic inflammation plays an important role. Inflammation develops the injury and the fibrosis that ultimately distorts and impairs the structure and function of the alveolocapillary gas exchange surface.

IPF is slightly more common in men than in women and is more common in smokers than in nonsmokers. It usually affects people ages 50 to 70.

Signs and symptoms

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 S_2 and S_3

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

Diagnosis

Diagnosis begins with a thorough patient history to exclude a more common cause of interstitial lung disease.

CONFIRMING DIAGNOSIS

Lung biopsy is helpful in the diagnosis of IPF. In the past, an open lung biopsy was the only acceptable procedure, but now biopsies may be done through a thoracoscope or bronchoscope.

Histologic features of the biopsy tissue vary, depending on the stage of the disease and other factors that aren't yet completely understood. The alveolar walls are swollen with chronic inflammatory cellular infiltrate composed of mononuclear cells and polymorphonuclear leukocytes. Intraalveolar inflammatory cells may be found in early stages. As the disease progresses, excessive collagen and fibroblasts fill the interstitium. In advanced stages, alveolar walls are destroyed and are replaced by honeycombing cysts.

Chest X-rays may show one of four distinct patterns: interstitial, reticulonodular, ground-glass, or honeycomb. Although chest X-rays are helpful in identifying the presence of an abnormality, they don't correlate well with histologic findings or pulmonary function tests (PFTs) in determining the severity of the disease. They also don't help distinguish inflammation from fibrosis. However, serial X-rays may help track the progression of the disease.

High-resolution computed tomography scans provide superior views of the four patterns seen on routine X-ray film and are used routinely to help establish the diagnosis of IPF. Research is currently underway to determine whether the four patterns of abnormality seen on these scans correlate with responsiveness to treatment.

PFTs show reductions in vital capacity and total lung capacity and impaired diffusing capacity for carbon monoxide. Arterial blood gas (ABG) analysis and pulse oximetry reveal hypoxemia, which may be mild when the patient is at rest early in the disease but may become severe later in the disease. Oxygenation will always deteriorate, usually to a severe level, with

exertion. Serial PFTs (especially carbon monoxide diffusing capacity) and ABG values may help track the course of the disease and the patient's response to treatment.

Treatment

Although it can't change the pathology of IPF, oxygen therapy can prevent the problems related to dyspnea and tissue hypoxia in the early stages of the disease process.

The patient may require little or no supplemental oxygen while at rest initially, but he'll need more as the disease progresses and during exertion.

No known cure exists. Corticosteroids and cytotoxic drugs may be given to suppress inflammation but are usually unsuccessful. Recently, interferon-gamma-1B has shown some promise in treating the disease.

Lung transplantation may be successful for younger, otherwise healthy individuals.

Special considerations

- Explain all diagnostic tests to the patient, who may experience anxiety and frustration about the many tests required to establish the diagnosis.
- Monitor oxygenation at rest and with exertion. The physician may prescribe one oxygen flow rate for use when the patient is at rest and a higher one for use during exertion to maintain adequate oxygenation. Instruct the patient to increase his oxygen flow rate to the appropriate level for exercise.
- As IPF progresses, the patient's oxygen requirements will increase. He may need a nonrebreathing mask to supply high oxygen percentages. Eventually, maintaining adequate oxygenation may become impossible despite maximum oxygen flow.
- Most patients will need oxygen at home. Make appropriate referrals to discharge planners, respiratory care practitioners, and home equipment vendors to ensure continuity of care.
- Teach breathing, relaxation, and energy conservation techniques to help the patient manage severe dyspnea.

- Encourage the patient to be as active as possible. Refer him to a pulmonary rehabilitation program.
- Monitor the patient for adverse reactions to drug therapy.
- Teach the patient about prescribed medications, especially adverse effects. Teach the patient and his family members infection prevention techniques.
- Encourage good nutritional habits. Small, frequent meals with high nutritional value may be necessary if dyspnea interferes with eating.
- Provide emotional support for the patient and his family as they deal with the patient's increasing disability, dyspnea, and probable death.

Tuberculosis

An acute or chronic infection caused by *Mycobacterium tuberculosis,* tuberculosis (TB) is characterized by pulmonary infiltrates, formation of granulomas with caseation, fibrosis, and cavitation. People who live in crowded, poorly ventilated conditions and those who are immunocompromised are most likely to become infected. In patients with strains that are sensitive to the usual antitubercular agents, the prognosis is excellent with correct treatment. However, in those with strains that are resistant to two or more of the major antitubercular agents, mortality is 50%.

Causes and incidence

After exposure to *M. tuberculosis*, roughly 5% of infected people develop active TB within 1 year; in the remainder, microorganisms cause a latent infection. The host's immune system usually controls the tubercle bacillus by enclosing it in a tiny nodule (tubercle). The bacillus may lie dormant within the tubercle for years and later reactivate and spread.

Although the primary infection site is the lungs, mycobacteria commonly exist in other parts of the body. Several factors increase the risk of infection reactivation: gastrectomy, uncontrolled diabetes mellitus, Hodgkin's lymphoma, leukemia, silicosis, acquired immunodeficiency syndrome, treatment with corticosteroids or immunosuppressants, and advanced age.

Transmission is by droplet nuclei produced when infected persons cough or sneeze. Persons with a cavitary lesion are particularly infectious because

their sputum usually contains 1 to 100 million bacilli per milliliter. If an inhaled tubercle bacillus settles in an alveolus, infection occurs, with alveolocapillary dilation and endothelial cell swelling. Alveolitis results, with replication of tubercle bacilli and influx of polymorphonuclear leukocytes. These organisms spread through the

lymph system to the circulatory system and then through the body.

Cell-mediated immunity to the mycobacteria, which develops 3 to 6 weeks later, usually contains the infection and arrests the disease. If the infection reactivates, the body's response characteristically leads to caseation—the conversion of necrotic tissue to a cheeselike material. The caseum may localize, undergo fibrosis, or excavate and form cavities, the walls of which are studded with multiplying tubercle bacilli. If this happens, infected caseous debris may spread throughout the lungs by the tracheobronchial tree. Sites of extrapulmonary TB include the pleurae, meninges, joints, lymph nodes, peritoneum, genitourinary tract, and bowel.

The incidence of TB has been increasing in the United States secondary to homelessness, drug abuse, and human immunodeficiency virus infection. Globally, TB is the leading infectious cause of morbidity and mortality, generating 8 to 10 million new cases each year.

Complications

- Respiratory failure
- Bronchopleural fistulas
- Pneumothorax
- Hemorrhage
- Pleural effusion
- Pneumonia

Signs and symptoms

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.

Diagnosis

CONFIRMING DIAGNOSIS

Diagnostic tests include chest X-rays, a tuberculin skin test, and sputum smears and cultures to identify M. tuberculosis. *The diagnosis must be precise because several other diseases (such as lung cancer, lung abscess, pneumoconiosis, and bronchiectasis) may mimic TB.*

These procedures aid in diagnosis:

- Auscultation detects crepitant crackles, bronchial breath sounds, wheezing, and whispered pectoriloquy.
- Chest percussion detects dullness over the affected area, indicating consolidation or pleural fluid.
- Chest X-ray shows nodular lesions, patchy infiltrates (mainly in upper lobes), cavity formation, scar tissue, and calcium deposits; however, it may not be able to distinguish active from inactive TB.
- Tuberculin skin test detects TB infection. Intermediate-strength purified protein derivative or 5 tuberculin units (0.1 ml) are injected intracutaneously on the forearm. The test results are read in 48 to 72 hours; a positive reaction (induration of 5 to 15 mm or more, depending on risk factors) develops 2 to 10 weeks after infection in active and inactive TB. However, severely immunosuppressed patients may never develop a positive reaction.

CONFIRMING DIAGNOSIS

Stains and cultures (of sputum, cerebrospinal fluid, urine, drainage from abscess, or pleural fluid) show heat-sensitive, nonmotile, aerobic, acid-fast bacilli.

Treatment

First-line agents for the treatment of TB are isoniazid (INH), rifampin (RIF), ethambutol (EMB), and pyrazinamide. Latent TB is usually treated with daily INH for 9 months. RIF daily for 4 months may be used for people with latent TB whose contacts are INH resistant. For most adults with active TB, the recommended dosing includes the administration of all four drugs daily for 2 months, followed by 4 months of INH and RIF. Drug therapy must be selected according to patient condition and organism susceptibility. Another first-line drug used for TB is rifapentine. Second-line agents, such as cycloserine,

ethionamide, p-Aminosalicylic acid, streptomycin, and capreomycin, are reserved for special circumstances or drug-resistant strains. Interruption of drug therapy may require initiation of therapy from the beginning of the regimen or additional treatment.

PREVENTION

PREVENTING TUBERCULOSIS

The best way to prevent tuberculosis (TB) is early detection to prevent it from becoming active. Hospitalized patients with TB should be isolated from other patients using airborne precautions. Staff members should also use disposable HEPA filter masks which serve as adequate respiratory protection when caring for patients who are in airborne isolation.

Other ways to prevent the spread of TB include:

- If a patient has a weakened immune system or has human immunodeficiency virus, it is recommended that he receive annual TB testing. Annual testing is also recommended for health care workers, those who work in a prison or a long-term care facility, and those with a substantially increased risk of exposure to the disease.
- If a patient tests positive for latent TB infection but has no evidence of active TB, he may be able to reduce his

risk of developing active TB by taking a course of therapy with isoniazid.

To prevent the spread of disease from those with active TB or from those who are receiving treatment, the following recommendations should be followed:

- Stress the need to maintain the treatment regimen and to not stop or skip doses. When the treatment regimen is stopped, the TB bacteria can mutate and become drug resistant.
- The patient who is on a treatment regimen is still contagious until he has been taking the medications for two to three weeks. Encouraging the patient to stay indoors and home from school or work is recommended. If he must leave his home, a mask is recommended during this initial treatment time to lessen the risk of transmission.

Directly observed therapy (DOT) may be selected or required. In this therapy, an assigned caregiver directly observes the administration of the drug. The goal of DOT is to monitor the treatment regimen and reduce the development of resistant organisms.

Special considerations

- Initiate acid-fast bacillus (AFB) isolation precautions immediately for all patients suspected or confirmed to have TB. AFB isolation precautions include the use of a private room with negative pressure in relation to surrounding areas and a minimum of six air exchanges per hour (air exhausted should be exhausted directly to the outside).
- Continue AFB isolation until there's clinical evidence of reduced infectiousness (substantially decreased cough, fewer organisms on sequential sputum smears).
- Teach the infectious patient to cough and sneeze into tissues and to dispose of all secretions properly. Place a covered trash can nearby or tape a lined bag to the side of the bed to dispose of used tissues.
- Instruct the patient to wear a mask when outside his room.

- Visitors and staff members should wear particulate respirators that fit closely around the face when they're in the patient's room.
- Remind the patient to get plenty of rest. Stress the importance of eating balanced meals to promote recovery. If the patient is anorexic, urge him to eat small meals frequently. Record weight weekly.

- Be alert for adverse effects of medications. Because INH sometimes leads to hepatitis or peripheral neuritis, monitor aspartate aminotransferase and alanine aminotransferase levels. To prevent or treat peripheral neuritis, give pyridoxine (vitamin B6), as ordered. If the patient receives EMB, watch for optic neuritis; if it develops, discontinue the drug. If he receives RIF, watch for hepatitis and purpura. Observe the patient for other complications such as hemoptysis.
- Before discharge, advise the patient to watch for adverse effects from the medication and report them immediately. Emphasize the importance of regular follow-up examinations. Instruct the patient and his family concerning the signs and symptoms of recurring TB. Stress the need to follow long-term treatment faithfully.
- Emphasize to the patient the importance of taking the medications daily as prescribed. He may enroll in a supervised administration program to avoid the development of drug-resistant organisms. (See *Preventing tuberculosis*.)

PNEUMOCONIOSES

Silicosis

Silicosis is a progressive disease characterized by nodular lesions that commonly progress to fibrosis. The most common form of pneumoconiosis, silicosis can be classified according to the severity of pulmonary disease and the rapidity of its onset and progression. It usually occurs as a simple asymptomatic illness.

Acute silicosis develops after 1 to 3 years in workers exposed to very high concentrations of respirable silica (sand blasters and tunnel workers). *Accelerated silicosis* appears after an average of 10 years of exposure to lower concentrations of free silica. *Chronic silicosis* develops after 20 or more years of exposure to lower concentrations of free silica. (Chronic silicosis is further subdivided into simple and complicated forms.)

The prognosis is good unless the disease progresses into the complicated fibrotic form, which causes respiratory insufficiency and cor pulmonale. It's also associated with pulmonary tuberculosis (TB).

Causes and incidence

Silicosis results from the inhalation and pulmonary deposition of respirable crystalline silica dust, mostly from quartz. The danger to the worker depends on the concentration of dust in the atmosphere, the percentage of respirable free silica particles in the dust, and the duration of exposure. Respirable particles are less than 10 microns in diameter, but the disease-causing particles deposited in the alveolar space are usually 1 to 3 microns in diameter.

Industrial sources of silica in its pure form include the manufacture of ceramics (flint) and building materials (sandstone). It occurs in mixed form in the production of construction materials (cement). It's found in powder form (silica flour) in paints, porcelain, scouring soaps, and wood fillers as well as in the mining of gold, coal, lead, zinc, and iron. Foundry workers, boiler scalers, and stonecutters are all exposed to silica dust and, therefore, are at high risk for developing silicosis.

Nodules result when alveolar macrophages ingest silica particles, which they're unable to process. As a result, the macrophages die and release proteolytic enzymes into the surrounding tissue. The subsequent inflammation attracts other macrophages and fibroblasts into the region to produce fibrous tissue and wall off the reaction. The resulting nodule has an onionskin appearance when viewed under a microscope. Nodules develop adjacent to terminal and respiratory bronchioles, concentrate in the upper lobes, and are commonly accompanied by bullous changes in both lobes. If the disease process doesn't progress, minimal physiologic disturbances and no disability occur. Occasionally, however, the fibrotic response accelerates, engulfing and destroying large areas of the lung (progressive massive fibrosis or conglomerate lesions). Fibrosis may continue even after exposure to dust has ended.

The incidence of silicosis has decreased since the Occupational Safety and Health

Administration instituted regulations requiring the use of protective equipment that limits the amount of silica dust inhaled.

Complications

- Pulmonary fibrosis
- Cor pulmonale
- Ventricular or respiratory failure
- Pulmonary tuberculosis

Signs and symptoms

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.

Diagnosis

The patient history reveals occupational exposure to silica dust. The physical examination is normal in simple silicosis; in chronic silicosis with conglomerate lesions, it may reveal decreased chest expansion, diminished intensity of breath sounds, areas of hyporesonance and hyperresonance, fine to medium crackles, and tachypnea.

In simple silicosis, chest X-rays show small, discrete, nodular lesions distributed throughout both lung fields but typically concentrated in the upper lung zones; the hilar lung nodes may be enlarged and exhibit

“eggshell” calcification. In complicated silicosis, X-rays show one or more conglomerate masses of dense tissue.

Pulmonary function tests show:

- Forced vital capacity (FVC)—reduced in complicated silicosis
- Forced expiratory volume in 1 second (FEV_1)—reduced in obstructive disease (emphysematous areas of silicosis); reduced in complicated silicosis, but ratio of FEV_1 to FVC is normal or high
- Maximal voluntary ventilation—reduced in restrictive and obstructive diseases
- Carbon dioxide diffusing capacity—reduced when fibrosis destroys alveolar walls and obliterates pulmonary capillaries or when fibrosis thickens the alveolocapillary membrane.

Treatment

The goal of treatment is to relieve respiratory symptoms, to manage hypoxemia and cor pulmonale, and to prevent respiratory tract irritation and infections. Treatment also includes careful observation for the development of TB. Respiratory symptoms may be relieved through daily use of inhaled bronchodilators and increased fluid intake (at least 3 qt [3 L] daily). Steam inhalation and chest physiotherapy techniques, such as controlled coughing and segmental bronchial drainage with chest percussion and vibration, help clear secretions. In severe cases, it may be necessary to administer oxygen by cannula or mask (1 to 2 L/minute) for the patient with chronic hypoxemia or by mechanical ventilation if arterial oxygen can't be maintained above 40 mm Hg. Respiratory infections require prompt administration of antibiotics.

Special considerations

- Teach the patient to prevent infections by avoiding crowds and persons with respiratory infections and by receiving influenza and pneumococcal vaccines.
- Increase exercise tolerance by encouraging regular activity. Advise the patient to plan his daily activities to decrease the work of breathing. The patient should be instructed to pace himself, rest often, and generally move slowly through his daily routine.

Asbestosis

Asbestosis is a form of pneumoconiosis characterized by diffuse interstitial fibrosis. It can develop as long as 15 to 20 years after regular exposure to asbestos has ended. Asbestos also causes pleural plaques and mesotheliomas of pleura and the peritoneum. A potent co-carcinogen, asbestos increases the risk of lung cancer in cigarette smokers.

Causes and incidence

Asbestosis results from the inhalation of respirable asbestos fibers (50 microns or more in length and 0.5 microns or less in diameter), which assume a longitudinal orientation in the airway and move in the direction of airflow. The fibers penetrate respiratory bronchioles and alveolar walls. Sources include the mining and milling of asbestos, the construction industry, and the fireproofing and textile industries. Asbestos was also used in the production of paints, plastics, and brake and clutch linings.

Asbestos-related diseases develop in families of asbestos workers as a result of exposure to fibrous dust shaken off workers' clothing at home. Such diseases develop in the general public as a result of exposure to fibrous dust or waste piles from nearby asbestos plants, but exposures for occupants of typical buildings are quite low and not in a range associated with asbestosis.

Inhaled fibers become encased in a brown, proteinlike sheath rich in iron (ferruginous bodies or asbestos bodies), found in sputum and lung tissue. Interstitial fibrosis develops in lower lung zones, causing obliterative changes in lung parenchyma and pleurae. Raised hyaline plaques may form in parietal pleura, diaphragm, and pleura contiguous with the pericardium.

Asbestosis occurs in 4 of every 10,000 people.

Complications

- Pulmonary fibrosis
- Respiratory failure
- Pulmonary hypertension
- Cor pulmonale

Signs and symptoms

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.

Diagnosis

The patient history reveals occupational, family, or neighborhood exposure to asbestos fibers. Physical examination reveals characteristic dry crackles at lung bases. Chest X-rays show fine, irregular, and linear diffuse infiltrates; extensive fibrosis results in a “honeycomb” or “ground-glass” appearance. X-rays may also show pleural thickening and calcification, with bilateral obliteration of costophrenic angles. In later stages, an enlarged heart with a classic “shaggy” heart border may be evident. Computed tomography scan of the lungs also aids in diagnosis.

Pulmonary function tests show:

- Vital capacity, forced vita capacity, and total lung capacity—decreased
- Forced expiratory volume in 1 second—decreased or normal
- Carbon monoxide diffusing capacity—reduced when fibrosis destroys alveolar walls and thickens alveolocapillary membranes.

Arterial blood gas analysis reveals:

- Partial pressure of arterial oxygen—decreased
- Partial pressure of arterial carbon dioxide—low due to hyperventilation.

Treatment

The goal of treatment is to relieve respiratory symptoms and, in advanced disease, manage hypoxemia and cor pulmonale. Respiratory symptoms may

be relieved by chest physiotherapy techniques, such as controlled coughing and segmental bronchial drainage, chest percussion, and vibration.

Aerosol therapy, inhaled mucolytics, and increased fluid intake (at least 3 qt [3 L] daily) may also relieve symptoms.

Diuretics, cardiac glycosides, and salt restriction may be indicated for patients with cor pulmonale. Hypoxemia requires oxygen administration by cannula or mask (1 to 2 L/minute) or by mechanical ventilation if arterial oxygen can't be maintained above 40 mm Hg. Respiratory infections require prompt administration of antibiotics.

Special considerations

- Teach the patient to prevent infections by avoiding crowds and persons with infections and by receiving influenza and pneumococcal vaccines.
- Improve the patient's ventilatory efficiency by encouraging physical reconditioning, energy conservation in daily activities, and relaxation techniques.

Coal worker's pneumoconiosis

A progressive nodular pulmonary disease, coal worker's pneumoconiosis (CWP) occurs in two forms. Simple CWP is characterized by small lung opacities; in complicated CWP, also known as *progressive massive fibrosis,* masses of fibrous tissue occasionally develop in the patient's lungs. The risk of developing CWP (also known as black lung disease, coal miner's disease, miner's asthma, anthracosis, and anthracosilicosis) depends upon the duration of exposure to coal dust (usually 15 years or longer), intensity of exposure (dust count and particle size), location of the mine, silica content of the coal (anthracite coal has the highest silica content), and the worker's susceptibility.

The prognosis varies. Simple asymptomatic disease is self-limiting, although progression to complicated CWP is more likely if CWP begins after a relatively short period of exposure. Complicated CWP may be disabling, resulting in severe ventilatory failure and cor pulmonale.

Causes and incidence

CWP is caused by the inhalation and prolonged retention of respirable coal dust particles (less than 5 microns in diameter). Simple CWP results in the formation of macules (accumulations of macrophages laden with coal dust) around the terminal and respiratory bronchioles, surrounded by a halo of dilated alveoli. Macule formation leads to atrophy of supporting tissue, causing permanent dilation of small airways (focal emphysema).

Simple disease may progress to complicated CWP, involving one or both lungs. In this form of the disease, fibrous tissue masses enlarge and coalesce, causing gross distortion of pulmonary structures (destruction of vasculature alveoli and airways).

The incidence of CWP is highest among anthracite coal miners in the eastern United States.

Complications

- Pulmonary hypertension
- Pulmonary tuberculosis
- Cor pulmonale

Signs and symptoms

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.

Diagnosis

The patient history reveals exposure to coal dust. Physical examination shows barrel chest, hyperresonant lungs with areas of dullness, diminished breath sounds, crackles, rhonchi, and wheezes. In *simple CWP,*

chest X-rays show small opacities (less than 10 mm in diameter). These may be present in all lung zones but are more prominent in the upper lung zones. In *complicated CWP,* one or more large opacities (1 to 5 cm in diameter), possibly exhibiting cavitation, are seen.

Pulmonary function tests show:

- Vital capacity—normal in simple CWP but decreased with complicated CWP
- Forced expiratory volume in 1 second—decreased in complicated disease
- Residual volume and total lung capacity—normal in simple CWP; decreased in complicated CWP
- Carbon monoxide diffusing capacity—significantly decreased in complicated CWP as alveolar septae are destroyed and pulmonary capillaries obliterated
- Partial pressure of arterial carbon dioxide—may be increased with concomitant chronic obstructive pulmonary disease.

Treatment

There's no specific treatment. The goal of treatment is to relieve respiratory symptoms, manage hypoxia and cor pulmonale, and avoid respiratory tract irritants and infections. Treatment also includes careful observation for the development of TB. Chest physiotherapy techniques, such as controlled coughing and segmental bronchial drainage combined with chest percussion and vibration, help remove secretions.

Other measures include increased fluid intake (at least 3 qt [3 L] daily) and respiratory therapy techniques, such as aerosol therapy, inhaled mucolytics, and intermittent positive pressure breathing. Diuretics, cardiac glycosides, and salt restriction may be indicated in cor pulmonale. In severe cases, it may be necessary to administer oxygen for hypoxemia by cannula or mask (1 to 2 L/minute) if the patient has chronic hypoxia; mechanical ventilation is utilized if arterial oxygen can't be maintained above 40 mm Hg. Respiratory infections require prompt administration of antibiotics.

Special considerations