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Musculoskeletal Disorders

Introduction

A complex system of bones, muscles, ligaments, tendons, and other connective tissue, the musculoskeletal system gives the body its form and shape. It also protects vital organs, makes movement possible, stores calcium and other minerals, and provides sites for hematopoiesis. A fibrous layer called the *periosteum* covers all bones, except at joints, where they're covered by articular cartilage.

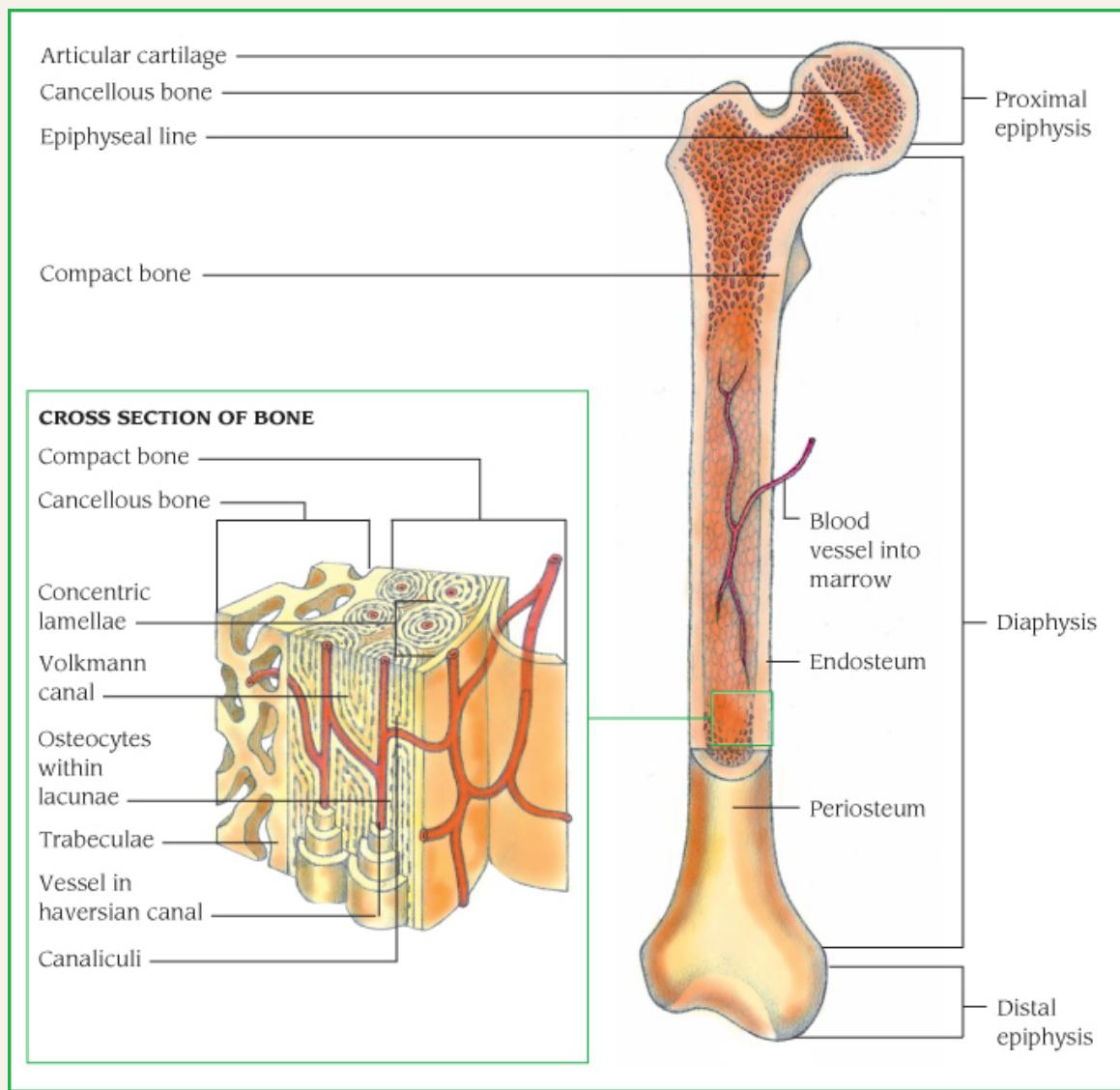
The human skeleton contains 206 bones, which are composed of inorganic salts, such as calcium and phosphate, embedded in a framework of collagen fibers. Bones are classified by shape as long, short, flat, or irregular.

LONG BONES

Long bones, which are found in the limbs, include the humerus, radius, and ulna of the arm; the femur, tibia, and fibula of the leg; and the phalanges, metacarpals, and metatarsals in the hands and feet. These bones have a long shaft, or *diaphysis*, and widened, bulbous ends, called *epiphyses*. A long bone is made up mainly of compact bone, which surrounds the medullary cavity (also called the *yellow marrow*), a storage site for fat. The lining of the medullary cavity (the *endosteum*) is a thin layer of connective tissue. The outer layer is the periosteum. (See *Long-bone structure*, page 288.)

Long-bone Structure

Long-bone composition is depicted below, with an illustrated cross section.



In children and young adults, lengthwise growth occurs at the epiphyseal cartilage between the diaphysis and epiphysis. In adults, in whom bone growth is complete, this cartilage is ossified and forms the epiphyseal line. The epiphysis also has a surface layer made up of compact bone, but its

center is made of spongy or cancellous bone. Cancellous bone contains open spaces between thin threads of bone, called *trabeculae*, which are arranged in various directions to correspond with the lines of maximum stress or pressure. This configuration gives the bone added structural strength.

Unlike cancellous bone, adult compact bone consists of numerous orderly networks of interconnecting canals that run parallel to the bone's long axis. Each of these networks, called a *haversian system*, consists of a central haversian canal surrounded by layers (*lamellae*) of bone. Between adjacent lamellae are small openings (*lacunae*), which contain bone cells (*osteocytes*). All lacunae are joined by an interconnecting network of tiny canals (*canaliculari*), each of which contains one or more capillaries and provides a route for movement of tissue fluids. The haversian system carries blood to the bone through blood vessels that enter the system through channels called *Volkmann's canals*.

SHORT, FLAT, OR IRREGULAR BONES

Short bones include the tarsal and carpal bones; flat bones, the frontal and parietal bones of the cranium, ribs, sternum, scapulae, ilium, and pubis; and irregular bones, the bones of the spine (vertebrae, sacrum, and coccyx) and certain bones of the skull (sphenoid, ethmoid, and mandible).

Short, flat, and irregular bones have an outer layer of compact bone and an inner portion of spongy bone. In the sternum and certain areas in the flat bones of the skull, the spongy bone contains red marrow.

JOINTS

The tissues connecting two bones make up a joint, which permits motion between the bones and provides stability. Joints, like bones, have varying forms.

- ◆ Fibrous joints (*synarthroses*) have only minute motion and provide stability when tight union is necessary, as in the seams, called *sutures*, that join the cranial bones.
- ◆ Cartilaginous joints (*amphiarthroses*) have limited motion, as between vertebrae and symphysis pubis.
- ◆ Synovial joints (*diarthroses*) are the most common and have the greatest degree of movement. Such joints include the elbows, shoulders, and

knees. Synovial joints have special characteristics: the articulating surfaces of each bone have a smooth hyaline covering (articular cartilage), which is resilient to pressure; their opposing surfaces are congruous and glide smoothly on each other without touching each other; a fibrous (articular) capsule holds them together. Beneath the capsule and lining the joint cavity, the synovial membrane secretes the clear, viscous synovial fluid. This fluid lubricates the two opposing surfaces during motion and also nourishes the articular cartilage. Surrounding a synovial joint are ligaments, muscles, and tendons, which strengthen and stabilize the joint but allow free movement.

In some synovial joints, the synovial membrane forms two additional structures—bursae and tendon sheaths—which reduce friction that normally accompanies movement. Bursae are small, cushionlike sacs lined with synovial membranes and filled with synovial fluid; most are located between tendons and bones. Tendon sheaths wrap around the tendon and cushion it as it crosses the joint.

The synovial joints permit angular and circular movements. Angular movements include *flexion* (decrease in joint angle), *extension* (increase in joint angle), and *hyperextension* (increase in the angle of extension beyond the usual arc). Joints of the knees, elbows, and phalanges permit such movement. Other angular movements are *abduction* (movement away from the body's midline) and *adduction* (movement toward the body's midline).

Circular movements include *rotation* (motion around a central axis), as in the ball-and-socket joints of the hips and shoulders; *pronation* (wrist motion to place palmar surface of the hand down, with the thumb toward the body); *supination* (begging position, with palm up). Other kinds of movement are *inversion* (movement facing inward), *eversion* (movement facing outward), *protraction* (as in forward motion of the mandible), and *retraction* (returning protracted part into place).

MUSCLES

Muscle tissues' most specialized feature—*contractility*—makes movement of bones and joints possible. Muscles also pump blood through the body, move food through the intestines, and make breathing possible. Muscular activity produces heat, so it's an important component in temperature regulation. Muscles maintain body positions, such as sitting and standing.

Muscle mass accounts for about 40% of the body weight of a person of average size.

Muscles are classified in many ways. *Skeletal* muscles are attached to bone, *visceral* muscles permit function of internal organs, and *cardiac* muscles make up the heart wall. Also, muscles may be striated or nonstriated (smooth), depending on their cellular configuration.

Muscles classified according to activity are called *voluntary* or *involuntary*. Voluntary muscles can be controlled at will and are under the influence of the somatic nervous system; these are the skeletal muscles. Involuntary muscles, controlled by the autonomic nervous system, include the cardiac and visceral muscles.

Each skeletal muscle consists of many elongated muscle cells, called *muscle fibers*, through which run slender threads of protein, called *myofibrils*. Muscle fibers are held together in bundles by sheaths of fibrous tissue, called *fascia*. Blood vessels and nerves pass through the fascia to reach the individual muscle fibers.

Skeletal muscles are attached to bone directly or indirectly by fibrous cords called *tendons*. The least movable end of the muscle attachment is called *the point of origin*; the most movable end is *the point of insertion*.

MECHANISM OF CONTRACTION

To stimulate muscle contraction and movement, the brain sends motor impulses through the peripheral motor nerves to motor nerve fibers in the voluntary muscle. These nerve fibers reach membranes of skeletal muscle cells at neuromuscular (*myoneural*) junctions. When an impulse reaches the myoneural junction, it triggers the following sequence: release of the neurochemical acetylcholine, transient release of calcium from the sarcoplasmic reticulum (a membranous network in the muscle fiber), and muscle contraction. The arriving impulse at the myoneural junction also triggers release of adenosine triphosphate, the energy source for muscle contraction. Muscle relaxation is believed to take place by reversal of the above mechanisms.

MUSCULOSKELETAL ASSESSMENT

Most patients with musculoskeletal disorders are elderly, have concurrent medical conditions, or have experienced trauma. Younger patients tend to experience more benign, self-limited conditions. Generally, they face

prolonged immobilization. These factors make thorough assessment essential. Your assessment should include a complete history and a careful physical examination to determine a possible cause of the symptoms.

Interview the patient carefully to obtain a complete medical, social, and personal history. Ask about general activity, for example jogging daily or sedentary life which may be significantly altered by musculoskeletal disease or trauma. Does the patient have any systemic symptoms, such as fever, chills, weight loss, or skin rashes? Obtain information about occupation, diet, sexual activity, and elimination habits, drugs taken, and use of safety devices, and try to assess how the problem will affect body image. Also, ask how the patient functions at home. Is the patient independent with activities of daily living, are there stairs to the bedroom or bathroom, any prosthetic devices, any family members who help with personal care?

Get an accurate account of the musculoskeletal problem. Ask if it has caused any changes to everyday routines. When did symptoms begin and how did they progress? Has the patient received treatment for this problem? Has there been any trauma? If so, find out the details.

Assess the level of pain. Is the patient in pain now? Ask what makes the discomfort worse or better (movement, position, and so forth). Evaluate past and present responses to treatment. For instance, if the patient has arthritis and uses corticosteroids, ask about their effectiveness. Is more or less medication needed than before? Are there any issues with adherence to the prescribed treatment?

Physical examination helps to determine the diagnosis and reveals any existing disabilities. (These baseline data will help when the effects of treatment are evaluated.) Observe the patient's appearance. Look for localized edema, pigmentation, redness and tenderness at pressure points, and other deformities such as atrophy. Note mobility, strength, and gait. To check range of motion (ROM), ask the patient to abduct, adduct, or flex the muscles in question. Obtain height, weight, and vital signs. Check neurovascular status, including motion sensation and circulation. Measure and record discrepancies in muscle circumference or leg length. Compare one side or limb to the other. If a neck injury is suspected, don't force ROM.

DIAGNOSTIC TOOLS

- ◆ X-rays are a useful diagnostic tool to evaluate musculoskeletal diseases. They can help to identify joint disruption, bone deformities, calcifications, and bone destruction and fractures. X-rays also measure bone density.
- ◆ Myelography is an invasive procedure used to evaluate abnormalities of the spinal canal and cord. It entails injection of a radiopaque contrast medium into the subarachnoid space of the spine. Serial X-rays visualize the progress of the contrast medium as it passes through the subarachnoid space. Displacement of the medium indicates a space-occupying lesion, such as a herniated disk or a tumor.
- ◆ Magnetic resonance imaging (MRI) is useful in evaluating soft-tissue injuries or ligament tears, such as rotator cuff tears or meniscal tears.
- ◆ Computed tomography (CT) scan can be used to identify injuries to bones, soft tissue, ligaments, tendons, and muscles.
- ◆ Arthroscopy is the visual examination of the interior of a joint with a fiberoptic endoscope.

Other useful tests include bone and muscle biopsies, electromyography, microscopic examination of synovial fluid, and multiple laboratory studies of urine and blood to identify systemic abnormalities.

PATIENT CARE

Each patient with musculoskeletal disease needs an individual care plan formulated early in the hospital stay by the entire clinical team, including the physician, physical therapist, and occupational therapist. Develop this plan with short- and long-term goals, during and after hospitalization.

Caring for the patient with a musculoskeletal disease usually includes at least one of the following: traction, casts, braces, splints, crutches, intermittent ROM devices, prolonged immobilization, physical therapy, occupational therapy, and self-care measures; adequate vitamin D intake, weight loss, dietary modifications, and drugs.

Traction is the manual or mechanical application of a steady pulling force to reduce a fracture, minimize muscle spasms, or immobilize or align a joint.

- ◆ Skin traction is the indirect application of traction to the skeletal system through skin and soft tissues.

- ◆ Skeletal traction is the direct application of traction to bones by means of a pin (Steinmann pin) or wire (Kirschner wire) through the affected bone or by calipers or a tonglike device (Gardner-Wells tongs) that grips the bone.
- ◆ Manual traction, for emergency use, is the direct application of traction to a body part by hand.

During the use of all types of traction:

- ◆ Explain to the patient how traction works and advise the patient about permissible amounts of activity and elevation of the head of the bed. Inform the patient of the anticipated duration of traction and whether the traction is removable. Teach active ROM exercises.
- ◆ Check neurovascular status to prevent nerve damage. Also, make sure the mattress is firm, that the traction ropes aren't frayed, that they're on the center track of the pulley, and that traction weights are hanging free. Thoroughly investigate any complaint the patient makes.
- ◆ Check for signs of infection (odor, local inflammation and drainage, or fever) at pin sites if the patient is in skeletal traction. Also, check with the physician's or the facility's procedure regarding pin-site care, such as use of peroxide or povidone-iodine.

Ideally, a cast immobilizes without adding too much weight. It's snug-fitting but doesn't constrict and has a smooth inner surface and smooth edges to prevent pressure or skin irritation. Casts require comprehensive patient education.

- ◆ A plaster cast takes 24 to 48 hours to dry. To prevent indentations, tell the patient not to squeeze the cast, not to cover or walk on the cast until it has dried, and not to bump a damp cast on hard surfaces because dents can cause pressure areas. Warn the patient that while the cast is drying, there may be a temporary sensation of heat under the cast.
- ◆ If fiberglass is used, the cast may feel dry and the patient may be able to bear weight immediately. Advise the patient, however, not to get the cast wet. Although the fiberglass won't disintegrate as plaster would, the padding will become wet and potentially cause maceration of the skin.
- ◆ Emphasize the need to keep the cast above heart level for *24 hours* after its application to reduce swelling in the limb.

- ◆ While the cast is drying and after drying is complete, the patient should watch for and immediately report persistent pain in the limb inside or distal to the cast as well as edema, changes in skin color, coldness, or tingling or numbness in this area. If any of these signs occur, tell the patient to position the casted body part above heart level and notify the physician.
- ◆ The patient should also report drainage through the cast or an odor that may indicate infection. Warn against inserting foreign objects under the cast, getting it wet, pulling out its padding, or scratching inside it. Tell the patient to seek immediate attention for a broken cast.
- ◆ Instruct the patient to exercise the joints above and below the cast to prevent stiffness and contracture.

Braces, splints, and slings also provide alignment, immobilization, and pain relief for musculoskeletal diseases. Slings and splints are usually used for short-term immobilization. Explain to the patient and the family why these appliances are necessary and show them the proper way to apply the sling, splint, or brace for optimal benefit. Tell the patient how long the appliance will have to be worn and advise the patient of any activity limitations that must be observed. If the patient has a brace, check with the orthotist (orthopedic appliance specialist) about proper care. Encourage the patient to refer additional questions to the physician. Teach proper crutch walking.

COPING WITH IMMOBILITY

Immobilized patients require meticulous care to prevent complications. Without constant care, the bedridden patient becomes susceptible to pressure ulcers, caused by the increased pressure on tissue over bony prominences, and is especially vulnerable to cardiopulmonary complications.

- ◆ To prevent pressure ulcers, turn the patient every 2 hours and, if possible, reposition in a 30-degree side-lying position for short periods. In addition, place a flotation pad or sheepskin pad under bony prominences, or use an alternating-air-current, convoluted foam, or foam mattress. Show the patient how to use a Balkan frame with a trapeze to move about in bed.
- ◆ Keep the patient's skin dry and clean.

- ◆ Keep the sheets wrinkle-free.
- ◆ Increase fluid intake to minimize risk of renal calculi.
- ◆ Provide adequate nutrition; a high-protein diet is preferred, if tolerated.
- ◆ Perform passive ROM exercises on the affected side, as ordered, to prevent contractures, and instruct the patient in active ROM exercises on the unaffected side. Apply footboards or high-topped sneakers to prevent footdrop. Keep the patient's heels off the bed to prevent heel breakdown. Also, watch for reddened elbows.
- ◆ Because most bedridden patients involuntarily perform a Valsalva's maneuver when using the upper arms and trunk to move, instruct the patient to exhale (instead of holding their breath) while turning. This will prevent possible cardiac complications that result from increased intrathoracic pressure.
- ◆ Emphasize the importance of coughing and deep breathing and teach the patient how to use the incentive spirometer if ordered.
- ◆ Because constipation is a common problem in bedridden patients, establish a bowel program (fluids, fiber, laxatives, stool softeners), as needed.

REHABILITATION

Restoring the patient to a former state of health isn't always possible. When it isn't, help the patient adjust to a modified lifestyle. During hospitalization, promote independence by letting patients finish difficult tasks independently. If necessary, refer the patient to a community facility for continued rehabilitation.

Congenital Disorders

CLUBFOOT

Clubfoot, or *talipes*, is the most common congenital disorder of the lower limbs. It's marked primarily by a deformed talus and shortened Achilles tendon, which give the foot a characteristic clublike appearance. In talipes equinovarus, the foot points downward (equinus) and turns inward (varus), whereas the front of the foot curls toward the heel (forefoot adduction).

Causes and Incidence

It is no longer believed that clubfoot is caused by fetal position in utero. Heredity is a definite factor in some cases, although the mechanism of transmission is undetermined. In children without a family history of clubfoot, this anomaly seems linked to arrested development during the 9th and 10th weeks of embryonic life, when the feet are formed. Researchers also suspect muscle abnormalities, leading to variations in length and tendon insertions, as possible causes of clubfoot. Environmental factors play a role. Studies strongly link clubfoot to cigarette smoking during pregnancy, especially if there is a family history of clubfoot.

Clubfoot, which has an incidence of about 1 per 1,000 live births, usually occurs bilaterally and is twice as common in boys. It may be associated with other birth defects, such as myelomeningocele, spina bifida, and arthrogryposis. However, most cases are sporadic occurrences.

Pathophysiology

Abnormal development of the foot during fetal growth leads to abnormal muscles and joints and contracture of soft tissue. Clubfoot can also occur because of paralysis, poliomyelitis, or cerebral palsy. The condition called *apparent clubfoot* results when a fetus maintains a position in utero that gives feet a clubfoot appearance at birth; it can usually be corrected manually. Another form of apparent clubfoot is inversion of the feet, resulting from the denervation type of progressive muscular atrophy and progressive muscular dystrophy.

Complication

- ◆ Retention deformity

Signs and Symptoms

Talipes equinovarus varies greatly in severity. Deformity may be so extreme that the toes touch the inside of the ankle, or it may be only vaguely apparent. In every case, the talus is deformed, the Achilles tendon shortened, and the calcaneus somewhat shortened and flattened. Depending on the degree of the varus deformity, the calf muscles are shortened and underdeveloped, and soft-tissue contractures form at the site of the deformity. The foot is tight in its deformed position and resists manual efforts to push it into normal position. Clubfoot is painless, except in elderly, arthritic patients. In older children, clubfoot may be secondary to

paralysis, poliomyelitis, or cerebral palsy, in which case treatment must include management of the underlying disease.

Diagnosis

Early diagnosis of clubfoot is usually possible because the deformity is obvious. In subtle deformity, however, true clubfoot must be distinguished from apparent clubfoot (metatarsus varus or pigeon toe). Apparent clubfoot is inversion of the feet, resulting from the peroneal type of progressive muscular atrophy and progressive muscular dystrophy. In true clubfoot, X-rays show superimposition of the talus and the calcaneus and a ladderlike appearance of the metatarsals. (See *Recognizing clubfoot*.)

Recognizing Clubfoot

Congenital skeletal anomalies frequently involve the foot and the ankle and are referred to as “clubfoot.”



Treatment

Clubfoot is correctable with prompt treatment, which is performed in three stages: correcting the deformity, maintaining the correction until the foot regains normal muscle balance, and observing the foot closely for several years to prevent the deformity from recurring. In neonates with true clubfoot, corrective treatment should begin at once. An infant's foot contains large amounts of cartilage; the muscles, ligaments, and tendons are

supple. The ideal time to begin treatment is during the first few days and weeks of life, when the foot is most malleable.

Clubfoot deformities are usually corrected in sequential order. Several therapeutic methods have been found effective in correcting clubfoot. In all patients, the first procedure should be simple manipulation and casting, whereby the foot is gently manipulated into a partially corrected position and held in place by a cast for several days or weeks. (The skin should be painted with a nonirritating adhesive liquid beforehand to prevent the cast from slipping.) After the cast is removed, the foot is manipulated into an even better position and casted again. This procedure is repeated as many times as necessary. In some cases, the shape of the cast can be transformed through a series of wedging maneuvers instead of changing the cast each time.

After correction of clubfoot, proper foot alignment should be maintained through exercise, night splints, and orthopedic shoes. With manipulating and casting, correction usually takes about 3 months. The Denis Browne splint, a device that consists of two padded, metal footplates connected by a flat, horizontal bar, is sometimes used as a follow-up measure to help promote bilateral correction and strengthen the foot muscles.

Resistant clubfoot may require surgery. Older children, for example, with recurrent or neglected clubfoot usually need surgery. Tenotomy, tendon transfer, stripping of the plantar fascia, and capsulotomy are some of the surgical procedures that may be used. In severe cases, bone surgery (wedge resections, osteotomy, or astragalectomy) may be appropriate. After surgery, a cast is applied to preserve the correction. Clubfoot severe enough to require surgery is rarely totally correctable; however, surgery can usually ameliorate the deformity.

Special Considerations

The primary concern is recognition of clubfoot as early as possible, preferably in neonates.

- ◆ Look for any exaggerated attitudes in an infant's feet. Make sure you recognize the difference between true clubfoot and apparent clubfoot. Don't use excessive force in trying to manipulate a clubfoot. The foot with apparent clubfoot moves easily.
- ◆ Stress to parents the importance of prompt treatment. Make sure they understand that clubfoot demands immediate therapy and orthopedic

supervision until growth is completed.

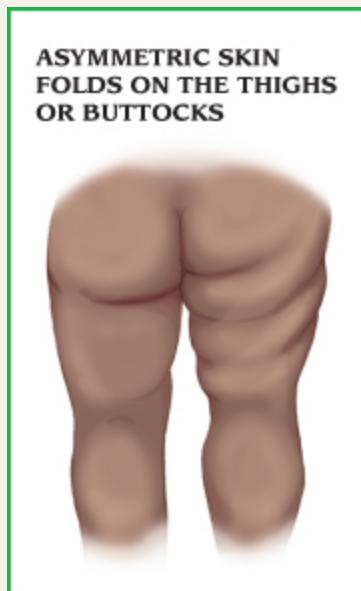
- ◆ After casting, elevate the child's feet with pillows. Check the toes every 1 to 2 hours for temperature, color, sensation, motion, and capillary refill time; watch for edema. Before a child in a clubfoot cast is discharged, teach parents to recognize circulatory impairment.
- ◆ Insert plastic petals over the top edges of a new cast while it's still wet to keep urine from soaking and softening the cast. This is done as follows: Cut a plastic sheet into strips long enough to cover the outside of the cast and tuck them about a finger length beneath the cast edges. Using overlapping strips of tape, tack the corner of each petal to the outside of the cast. When the cast is dry, petal the edges with adhesive tape to keep out plaster crumbs and prevent skin irritation. Perform good skin care under the cast edges every 4 hours, washing and carefully drying the skin. (Don't rub the skin with alcohol, and don't use oils or powders, which tend to macerate the skin.)
- ◆ If the child is old enough to walk, caution parents not to let the foot part of the cast get soft and thin from wear. If it does, much of the correction may be lost.
- ◆ When the wedging method of shaping the cast is being used, check circulatory status frequently; it may be impaired by increased pressure on tissues and blood vessels. The equinus (posterior release) correction especially places considerable strain on ligaments, blood vessels, and tendons.
- ◆ After surgery, elevate the child's feet with pillows to decrease swelling and pain. Report signs of discomfort or pain right away. Try to locate the source of pain; it may result from cast pressure rather than from the incision. If bleeding occurs under the cast, circle the location and mark the time on the cast. If bleeding spreads, report it.
- ◆ Explain to the older child and the parents that surgery can improve clubfoot with good function but can't totally correct it; the affected calf muscle will remain slightly underdeveloped.
- ◆ Emphasize the need for long-term orthopedic care to maintain correction. Teach parents the prescribed exercises that the child can do at home. Urge them to make the child wear the corrective shoes ordered and the splints during naps and at night. Make sure they understand that treatment for clubfoot continues during the entire growth period. Correcting this defect permanently takes time and patience.

DEVELOPMENTAL DYSPLASIA OF THE HIP

Developmental dysplasia of the hip (DDH), an abnormality of the hip joint present from birth, is the most common disorder affecting hip joints of children younger than 3 years old. DDH can be unilateral or bilateral. (See *Characteristics of developmental hip dysplasia*, page 294.) This abnormality occurs in three forms of varying severity: *unstable hip dysplasia*, in which the hip is positioned normally but can be dislocated by manipulation; *subluxation or incomplete dislocation*, in which the femoral head rides on the edge of the acetabulum; and *complete or true congenital dislocation*, in which the femoral head is totally outside the acetabulum.

Characteristics of Developmental Hip Dysplasia

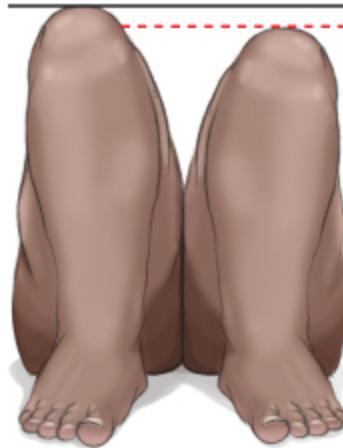
The classic characteristics of developmental hip dysplasia are illustrated below.



LIMITED ABDUCTION
OF THE HIP



LEG-LENGTH
DISCREPANCY



Developmental hip subluxation or dislocation can cause abnormal acetabular development and permanent disability.

Causes and Incidence

Experts are uncertain about the causes of DDH. Dislocation is 10 times more common after breech delivery (malpositioning in utero) than after cephalic delivery, and it's also more common among large neonates and

twins. It's a lot more common in firstborn children. Girls are affected more often than boys and white children more than black children. Genetic factors may also play a role.

Although DDH is found throughout the world, incidence is particularly high among Native Americans.

Pathophysiology

The precise cause of congenital dislocation is unknown. Excessive or abnormal movement of the joint during a traumatic birth may cause dislocation. Displacement of bones within the joint may damage joint structures, including articulating surfaces, blood vessels, tendons, ligaments, and nerves. This may lead to ischemic necrosis because of the disruption of blood flow to the joint.

Complications

- ◆ Degenerative hip changes (if treatment is delayed)
- ◆ Lordosis
- ◆ Joint malformation
- ◆ Crippling osteoarthritis

Signs and Symptoms

Clinical effects of hip dysplasia vary with age. In neonates, dysplasia doesn't cause gross deformity or pain. However, in complete dysplasia, the hip rides above the acetabulum, causing the level of the knees to be uneven. As the child grows older and begins to walk, the abduction on the dislocated side is limited. Uncorrected bilateral dysplasia may cause the child to sway from side to side, a condition known as "duck waddle"; unilateral dysplasia may produce a limp. If corrective treatment isn't begun until after age 2, DDH may cause degenerative hip changes, lordosis, joint malformation, and soft-tissue damage.

Diagnosis

Several observations during physical examination of the relaxed child strongly suggest DDH. First, place the child on the back, and inspect the folds of skin over the thighs. Usually, a child in this position has an equal number of thigh folds on each side, but a child with subluxation or

dislocation may have an extra fold on the affected side (this extra fold is also apparent when the child lies prone). Next, with the child lying prone, check for alignment of the buttock fold. In a child with dysplasia, the buttock fold on the affected side is higher. In addition, abduction of the affected hip is restricted.

Dx **CONFIRMING DIAGNOSIS** *A positive Ortolani or Trendelenburg sign confirms DDH. To elicit Ortolani sign, place the infant on back, with hip flexed and abducted. Adducting the hip while pressing the femur downward will dislocate the hip. Then, abducting the hip while moving the femur upward will move the femoral head over the acetabular rim. If you hear a click or feel a jerk as the femoral head moves, the test is positive. This sign indicates subluxation in a neonate younger than 1 month and subluxation or complete dislocation in an older infant.*

To elicit Trendelenburg sign, have the child put weight on the side of the dislocation and lift the other knee. The pelvis drops on the normal side because of weak abductor muscles in the affected hip. However, when the child stands with weight on the normal side and lifts the other knee, the pelvis remains horizontal.

Ultrasound of the hip reveals hip deformity. X-rays show the location of the femur head and a shallow acetabulum. X-rays may also show acetabular dysplasia or a teratological dislocation. MRI may also be used to assess reduction.

Treatment

The earlier the infant receives treatment, the better the chances are for normal development. Treatment varies with the patient's age and is tailored to the specific pathological condition. In infants younger than 6 months, treatment includes *gentle* manipulation to reduce the dislocation, followed by holding the hips in a flexed and abducted position with a splint-brace or harness to maintain the reduction. The infant must wear this apparatus continuously for 2 to 3 months and then use a night splint for another month, so the joint capsule can tighten and stabilize in correct alignment.

If treatment doesn't begin until after age 3 months, it may include bilateral skin traction (in infants) or skeletal traction (in children who have started walking) to reduce the dislocation by gradually abducting the hips.

In Bryant's traction, or divarication traction, both legs are placed in traction, even if only one is affected, to help maintain immobilization. This type of traction is used in children who are younger than 3 years and weigh less than 35 lb (16 kg). The length of treatment is 2 to 3 weeks.

If traction fails, gentle closed reduction under general anesthetic can further abduct the hips; the child is then placed in a spica cast for 4 to 6 months. If closed treatment fails, open reduction, followed by immobilization in a spica cast for an average of 6 months, or osteotomy may be considered.

In the child 2 to 5 years old, treatment is difficult and includes skeletal traction and subcutaneous adductor tenotomy. Treatment begun after age 5 rarely restores satisfactory hip function.

Special Considerations

The child who must wear a splint, brace, or body cast needs special personal care that requires parent education.

- ◆ Teach parents how to correctly splint or brace the hips, as ordered. Stress the need for frequent checkups.
- ◆ Listen sympathetically to the parents' expressions of anxiety and fear. Explain possible causes of DDH, and give reassurance that early, prompt treatment will probably result in complete correction.
- ◆ During the child's first few days in a cast or splint-brace, expect some irritability due to the unaccustomed restricted movement. Encourage the parents to stay with the child as much as possible and to calm to provide reassurance.
- ◆ Assure parents that the child will adjust to this restriction and return to normal sleeping, eating, and playing behavior in a few days.
- ◆ Instruct parents to remove braces and splints while bathing the infant but to replace them immediately afterward. Stress good hygiene; parents should bathe and change the child frequently and wash perineum with warm water and soap at each diaper change.

If treatment requires a spica cast:

- ◆ When transferring the child immediately after casting, use your palms to avoid making dents in the cast. Such dents predispose the patient to pressure sores. Remember that the plaster cast needs 24 to 48 hours to

dry naturally. Don't use heat to make it dry faster because heat also makes it more fragile.

- ◆ Immediately after the cast is applied, use a plastic sheet to protect it from moisture around the perineum and buttocks. Cut the sheet into strips long enough to cover the outside of the cast and tuck them about a finger length beneath the cast edges. Using overlapping strips of tape, tack the corner of each petal to the outside of the cast. Remove the plastic under the cast every 4 hours; then wash, dry, and retuck it. Disposable diapers folded lengthwise over the perineum may also be used.
- ◆ Position the child either on a Bradford frame elevated on blocks, with a bedpan under the frame, or on pillows to support the child's legs. Be sure to keep the cast dry and change the child's diapers often.
- ◆ Turn the child every 2 hours during the day and every 4 hours at night. Check color, sensation, and motion of the infant's legs and feet. Be sure to examine all toes. Notify the physician of dusky, cool, or numb toes.
- ◆ Check the cast daily for odors, which may herald infection.
- ◆ If the child complains of itching, he may benefit from diphenhydramine, or you may aim a hair dryer set on cool at the cast edges to relieve itching. Don't scratch or probe under the cast. Investigate any persistent itching.
- ◆ Provide adequate nutrition and maintain adequate fluid intake to avoid renal calculi and constipation, both complications of inactivity.
- ◆ Provide adequate stimuli to promote growth and development.
- ◆ Tell parents to watch for signs that the child is outgrowing the cast (cyanosis, cool limbs, or pain).
- ◆ Tell parents that treatment may be prolonged and requires patience.
- ◆ The patient in Bryant's traction may be cared for at home if the parents are taught traction application and maintenance.
- ◆ Encourage the parents to cuddle and hold the child and encourage interactions with siblings and friends.
- ◆ Maintain skin integrity and check circulation at least every 2 hours.
- ◆ Feed the child carefully to avoid aspiration and choking.
- ◆ Refer the child and parents to a child life specialist to ensure continued developmental progress.

MUSCULAR DYSTROPHY

Muscular dystrophy is a group of congenital disorders characterized by progressive symmetrical wasting of skeletal muscles without neural or sensory defects. Paradoxically, these wasted muscles tend to enlarge because of connective tissue and fat deposits, giving an erroneous impression of muscle strength. The main types of muscular dystrophy are Duchenne (pseudohypertrophic), Becker (benign pseudohypertrophic), facioscapulohumeral (Landouzy–Dejerine), limb-girdle dystrophy, Emery–Dreifuss muscular dystrophy, and myotonia congenita.

The prognosis varies. Duchenne muscular dystrophy generally strikes during early childhood and usually results in death in the 20s or early 30s. Patients with Becker muscular dystrophy typically live into their 40s. Facioscapulohumeral and limb-girdle dystrophies usually don't shorten life.

Causes and Incidence

Muscular dystrophy is caused by various genetic mechanisms. Duchenne and Becker muscular dystrophies are X-linked recessive disorders. Both result from defects in the gene coding for the muscle protein dystrophin; the gene has been mapped to the Xp21 locus.

The incidence of muscular dystrophy is about 1 in 651,450 persons in the United States. Duchenne and Becker muscular dystrophies affect males almost exclusively.

Facioscapulohumeral dystrophy is an autosomal dominant disorder. Limb-girdle dystrophy is usually autosomal recessive. These two types affect both sexes about equally.

Pathophysiology

Abnormally permeable cell membranes allow leakage of various muscle enzymes, particularly creatine kinase. This metabolic defect that causes the muscle cells to die is present from fetal life onward. The absence of progressive muscle wasting at birth suggests that other factors compound the effect of dystrophin deficiency. The specific trigger is unknown, but phagocytosis of the muscle cells by inflammatory cells causes scarring and loss of muscle function.

As the disease progresses, skeletal muscle becomes almost totally replaced by fat and connective tissue. The skeleton eventually becomes deformed, causing progressive immobility. Cardiac and smooth muscle of

the gastrointestinal (GI) tract typically become fibrotic. No consistent structural abnormalities are seen in the brain.

Complications

- ◆ Inhibited pulmonary function due to deformities
- ◆ Greater risk for pneumonia
- ◆ Respiratory problems lead to arrhythmias and hypertrophy

Signs and Symptoms

Although all four types of muscular dystrophy cause progressive muscular deterioration, the degree of severity and age of onset vary.

Duchenne muscular dystrophy begins insidiously, between ages 2 and 3. Initially, it affects leg and pelvic muscles but eventually spreads to the involuntary muscles. Muscle weakness produces a waddling gait, toe walking, and lordosis. Children with this disorder have difficulty climbing stairs, fall often, can't run properly, and their scapulae flare out (or "wing") when they raise their arms. Calf muscles especially become enlarged and firm. Muscle deterioration progresses rapidly, and contractures develop. Some have abrupt intermittent oscillations of the irises in response to light (Gower sign). Usually, these children are confined to wheelchairs by ages 9 to 12. Late in the disease, progressive weakening of cardiac muscle causes tachycardia, electrocardiogram abnormalities, and pulmonary complications. Death commonly results from sudden heart failure, respiratory failure, or infection.

Signs and symptoms of Becker muscular dystrophy resemble those of Duchenne muscular dystrophy, but they progress more slowly. It generally affects older boys and young men. Children affected usually walk through their teens and into adulthood—sometimes into their 40s. Cardiac involvement is much less frequent.

Facioscapulohumeral dystrophy is a slowly progressive and relatively benign form of muscular dystrophy that commonly occurs before age 10 but may develop during early adolescence. The earlier the disease occurs, the more rapid and progressive it is. Initially, it weakens the muscles of the face, shoulders, and upper arms but eventually spreads to all voluntary muscles, producing a pendulous lower lip and absence of the nasolabial fold. Early symptoms include the inability to pucker the mouth or whistle, abnormal facial movements, and the absence of facial movements when

laughing or crying. Other signs consist of diffuse facial flattening that leads to a masklike expression, winging of the scapulae, the inability to raise the arms above the head and, in infants, the inability to suckle.

Limb-girdle dystrophy follows a similarly slow course and commonly causes only slight disability. Usually, it begins between ages 6 and 10; less commonly, in early adulthood. The later the onset, the more rapid the progression. Muscle weakness first appears in the upper arm and pelvic muscles. Other symptoms include winging of the scapulae, lordosis with abdominal protrusion, waddling gait, poor balance, and the inability to raise the arms.

Diagnosis

Diagnosis depends on typical clinical findings, family history, and diagnostic test findings. If another family member has muscular dystrophy, its clinical characteristics can indicate the type of dystrophy the patient has and how he may be affected.

Electromyography typically demonstrates short, weak bursts of electrical activity or high-frequency, repetitive waxing and waning discharges in affected muscles. Muscle biopsy shows variations in the size of muscle fibers and, in later stages, shows fat and connective tissue deposits; dystrophin is absent in Duchenne dystrophy and diminished in Becker dystrophy. Serum creatine kinase level is markedly elevated in Duchenne, but only moderately elevated in Becker and facioscapulohumeral dystrophies.

Immunologic and molecular biologic assays available in specialized medical centers facilitate accurate prenatal and postnatal diagnosis of Duchenne and Becker muscular dystrophies and are replacing muscle biopsy and elevated serum creatine kinase levels in diagnosing these dystrophies. These assays can also help to identify carriers.

Treatment

No treatment stops the progressive muscle impairment of muscular dystrophy. However, orthopedic appliances, exercise, physical therapy, and surgery to correct contractures can help preserve the patient's mobility and independence. Prednisone improves muscle strength in patients with Duchenne.

Special Considerations

Comprehensive long-term care and follow-up, patient and family teaching, and psychological support can help the patient and the family deal with this disorder.

- ◆ When respiratory involvement occurs in Duchenne muscular dystrophy, encourage coughing, deep-breathing exercises, and diaphragmatic breathing. Teach parents how to recognize early signs of respiratory complications.
- ◆ Encourage and assist with active and passive ROM exercises to preserve joint mobility and prevent muscle atrophy.
- ◆ Advise the patient to avoid long periods of bed rest and inactivity; if necessary, limit TV viewing and other sedentary activities.
- ◆ Refer the patient for physical therapy. Splints, braces, trapeze bars, overhead slings, and a wheelchair can help preserve mobility. A footboard or high-topped sneakers and a foot cradle increase comfort and prevent footdrop.
- ◆ Refer the patient to surgery to correct contractures.
- ◆ Because inactivity may cause constipation, encourage adequate fluid intake, increase dietary bulk, and obtain an order for a stool softener. The patient is prone to obesity due to reduced physical activity; assist with planning a low-calorie, high-protein, high-fiber diet if needed.
- ◆ Always allow the patient plenty of time to perform even simple physical tasks because if feeling slow and awkward.
- ◆ Encourage communication between the patient's family members to help them deal with the emotional strain this disorder produces. Provide emotional support to help the patient cope with continual changes in body image.



PEDIATRIC TIP *Help the child with Duchenne muscular dystrophy to maintain peer relationships and to realize the intellectual potential by encouraging the parents to keep the child in a regular school if possible.*

- ◆ If necessary, refer adult patients for counseling. Refer those who must acquire new job skills for vocational rehabilitation. (Contact the Department of Labor and Industry in your state for more information.)

For information on social services and financial assistance, refer these patients and their families to the Muscular Dystrophy Association.

- ◆ Refer the patient's family members for genetic counseling.

Joints

SEPTIC ARTHRITIS

Septic, or infectious, arthritis is a medical emergency that occurs when bacterial invasion of a joint causes inflammation of the synovial lining, effusion and pyogenesis, and destruction of bone and cartilage. Septic arthritis can lead to ankylosis and even fatal septicemia. However, prompt antibiotic therapy and joint aspiration or drainage cures most patients.

Causes and Incidence

In most cases of septic arthritis, bacteria spread from a primary site of infection—usually in adjacent bone or soft tissue—through the bloodstream to the joint. Common infecting organisms in children are group B *Streptococcus* and *Haemophilus influenzae*. Adults are usually infected by *Staphylococcus*, *Streptococcus*, *Neisseria gonorrhoeae* (pneumonia), and group B *Streptococcus*, whereas chronic septic arthritis is caused by *Mycobacterium tuberculosis* and *Candida albicans*.

Various factors can predispose a person to septic arthritis. Any concurrent bacterial infection (of the genitourinary or the upper respiratory tract, for example) or serious chronic illness (such as malignancy, renal failure, rheumatoid arthritis, systemic lupus erythematosus, diabetes, or cirrhosis) heightens susceptibility. Consequently, elderly people and those who abuse I.V. drugs run a higher risk of developing septic arthritis. Of course, diseases that depress the immune system and immunosuppressant therapy increase susceptibility. Other predisposing factors include recent articular trauma, joint arthroscopy or other surgery, intra-articular injections, local joint abnormalities, animal or human bites, and nail puncture wounds.

Septic arthritis may be seen at any age in children, but it occurs most often in children younger than 3 years old. It's uncommon from age 3 until adolescence, at which time the incidence increases again.

Pathophysiology

Previously damaged joints, especially those damaged by rheumatoid arthritis, are the most susceptible to infection. The synovial membranes of these joints exhibit neovascularization and increased adhesion factors; both conditions increase the chance of bacteremia, resulting in a joint infection. Some microorganisms have properties that promote their tropism to the synovium. *Staphylococcus aureus* readily binds to articular sialoprotein, fibronectin collage, elastin, hyaluronic acid, and prosthetic material via specific tissue adhesion factors (microbial surface components recognizing adhesive matrix molecules). In adults, the arteriolar anastomosis between the epiphysis and the synovium permits the spread of osteomyelitis into the joint space.

Complications

- ◆ Joint degeneration
- ◆ Osteomyelitis

Signs and Symptoms

Acute septic arthritis begins abruptly, causing intense pain, inflammation, and swelling of the affected joint and low-grade fever. It usually affects a single joint. It most commonly develops in the large joints but can strike any joint, including the spine and small peripheral joints. The hip is a frequent site in infants. Systemic signs of inflammation may not appear in some patients. Migratory polyarthritis sometimes precedes localization of the infection. If the bacteria invade the hip, pain may occur in the groin, upper thigh, or buttock or may be referred to the knee.

Diagnosis

Dx CONFIRMING DIAGNOSIS *Identifying the causative organism in a Gram stain or culture of synovial fluid or a biopsy of synovial membrane confirms septic arthritis. When synovial fluid culture is negative, positive blood culture may confirm the diagnosis. Ultrasound of the hip is the modality of choice to detect fluid collections in the hip joint and can serve as a guide during aspiration procedures.*

Joint fluid analysis shows gross pus or watery, cloudy fluid of decreased viscosity, usually with $50,000/\mu\text{L}$ or more white cells, primarily neutrophils.

Synovial fluid glucose concentration is usually greater than 40 mg/dL. (See *Other types of arthritis*, page 299.)

Other Types of Arthritis

Hemophilic Arthrosis

Hemophilic arthrosis produces transient or permanent joint changes. Often precipitated by trauma, hemophilic arthrosis usually arises between ages 1 and 5 and tends to recur until about age 10. It usually affects only one joint at a time—most commonly the knee, elbow, or ankle—and tends to recur in the same joint. Initially, the patient may feel only mild discomfort; later, he may experience warmth, swelling, tenderness, and severe pain with adjacent muscle spasm that leads to flexion of the extremity.

Mild hemophilic arthrosis may cause only limited stiffness that subsides within a few days. In prolonged bleeding, however, symptoms may subside after weeks or months or not at all. Severe hemophilic arthrosis may be accompanied by fever and leukocytosis; severe, prolonged, or repeated bleeding may lead to chronic hemophilic joint disease.

Effective treatment includes I.V. infusion of the deficient clotting factor, bed rest with the affected extremity elevated, application of ice packs, analgesics, and joint aspiration. Physical therapy includes progressive range-of-motion and muscle-strengthening exercises to restore motion and to prevent contractures and muscle atrophy.

Intermittent Hydrarthrosis

Intermittent hydrarthrosis is a rare, benign condition characterized by regular, recurrent joint effusions. It most commonly affects the knee. The patient may have difficulty moving the affected joint but have no other arthritic symptoms. The cause of intermittent hydrarthrosis is unknown; onset is usually at or soon after puberty and may be linked to familial tendencies, allergies, or menstruation. No effective treatment exists.

Henoch–Schönlein Purpura

Henoch–Schönlein purpura—a vasculitic syndrome—is marked by palpable purpura, abdominal pain, and arthralgia that most commonly affects the knees and ankles, producing swollen, warm, and tender joints without joint erosion or deformity. Renal involvement is also common. Most patients have microscopic hematuria and proteinuria 4 to 8 weeks after onset. Incidence is highest in children and young adults, occurring most often in the spring after a respiratory infection. Treatment may include corticosteroids.

Traumatic Arthritis

Traumatic arthritis results from blunt, penetrating, or repeated trauma or from forced inappropriate motion of a joint or ligament. Clinical effects may include swelling, pain, tenderness, joint instability, and internal bleeding. Treatment includes analgesics, nonsteroidal anti-inflammatory drugs, application of cold followed by heat and, if needed, compression dressings, splinting, joint aspiration, casting, or possibly surgery.

Other diagnostic measures include the following:

- ◆ X-rays can show typical changes as early as 1 week after initial infection—distention of joint capsules, for example, followed by narrowing of joint space (indicating cartilage damage) and erosions of bone (joint destruction).
- ◆ White blood cell (WBC) count may be elevated, with many polymorphonuclear cells; erythrocyte sedimentation rate is increased.
- ◆ Triple-phase bone scan is often used in children. A whole body scan is preferred in very young children.
- ◆ CT and MRI can provide useful images to delineate the extent of the infection.

Treatment

Antibiotic therapy should begin as soon as a Gram stain has been done; it may be modified when drug sensitivity of the infecting organism is known. Bioassays or bactericidal assays of synovial fluid and bioassays of blood may confirm clearing of the infection.

Rest, immobilization, elevation, and warm compresses help with pain relief. Analgesics are given for pain, if needed. The affected joint can be

immobilized with a splint or put into traction until the patient can tolerate movement.

In severe cases, needle aspiration (arthrocentesis) or surgery may be done under sterile conditions to remove grossly purulent or infected joint fluid. Late reconstructive surgery is warranted only for severe joint damage and only after all signs of active infection have disappeared, which usually takes several months. Recommended procedures include arthroplasty and joint fusion. Prosthetic replacement remains controversial because it may exacerbate the infection, but it has helped patients with damaged femoral heads or acetabula.

Special Considerations

Management of septic arthritis demands meticulous supportive care, close observation, and control of infection.

- ◆ Practice strict sterile technique for all procedures. Wash hands carefully before and after giving care. Dispose of soiled linens and dressings properly. Prevent contact between immunosuppressed patients and infected patients.
- ◆ Watch for signs of joint inflammation: heat, redness, swelling, pain, or drainage. Monitor vital signs and fever pattern. Remember that corticosteroids mask signs of infection.
- ◆ Check splints or traction regularly. Keep the joint in proper alignment but avoid prolonged immobilization. Start passive ROM exercises immediately, and progress to active exercises as soon as the patient can move the affected joint and put weight on it.
- ◆ Monitor pain levels and medicate accordingly, especially before exercise, remembering that the pain of septic arthritis is easy to underestimate. Administer analgesics and opioids for acute pain and heat or ice packs for moderate pain.
- ◆ Warn the patient before the first aspiration that it will be *extremely* painful. Carefully evaluate the patient's condition after joint aspiration.



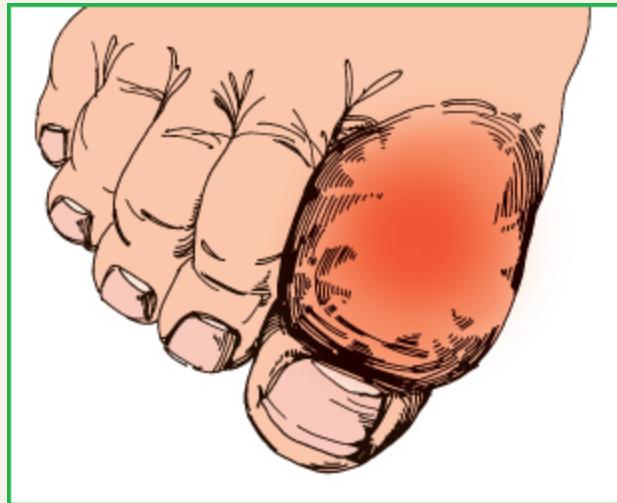
ELDER TIP Monitor older adults who are on long-term opioid therapy because these drugs can impair mental status and may contribute to falls and other accidents.

GOUT

Gout, also called *gouty arthritis*, is a metabolic disease marked by urate deposits, which cause painfully arthritic joints. (See *Gouty deposits*.) It can strike any joint but favors those in the feet and legs. Gout follows an intermittent course and typically leaves patients totally free from symptoms for years between attacks. It can cause chronic disability or incapacitation and, rarely, severe hypertension and progressive renal disease. The prognosis is good with treatment.

Gouty Deposits

The final stage of gout is marked by painful polyarthritis, with large, subcutaneous, tophaceous deposits in cartilage, synovial membranes, tendons, and soft tissue. The skin over the tophus is shiny, thin, and taut.



Causes and Incidence

Although the exact cause of primary gout remains unknown, it appears to be linked to a genetic defect in purine metabolism, which causes elevated blood levels of uric acid (hyperuricemia) due to overproduction of uric acid, retention of uric acid, or both. In secondary gout, which develops during the course of another disease (such as obesity, diabetes mellitus, hypertension, sickle cell anemia, and renal disease), hyperuricemia results from the breakdown of nucleic acids. Myeloproliferative and lymphoproliferative

diseases, psoriasis, and hemolytic anemia are the most common causes. Primary gout usually occurs in men and in postmenopausal women; secondary gout occurs in elderly people.

Secondary gout can also follow drug therapy that interferes with uric acid excretion. Increased concentration of uric acid leads to urate deposits (*tophi*) in joints or tissues and consequent local necrosis or fibrosis. The risk is greater in men, postmenopausal women, and those who use alcohol.

Pathophysiology

When uric acid becomes supersaturated in blood and other body fluids, it crystallizes and forms a precipitate of urate salts that accumulate in connective tissue throughout the body; these deposits are called *tophi*. The presence of the crystals triggers an acute inflammatory response when neutrophils begin to ingest the crystals. Tissue damage begins when the neutrophils release their lysosomes (see Chapter 8). The lysosomes not only damage the tissues but also perpetuate the inflammation.

In asymptomatic gout, the serum urate level increases, but the urate doesn't crystallize or produce symptoms. As the disease progresses, it may cause hypertension or urate kidney stones may form.

Complications

- ◆ Renal calculi
- ◆ Atherosclerotic disease
- ◆ Cardiovascular disease
- ◆ Stroke
- ◆ Coronary thrombosis
- ◆ Hypertension
- ◆ Infection when tophi rupture

Signs and Symptoms

Gout develops in four stages: asymptomatic, acute, intercritical, and chronic. In asymptomatic gout, serum urate levels rise but produce no symptoms. As the disease progresses, it may cause hypertension or nephrolithiasis, with severe back pain. The first acute attack strikes suddenly and peaks quickly. Although it generally involves only one or a few joints, this initial attack is extremely painful. Affected joints are hot, tender, inflamed, and appear dusky-red or cyanotic. The

metatarsophalangeal joint of the great toe usually becomes inflamed first (*podagra*), followed by the instep, ankle, heel, knee, or wrist joints. Sometimes a low-grade fever is present. Mild acute attacks usually subside quickly but tend to recur at irregular intervals. Severe attacks may persist for days or weeks.

Intercritical periods are the symptom-free intervals between gout attacks. Most patients have a second attack within 6 months to 2 years, but in some the second attack doesn't occur for 5 to 10 years. Delayed attacks are more common in untreated patients and tend to be longer and more severe than initial attacks. Such attacks are also polyarticular, invariably affecting joints in the feet and legs, and are sometimes accompanied by fever. A migratory attack sequentially strikes various joints and the Achilles tendon and is associated with either subdeltoid or olecranon bursitis.

Eventually, chronic polyarticular gout sets in. This final, unremitting stage of the disease is marked by persistent painful polyarthritides, with large, subcutaneous tophi in cartilage, synovial membranes, tendons, and soft tissue. Tophi form in fingers, hands, knees, feet, ulnar sides of the forearms, helix of the ear, Achilles tendons and, rarely, internal organs, such as the kidneys and myocardium. The skin over the tophus may ulcerate and release a chalky, white exudate or pus. Chronic inflammation and tophaceous deposits precipitate secondary joint degeneration, with eventual erosions, deformity, and disability. Kidney involvement, with associated tubular damage, leads to chronic renal dysfunction. Hypertension and albuminuria occur in some patients; urolithiasis is common.

Diagnosis



CONFIRMING DIAGNOSIS *The presence of monosodium urate monohydrate crystals in synovial fluid taken from an inflamed joint or tophus establishes the diagnosis.*

Aspiration of synovial fluid (arthrocentesis) or of tophaceous material reveals needlelike intracellular crystals of sodium urate. Although hyperuricemia isn't specifically diagnostic of gout, serum uric acid is above normal. Urinary uric acid is usually higher in secondary gout than in primary gout. In acute attacks, erythrocyte sedimentation rate and WBC count may be elevated, and WBC count shifts to the left.

Initially, X-rays are normal. However, in chronic gout, X-rays show “punched out” erosions, sometimes with periosteal overgrowth. Outward displacement of the overhanging margin from the bone contour characterizes gout. X-rays rarely show tophi. (See *Understanding pseudogout*.)

Understanding Pseudogout

Also known as *calcium pyrophosphate disease*, pseudogout results when calcium pyrophosphate crystals collect in periarticular joint structures.

Signs and Symptoms

Like true gout, pseudogout causes sudden joint pain and swelling, most commonly of the knee, wrist, and ankle or other peripheral joints.

Pseudogout attacks are self-limiting and triggered by stress, trauma, surgery, severe dieting, thiazide therapy, or alcohol abuse. Associated symptoms resemble those of rheumatoid arthritis and osteoarthritis. Many patients may be asymptomatic.

Establishing a Diagnosis

Diagnosis of pseudogout hinges on joint aspiration and synovial biopsy to detect calcium pyrophosphate crystals. X-rays show calcium deposits in the fibrocartilage and linear markings along the bone ends. Blood tests may detect an underlying endocrine or metabolic disorder.

Relief for Pressure and Inflammation

Management of pseudogout may include aspirating the joint to relieve pressure; instilling corticosteroids and administering analgesics, salicylates, phenylbutazone, or other nonsteroidal anti-inflammatory drugs to treat inflammation and, if appropriate, treating the underlying disorder. Without treatment, pseudogout leads to permanent joint damage in about half of those it affects, most of whom are older adults.

Treatment

Correct management seeks to terminate an acute attack, reduce hyperuricemia, and prevent recurrence, complications, and the formation of renal calculi. (See *Preventing gout*.) Colchicine is effective in reducing pain, swelling, and inflammation; pain often subsides within 12 hours of treatment and is completely relieved in 48 hours. Treatment for the patient with acute gout consists of bed rest; immobilization and protection of the inflamed, painful joints; and local application of heat or cold, whichever works for the patient. Maximal doses of nonsteroidal anti-inflammatory drugs (NSAIDs) usually provide excellent relief for patients who can tolerate them; doses should be gradually reduced after several days.



PREVENTION PREVENTING GOUT

Because the cause of gout is unknown, the disease can't be prevented. However, it's important to teach your patients how to prevent acute gout attacks to reduce the risk of joint damage. Acute gout attacks can be prevented by dietary changes, weight reduction, adequate fluid intake, and drugs.

Dietary Restrictions

Dietary changes include avoidance of foods high in purine, such as alcohol (especially beer and wine), organ meats, sardines, sweetbreads, peas, and lentils.

Weight Reduction

Obese patients need to lose weight at a slow rate. Losing weight rapidly may temporarily increase uric acid levels.

Fluid Intake

It's also important to drink adequate amounts of fluids to dilute the amount of uric acid in the blood. This will help decrease the risk of kidney stone formation. Taking the prescribed drug slows the production of uric acid and speeds its elimination from the body.



ELDER TIP Older patients are at risk for GI bleeding associated with NSAID use. Encourage the elderly patient to take these drugs with meals and monitor the patient's stools for occult blood.

Resistant inflammation may require oral corticosteroids or intra-articular corticosteroid injection to relieve pain. Treatment for chronic gout aims to decrease serum uric acid level. Continuing maintenance dosage of allopurinol may be given to suppress uric acid formation or control uric acid levels, preventing further attacks. However, this powerful drug should be used cautiously in patients with renal failure. Uricosuric agents promote uric acid excretion and inhibit accumulation of uric acid, but their value is limited in patients with renal impairment. These medications shouldn't be given to patients with renal calculi.

Adjunctive therapy emphasizes a few dietary restrictions, primarily the avoidance of alcohol and purine-rich foods (organ meats, beer, wine, and certain types of fish are high in purines). Obese patients should try to lose weight because obesity puts additional stress on painful joints.

In some cases, surgery may be necessary to improve joint function or correct deformities. Tophi must be excised and drained if they become infected or ulcerated. They can also be excised to prevent ulceration, improve the patient's appearance, or make it easier for the patient to wear shoes or gloves.

Special Considerations

Patient care for gout includes these interventions:

- ◆ Encourage bed rest but use a bed cradle to keep bedcovers off extremely sensitive, inflamed joints.
- ◆ Give pain medication, as needed, especially during acute attacks. Apply hot or cold packs to inflamed joints according to what the patient finds effective. Administer anti-inflammatory medication and other drugs, as ordered. Watch for adverse effects. Be alert for GI disturbances with colchicine.
- ◆ Watch for acute gout attacks 24 to 96 hours after surgery. Even minor surgery can precipitate an attack. Before and after surgery, administer colchicine as ordered, to help prevent gout attacks.
- ◆ Tell the patient to avoid high-purine foods, such as anchovies, liver, sardines, kidneys, sweetbreads, lentils, and alcoholic beverages—

especially beer and wine—which raise the urate level. Explain the principles of a gradual weight-reduction diet to obese patients.

- ◆ Advise the patient to report any adverse effects of allopurinol, such as drowsiness, dizziness, nausea, vomiting, urinary frequency, or dermatitis.

NEUROGENIC ARTHROPATHY

Neurogenic arthropathy, also called *Charcot arthropathy*, is a progressively degenerative disease of peripheral and axial joints, resulting from impaired sensory innervation. The loss of sensation in the joints causes progressive deterioration, resulting from trauma or primary disease, which leads to laxity of supporting ligaments and eventual disintegration of the affected joints.

Causes and Incidence

Neurogenic arthropathy is most common in men older than 40 years. In adults, the most common cause of neurogenic arthropathy is diabetes mellitus. Other causes include tabes dorsalis (especially among patients 40 to 60 years old), syringomyelia (progresses to neurogenic arthropathy in about 25% of patients), myelopathy of pernicious anemia, spinal cord trauma, paraplegia, hereditary sensory neuropathy, and Charcot–Marie–Tooth disease. Amyloidosis, peripheral nerve injury, myelomeningocele (in children), leprosy, and alcoholism may cause neurogenic arthropathy, but only in rare occurrences.

Frequent intra-articular injection of corticosteroids has also been linked to neurogenic arthropathy. The analgesic effect of the corticosteroids may mask symptoms and allow continuous stress to accelerate joint destruction.

Pathophysiology

Many conditions predispose to neurogenic arthropathy. Impaired deep pain sensation or proprioception affects the joint's normal protective reflexes, often allowing trauma (especially repeated minor episodes) and small periarticular fractures to go unrecognized. Increased blood flow to bone from reflex vasodilation, resulting in active bone resorption, contributes to bone and joint damage.

Each new injury sustained by the joint causes more distortion as it heals. Hemorrhagic joint effusions and multiple small fractures can occur,

accelerating disease progression. Ligamentous laxity, muscular hypotonia, and rapid destruction of joint cartilage are common, predisposing to joint dislocations, which also accelerate disease progression. Advanced neurogenic arthropathy can cause hypertrophic changes, destructive changes, or both.

Complications

- ◆ Joint subluxation or dislocation
- ◆ Pathologic fractures
- ◆ Infection
- ◆ Pseudogout
- ◆ Neurovascular compression

Signs and Symptoms

Neurogenic arthropathy begins insidiously with swelling, warmth, decreased mobility, and instability in a single joint or in many joints. It can progress to deformity. The first clue to vertebral neuroarthropathy, which progresses to gross spinal deformity, may be nothing more than a mild, persistent backache. Characteristically, pain is minimal despite obvious deformity.

The specific joint affected varies according to the underlying cause. Diabetes usually attacks the joints and bones of the feet; tabes dorsalis attacks the large weight-bearing joints, such as the knee, hip, ankle, or lumbar and dorsal vertebrae (Charcot spine); syringomyelia causes occurrence in the shoulder, elbow, or cervical intervertebral joint. Neurogenic arthropathy caused by intra-articular injection of corticosteroids usually develops in the hip or knee joint.

Diagnosis

Patient history of painless joint deformity and underlying primary disease suggests neurogenic arthropathy. Physical examination may reveal bone fragmentation in advanced disease. X-rays confirm diagnosis and assess severity of joint damage. In the early stage of the disease, soft-tissue swelling or effusion may be the only overt effect; in the advanced stage, articular fracture, subluxation, erosion of articular cartilage, periosteal new bone formation, and excessive growth of marginal loose bodies

(osteophytosis) or resorption may be seen. CT scan helps define the extent of disease.

Other diagnostic measures include:

- ◆ vertebral examination: narrowing of disk spaces, deterioration of vertebrae, and osteophyte formation, leading to ankylosis and deforming kyphoscoliosis
- ◆ synovial biopsy: bony fragments and bits of calcified cartilage.

Treatment

Effective management relieves pain with analgesics and immobilization using crutches, splints, braces, and restriction of weight bearing to the affected joint.

In severe disease, surgery may include arthrodesis or, in severe diabetic neuropathy, amputation. However, surgery risks further damage through nonunion and infection.

Special Considerations

Assess the pattern of pain and give analgesics, as needed. Check sensory perception, ROM, alignment, joint swelling, and the status of underlying disease.

- ◆ Teach the patient to use joint protection techniques, to avoid physically stressful actions that may cause pathologic fractures, and to take safety precautions, such as removing throw rugs and other objects over which the patient may trip.
- ◆ Advise the patient to report severe joint pain, swelling, or instability. Warm compresses may be applied to relieve local pain and tenderness.
- ◆ Instruct the patient in the proper technique for crutches or other orthopedic devices. Stress the importance of proper fitting and regular professional readjustment of such devices. Warn the patient that impaired sensation might allow damage from these aids to occur and progress without discomfort.
- ◆ Emphasize the need to continue regular treatment of the underlying disease.

OSTEOARTHRITIS

Osteoarthritis, the most common form of arthritis, is a chronic disease that causes deterioration of the joint cartilage and formation of reactive new bone at the margins and subchondral areas of the joints. This degeneration results from a breakdown of chondrocytes, most commonly in the distal interphalangeal and proximal interphalangeal joints, but also in the hip and knee joints.

Osteoarthritis is widespread, occurring equally in both sexes. Its earliest symptoms typically begin after age 40 and may progress with advancing age.

Disability depends on the site and severity of involvement and can range from minor limitation of the dexterity of the fingers to severe disability in persons with hip or knee involvement. The rate of progression varies, and joints may remain stable for years in an early stage of deterioration.

Causes and Incidence

Studies indicate that osteoarthritis is acquired and probably results from a combination of metabolic, genetic, chemical, and mechanical factors. Secondary osteoarthritis usually follows an identifiable predisposing event—most commonly trauma, metabolic conditions, congenital deformity, or obesity—and leads to degenerative changes.

Osteoarthritis may first appear between ages 30 and 40 and is present in almost everyone by age 70. Before age 55, it affects men and women equally, but after age 55 the incidence is higher in women.



ELDER TIP Primary osteoarthritis is strongly associated with aging, and indeed aging may predispose to the cartilage degeneration common in persons with osteoarthritis.

Pathophysiology

The major defect in primary and secondary osteoarthritis is loss of articular cartilage. Articular cartilage is probably lost through enzymatic breakdown of the cartilage matrix—the proteoglycans, glycosaminoglycans, and collagen. Other studies indicate that interleukin-1 may play a part in cartilage destruction.

Osteoarthritis occurs in synovial joints. The joint cartilage deteriorates, and reactive new bone forms at the margins and subchondral areas of the joints. The degeneration results from damage to the chondrocytes. Cartilage softens with age, narrowing the joint space. Mechanical injury erodes

articular cartilage, leaving the underlying bone unprotected. This causes sclerosis or thickening and hardening of the bone underneath the cartilage.

Articular cartilage particles within the joint irritate the synovial lining, which becomes fibrotic and limits joint movement. Synovial fluid may be forced into defects in the bone, causing cysts. New bone, called *osteophyte* (bone spur), forms at joint margins as the articular cartilage erodes, causing gross alteration of the bony contours and enlargement of the joint. The spurlike bony projections enlarge until small pieces called joint mice break off into the synovial cavity.

Complications

- ◆ Flexion contractures
- ◆ Subluxation and deformity
- ◆ Ankylosis
- ◆ Bony cysts
- ◆ Gross bone overgrowth
- ◆ Central cord syndrome
- ◆ Nerve root compression
- ◆ Cauda equina syndrome

Signs and Symptoms

The most common symptom of osteoarthritis is a deep, aching joint pain, particularly after exercise or weight bearing, usually relieved by rest. Other symptoms include stiffness in the morning and after inactivity (improves with activity), aching during changes in weather, “grating” of the joint during motion, altered gait contractures, joint instability, and limited movement. These symptoms increase with poor posture, obesity, and stress to the affected joint.

Osteoarthritis of the interphalangeal joints produces irreversible joint changes and node formation. The nodes eventually become red, swollen, and tender, causing numbness and loss of dexterity. (See *What happens in osteoarthritis.*)



PATHOPHYSIOLOGY WHAT HAPPENS IN OSTEOARTHRITIS

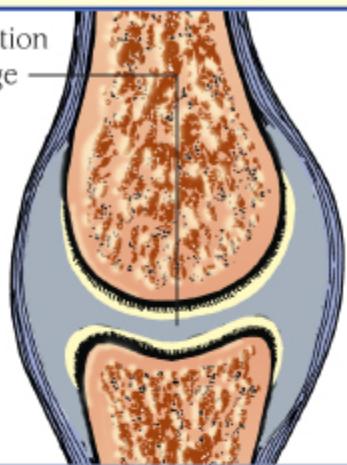
The characteristic breakdown of articular cartilage is a gradual response to aging or to predisposing factors, such as joint abnormalities or traumatic injury.

Chondrocytes break down.



Cartilage degenerates.

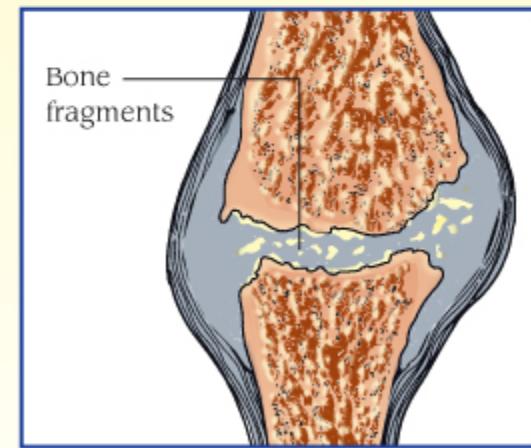
Degeneration
of cartilage



Osteophytes (bony spurs) form.

Fragments of bone float freely
in joint.

Bone
fragments



Stiffness and decreased
movement occur.

Diagnosis

A thorough physical examination confirms typical symptoms, and absence of systemic symptoms rules out an inflammatory joint disorder. X-rays of the affected joint help confirm diagnosis of osteoarthritis but may be normal in the early stages. X-rays may require many views and typically show:

- ◆ narrowing of joint space or margin
- ◆ cystlike bony deposits in joint space and margins and sclerosis of the subchondral space
- ◆ joint deformity due to degeneration or articular damage
- ◆ bony growths at weight-bearing areas
- ◆ fusion of joints. (See *A close look at the effects of osteoarthritis*, page 306.)

A Close Look at the Effects of Osteoarthritis

Involvement of the interphalangeal (finger bone) joints produces irreversible changes in the distal joints (Heberden nodes) and the proximal joints (Bouchard nodes), as shown below. These nodes can be painless initially, with gradual progression to or sudden flare-ups of redness, swelling, tenderness, and impaired sensation and dexterity.

Heberden nodes in the distal joints.



Bouchard nodes in the proximal joints.



MRI and CT scans may be used to show cartilage breakdown and bone abnormalities. Importantly, MRI can detect signs of inflammation of the bone or the synovial membrane.

Treatment

Treatment is aimed at relieving pain, maintaining or improving mobility, and minimizing disability. Medications include NSAIDs, cyclo-oxygenase-2 inhibitors and, in some cases, intra-articular injections of corticosteroids. Studies indicate that glucosamine and chondroitin may be useful in controlling symptoms and reducing functional impairment. Injecting artificial joint fluid into the knee can provide relief of pain for up to 6 months.

Effective treatment also reduces stress by weight loss and supporting or stabilizing the joint with crutches, braces, cane, walker, cervical collar, or traction. Exercise, such as through physical therapy, is integral to maintaining or improving joint mobility. Other supportive measures include massage, moist heat, paraffin dips for hands, protective techniques to prevent undue stress on the joints, and adequate rest (particularly after activity).

Surgical treatment, such as one of the following, is reserved for patients who have severe disability or uncontrollable pain:

- ◆ Arthroplasty (partial or total): replacement of deteriorated part of joint with prosthetic appliance
- ◆ Arthrodesis: surgical fusion of bones, used primarily in spinal surgery (laminectomy)
- ◆ Osteoplasty: scraping and lavage of deteriorated bone from joint
- ◆ Osteotomy: change in alignment of bone to relieve stress by excision of wedge of bone or cutting of bone

Special Considerations

Patient care for osteoarthritis includes the following:

- ◆ Promote adequate rest, particularly after activity. Plan rest periods during the day and provide for adequate sleep at night. Moderation is the key—teach the patient to pace daily activities.

- ◆ Assist with physical therapy, and encourage the patient to perform gentle, isometric ROM exercises.
- ◆ Provide emotional support and reassurance to help the patient cope with limited mobility. Explain that osteoarthritis *isn't* a systemic disease.

Specific patient care depends on the affected joint:

- ◆ *Hand*: Apply hot soaks and paraffin dips to relieve pain, as ordered.
- ◆ *Spine (lumbar and sacral)*: Recommend a firm mattress (or bed board) to decrease morning pain.
- ◆ *Spine (cervical)*: Check cervical collar for constriction; watch for redness with prolonged use.
- ◆ *Hip*: Use moist heat pads to relieve pain and administer antispasmodic drugs, as ordered. Assist with ROM and strengthening exercises, always making sure the patient gets the proper rest afterward. Check crutches, cane, braces, and walker for proper fit, and teach the patient to use them correctly. For example, the patient with unilateral joint involvement should use an orthopedic appliance such as a walker or a cane.
Recommend the use of cushions when sitting as well as the use of an elevated toilet seat.
- ◆ *Knee*: Twice daily, assist with prescribed ROM exercises, exercises to maintain muscle tone, and progressive resistance exercises to increase muscle strength. Provide elastic supports or braces if needed.

To minimize the long-term effects of osteoarthritis:

- ◆ Teach the patient to take medication exactly as prescribed and to report adverse effects immediately.
- ◆ Advise the patient to avoid overexertion, taking care to stand and walk correctly, to minimize high-impact activities, and to be especially careful when stooping or picking up objects.
- ◆ Instruct the patient to wear proper-fitting, supportive shoes and not to allow the heels to become worn down.
- ◆ Advise the patient to install safety devices at home such as guard rails in the bathroom.
- ◆ Instruct the patient to maintain proper body weight to lessen strain on joints.

Bones

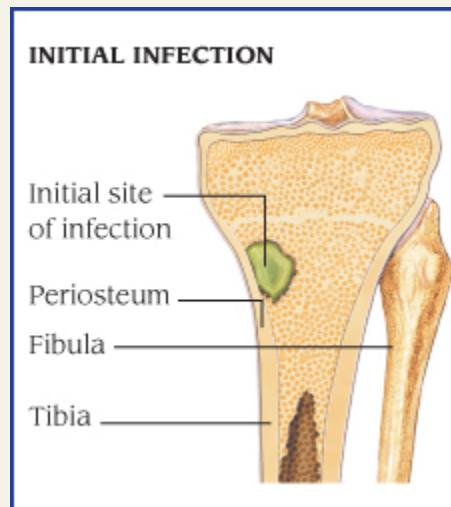
OSTEOMYELITIS

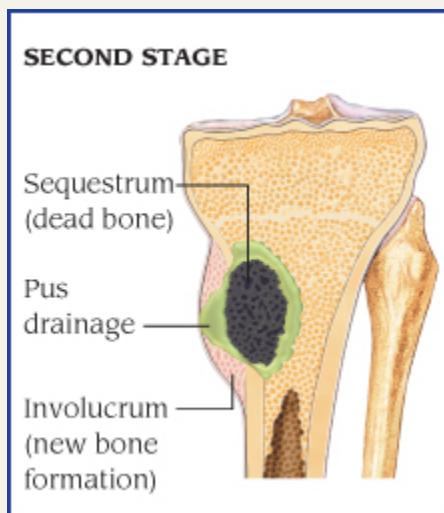
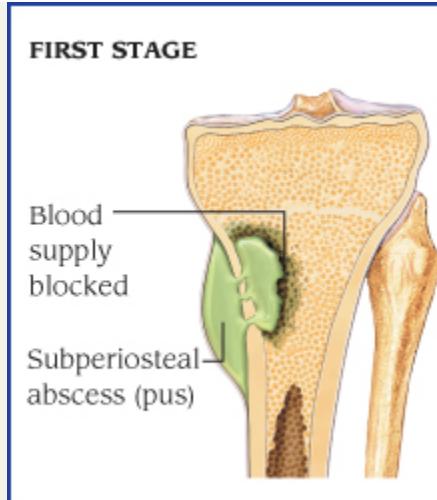
Osteomyelitis is a pyogenic bone infection that may be chronic or acute. It commonly results from a combination of local trauma, which is usually quite trivial but results in hematoma formation, and an acute infection originating elsewhere in the body. Although osteomyelitis usually remains localized, it can spread through the bone to the marrow, cortex, and periosteum. Acute osteomyelitis is usually a blood-borne disease, which most commonly affects rapidly growing children. Chronic osteomyelitis, which is rare, is characterized by multiple draining sinus tracts and metastatic lesions. (See *Stages of osteomyelitis*.)



PATHOPHYSIOLOGY STAGES OF OSTEOMYELITIS

The illustrations show the progression of osteomyelitis.





Causes and Incidence

Virtually any pathogenic bacteria can cause osteomyelitis under the right circumstances. Typically, these organisms find a culture site in a hematoma from recent trauma or in a weakened area, such as the site of surgery or local infection (for example, furunculosis), and spread directly to bone. As the organisms grow and form pus within the bone, tension builds within the rigid medullary cavity, forcing pus through the haversian canals. This forms a subperiosteal abscess that deprives the bone of its blood supply and may eventually cause necrosis. In turn, necrosis stimulates the periosteum to create new bone (*involucrum*); the old bone (*sequestrum*) detaches and

works its way out through an abscess or the sinuses. By the time sequestrum forms, osteomyelitis is chronic.

Osteomyelitis occurs more commonly in children (especially boys) than in adults—usually as a complication of an acute localized infection. The most common sites in children are the lower end of the femur and the upper end of the tibia, humerus, and radius. The most common sites in adults are the pelvis and vertebrae, generally because of contamination associated with surgery or trauma. Other common sites are sternoclavicular, sacroiliac, and symphysis pubis. The incidence of both chronic and acute osteomyelitis is declining, except in drug abusers. With prompt treatment, the prognosis for acute osteomyelitis is very good; for chronic osteomyelitis, which is more prevalent in adults, the prognosis is still poor.

Pathophysiology

Typically, bacteria find a culture site in a hematoma from recent trauma or in a weakened area, such as the site of local infection (e.g., furunculosis), and travel through the bloodstream to the metaphysis, the section of a long bone that's continuous with the epiphysis plates, where the blood flows into sinusoids. Predisposing factors include diabetes mellitus, sickle cell disease, and being immunocompromised.

Complications

- ◆ Chronic osteomyelitis
- ◆ Poor joint function
- ◆ Amputation of limb

Signs and Symptoms

The onset of acute osteomyelitis is usually rapid, with sudden pain accompanied by tenderness, heat, swelling, and restricted movement of the affected area. Associated systemic symptoms may include tachycardia, sudden fever, nausea, and malaise. Generally, the clinical features of both chronic and acute osteomyelitis are the same, except that chronic infection can persist intermittently for years, flaring up spontaneously after minor trauma. Sometimes, however, the only symptom of chronic infection is the persistent drainage of pus from an old pocket in a sinus tract.

Diagnosis

Patient history, physical examination, and blood tests help to confirm osteomyelitis:

- ◆ WBC count shows leukocytosis.
- ◆ Erythrocyte sedimentation rate or C-reactive protein is usually elevated but nonspecific in acute cases.
- ◆ Cultures of the lesion indicate the source of the organism. Blood cultures help identify causative organism.
- ◆ MRI is best for detecting spinal infection.
- ◆ CT is best for visualizing islands of dead bone.

X-rays may not show bone involvement until the disease has been active for some time, usually 2 to 3 weeks. Bone scans can detect early infection. Diagnosis must rule out poliomyelitis, rheumatic fever, myositis, and bone fractures.



CONFIRMING DIAGNOSIS *The gold standard for diagnosing osteomyelitis is histopathologic and microscopic examination of bone.*

Treatment

Treatment for acute osteomyelitis should begin before definitive diagnosis. Treatment includes administration of antibiotics after blood cultures are taken; early surgical drainage to relieve pressure buildup and sequestrum formation; immobilization of the affected bone by a cast, traction, or bed rest; and supportive measures, such as analgesics and I.V. fluids.

If an abscess forms, treatment includes incision and drainage, followed by a culture of the drained fluid. Intracavitary instillation of antibiotics may be done through closed-system continuous irrigation with low intermittent suction; limited irrigation with blood drainage system with suction; or local application of packed, wet, antibiotic-soaked dressings.

In addition to these therapies, chronic osteomyelitis usually requires surgery to remove dead bone (*sequestrectomy*) and to promote drainage (*saucerization*). The area may be filled with bone graft or packing material to promote new bone tissue. An infected prosthesis is removed and a new one is implanted the same day or after resolution of the infection.

Some centers use hyperbaric oxygen to increase the activity of naturally occurring leukocytes. Free-tissue transfers and local muscle flaps are also used to fill in dead space and increase blood supply.

Special Considerations

Your major concerns are to control infection, protect the bone from injury, and offer meticulous supportive care.

- ◆ Use strict sterile technique when changing dressings and irrigating wounds. If the patient is in skeletal traction for compound fractures, cover insertion points of pin tracks with small, dry dressings, and instruct not to touch the skin around the pins and wires.
- ◆ Administer I.V. fluids to maintain adequate hydration as necessary. Provide a diet high in protein and vitamin C.
- ◆ Assess vital signs, wound appearance, and new pain, which may indicate secondary infection, daily.
- ◆ Carefully monitor suctioning equipment and the amount of solution it instills and suctions.
- ◆ Support the affected limb with firm pillows. Keep the limb level with the body; *don't* let it sag. Turn the patient gently every 2 hours and watch for signs of developing pressure ulcers. Report any signs of pressure ulcer formation immediately.
- ◆ Support the cast with firm pillows and smooth rough cast edges by petaling with pieces of adhesive tape or moleskin. Check circulation and drainage; if a wet spot appears on the cast, circle it with a marking pen, and note the time of appearance (on the cast). Be aware of how much drainage is expected. Check the circled spot at least every 4 hours and report any enlargement immediately.
- ◆ Protect the patient from mishaps, such as jerky movements and falls, which may threaten bone integrity. Report sudden pain, crepitus, or deformity immediately. Watch for any sudden malposition of the limb, which may indicate fracture.
- ◆ Provide emotional support and appropriate diversions. Before discharge, teach the patient how to protect and clean the wound and, most importantly, how to recognize signs of recurring infection (increased temperature, redness, localized heat, and swelling). Stress the need for follow-up examinations. Instruct the patient to seek prompt treatment for possible sources of recurrence—blisters, boils, styes, and impetigo.

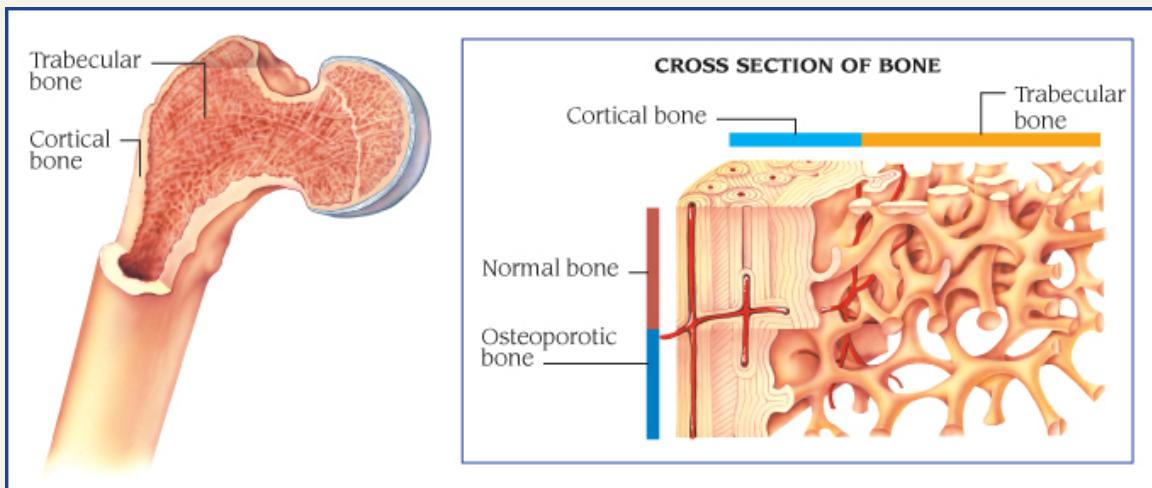
OSTEOPOROSIS

Osteoporosis is a metabolic bone disorder in which the rate of bone resorption accelerates while the rate of bone formation slows down, causing a loss of bone mass. Bones affected by this disease lose calcium and phosphate salts and thus become porous, brittle, and abnormally vulnerable to fractures. Osteoporosis may be primary or secondary to an underlying disease. Primary osteoporosis is commonly called *postmenopausal osteoporosis* because it typically develops in postmenopausal women. (See *What is osteoporosis*, page 309.)



PATHOPHYSIOLOGY WHAT IS OSTEOPOROSIS?

Osteoporosis is a metabolic disease of the skeleton that reduces the amount of bone tissue. Bones weaken as local cells resorb, or take up, bone tissue. Trabecular bone at the core becomes less dense, and cortical bone on the perimeter losses thickness.



Causes and Incidence

The cause of primary osteoporosis is unknown; however, a mild but prolonged negative calcium balance, resulting from an inadequate dietary intake of calcium, may be an important contributing factor—as may declining gonadal or adrenal function, faulty protein metabolism due to

estrogen deficiency, and sedentary lifestyle. (See *Preventing osteoporosis*, page 310.) Causes of secondary osteoporosis are many: prolonged therapy with steroids or heparin, cigarette smoking, total immobilization or disuse of a bone (as with hemiplegia, for example), alcoholism, malnutrition, malabsorption, celiac disease, scurvy, lactose intolerance, osteogenesis imperfecta, Sudeck atrophy (localized to hands and feet, with recurring attacks), and endocrine disorders (hypopituitarism, acromegaly, thyrotoxicosis, long-standing diabetes mellitus, hyperthyroidism).



PREVENTION PREVENTING OSTEOPOROSIS

To help prevent osteoporosis, tell the patient to follow these guidelines.

Maintain Adequate Calcium and Vitamin D Intake

Postmenopausal women and all men and women older than 65 years should consume 1,500 mg of calcium and at least 800 international units of vitamin D daily. Getting enough vitamin D is as important as getting enough calcium because vitamin D aids in absorption of calcium and improves muscle strength.

Most people get adequate amounts of vitamin D from sunlight; however, this may not be a good source for those who live in high latitudes, are housebound, or regularly use sunscreen or avoid the sun entirely because of the risk of skin cancer. Calcium supplements with added vitamin D are a good alternative.

Exercise

Exercise can help build strong bones and slow bone loss. Strength-training exercises should be combined with weight-bearing exercises. Strength training helps strengthen muscles and bones in the arms and upper spine, and weight-bearing exercises mainly affect the bones in the legs, hips, and lower spine.

Limit Alcohol Intake

Consuming more than two alcoholic drinks a day may decrease bone formation and reduce the body's ability to absorb calcium.

Limit Caffeine

Limit the amount of caffeinated beverages to about two to three cups of coffee a day. If the diet contains adequate calcium, moderate caffeine consumption won't harm you. Don't forget to count caffeine-containing beverages such as colas and teas.

The incidence of osteoporosis is high, with an estimated 10 million U.S. residents suffering from osteoporosis and another 18 million suffering from low bone mass, or osteopenia. Incidence is higher in women than in men, with women older than 50 years accounting for 20% of cases. Another 30% of women have osteopenia, which can deteriorate into osteoporosis.

Pathophysiology

In normal bone, the rates of bone formation and resorption are constant; replacement follows resorption immediately, and the amount of bone replaced equals the amount of bone resorbed. Osteoporosis develops when the remodeling cycle is interrupted, and new bone formation falls behind resorption.

When bone is resorbed faster than it forms, the bone becomes less dense. Men have about 30% greater bone mass than women, which may explain why osteoporosis develops later in men.

Complication

- ◆ Bone fractures, especially in vertebrae, femoral neck, and distal radius

Signs and Symptoms

Osteoporosis is usually discovered incidentally on X-ray; the patient may have been asymptomatic for years. Vertebral collapse, causing a backache with pain that radiates around the trunk, is the most common presenting feature. Any movement or jarring aggravates the backache.

In another common pattern, osteoporosis can develop insidiously, with increasing deformity, kyphosis, and loss of height. Sometimes a dowager hump is present. As bones weaken, spontaneous wedge fractures,

pathologic fractures of the neck or femur, Colles fractures after a minor fall, and hip fractures become increasingly common.



ELDER TIP Osteoporosis usually affects older people and is a major risk factor in vertebral compression fractures and hip fractures.

Osteoporosis primarily affects the weight-bearing vertebrae. Only when the condition is advanced or severe, as in Cushing syndrome or hyperthyroidism, do comparable changes occur in the skull, ribs, and long bones.

Diagnosis

Differential diagnosis must exclude other causes of rarefying bone disease, especially those affecting the spine, such as metastatic cancer and advanced multiple myeloma. The differential diagnosis should also exclude osteomalacia, osteogenesis imperfecta tarda, skeletal hyperparathyroidism, and hyperthyroidism. Initial evaluation attempts to identify the specific cause of osteoporosis through the patient history.

- ◆ Bone mineral density testing is performed in dual-energy X-ray absorptiometry (DEXA) and measures the mineralization of bones. It's the gold standard for evaluating osteoporosis.
- ◆ A spine CT scan shows demineralization. Quantitative CT can evaluate bone density but is less available and more expensive than DEXA.
- ◆ X-rays show fracture or vertebral collapse in severe cases.
- ◆ Urine calcium can provide evidence of bone turnover but is limited in value. Newer tests include urinary N-telopeptide to help diagnose osteoporosis.

Treatment

Treatment aims to slow down or prevent bone loss, prevent additional fractures, and control pain. A physical therapy program that emphasizes gentle exercise and activity is an important part of the treatment. Medications may include bisphosphonates, such as alendronate and risedronate, to prevent bone loss and reduce the risk of fractures. The physician may also recommend adequate calcium and vitamin D intake. Raloxifene and calcitonin have also been prescribed. Weakened vertebrae should be supported, usually with a back brace. Surgery can correct

pathologic fractures of the femur by open reduction and internal fixation. Colles fracture requires reduction with casting immobilization for 4 to 10 weeks.

The incidence of primary osteoporosis may be reduced through adequate intake of dietary calcium and regular exercise. Fluoride treatments may also offer some preventive benefit. Hormone replacement therapy (HRT) with estrogen and progesterone may retard bone loss and prevent the occurrence of fractures; however, this therapy remains controversial. HRT decreases bone reabsorption and increases bone mass. Secondary osteoporosis can be prevented through effective treatment of the underlying disease as well as corticosteroid therapy, early mobilization after surgery or trauma, careful observation for signs of malabsorption, and prompt treatment of hyperthyroidism. Men with osteoporosis, hypogonadism, and low libido may benefit from testosterone replacement therapy. Decreased alcohol consumption and caffeine use, as well as smoking cessation, are also helpful preventive measures.

Special Considerations

Your care plan should focus on the patient's fragility, stressing careful positioning, ambulation, and prescribed exercises.

- ◆ Check the patient's skin daily for redness, warmth, and new sites of pain, which may indicate new fractures. Encourage activity; help the patient walk several times daily. As appropriate, perform passive ROM exercises or encourage the patient to perform active exercises. Make sure the patient regularly attends scheduled physical therapy sessions.
- ◆ Impose safety precautions. Keep the side rails of the patient's bed in raised position. Move the patient gently and carefully always. Explain to the patient's family and ancillary health care personnel how easily an osteoporotic patient's bones can fracture.
- ◆ Provide a balanced diet, high in nutrients that support skeletal metabolism: vitamin D, calcium, and protein. Administer analgesics and heat to relieve pain.
- ◆ Make sure the patient and the family clearly understand the prescribed drug regimen. Tell them how to recognize significant adverse effects and to report them immediately. The patient should also report any new pain sites immediately, especially after trauma, no matter how slight. Advise

the patient to sleep on a firm mattress and avoid excessive bed rest. Ensure the patient knows how to wear the back brace.

- ◆ Thoroughly explain osteoporosis to the patient and the family. If the patient/family don't understand the nature of this disease, they may feel the fractures could have been prevented if they had been more careful.
- ◆ Teach the patient to use good body mechanics—to stoop before lifting anything and to avoid twisting movements and prolonged bending.

LEGG–CALVÉ–PERTHES DISEASE

Legg–Calv  –Perthes disease (also called *coxa plana*) is ischemic necrosis that leads to eventual flattening of the head of the femur caused by vascular interruption. The disease occurs in five stages.

- ◆ Growth arrest: Avascular phase; may last 6 to 12 months. Early changes include inflammation and synovitis of the hip and ischemic changes in the ossific nucleus of the femoral head.
- ◆ Subchondral fracture: Radiographic visualization of the fracture varies with the age of the child at clinical onset and the extent of epiphyseal involvement; may last 3 to 8½ months.
- ◆ Reabsorption, also called *fragmentation* or *necrosis*: The necrotic bone beneath the subchondral fracture is gradually and irregularly reabsorbed; lasts 6 to 12 months.
- ◆ Reossification, or healing stage: Ossification of the primary bone begins irregularly in the subchondral area and progresses centrally; takes 6 to 24 months.
- ◆ Healed stage, also called *residual stage*: Complete ossification of the epiphysis of the femoral head, with or without residual deformity.

Although this disease usually runs its course in 3 to 4 years, it may lead to premature osteoarthritis later in life from misalignment of the acetabulum and flattening of the femoral head.

Causes and Incidence

The exact vascular obstructive changes that initiate Legg–Calv  –Perthes disease are unknown. Current etiologic theories include venous obstruction with secondary intraepiphyseal thrombosis, trauma to retinacular vessels, vascular irregularities (congenital or developmental), vascular occlusion

secondary to increased intracapsular pressure from acute transient synovitis, and increased blood viscosity resulting in stasis and decreased blood flow.

Legg–Calvé–Perthes disease occurs most frequently in boys 4 to 10 years old and tends to occur in families. Although typically unilateral, it occurs bilaterally in 20% of patients.

Pathophysiology

The disease occurs in four stages. The first stage, synovitis, is characterized by synovial inflammation and increased joint fluid, and typically lasts 1 to 3 weeks. In the second (avascular) stage, vascular interruption causes necrosis of the ossification center of the femoral head (usually in several months to 1 year). In the third stage (which ordinarily lasts 2 to 4 years), revascularization, a new blood supply causes bone resorption and deposition of immature bone cells. New bone replaces necrotic bone and the femoral head gradually reforms. The final, or residual, stage involves healing and regeneration. Immature bone cells are replaced by normal bone cells, thereby fixing the joint's shape. There may or may not be residual deformity, based on the degree of necrosis that occurred in stage two.

Complications

- ◆ Permanent disability
- ◆ Premature osteoarthritis

Signs and Symptoms

The first indication of Legg–Calvé–Perthes disease is usually a persistent thigh pain or limp that becomes progressively severe. This symptom appears during the second stage, when bone resorption and deformity begin. Other effects may include mild pain in the hip, thigh, or knee that's aggravated by activity and relieved by rest; muscle spasm; atrophy of muscles in the upper thigh; slight shortening of the leg; and severely restricted abduction and internal rotation of the hip.

Diagnosis

 **CONFIRMING DIAGNOSIS** A thorough physical examination and clinical history suggest Legg–Calvé–Perthes disease. Hip X-rays confirm the diagnosis, with findings that vary according to the stage of the disease.

Anterior-posterior X-ray and MRI enhance early diagnosis of necrosis and visualization of articular surface.

Diagnostic evaluation must also differentiate between Legg-Calvé-Perthes disease (restriction of only the abduction and rotation of the hip) and infection or arthritis (restriction of all motion). Aspiration and culture of synovial fluid rule out joint sepsis.

Treatment

The aim of treatment is to protect the femoral head from further stress and damage by containing it within the acetabulum. After 1 to 2 weeks of bed rest, therapy may include reduced weight bearing by means of bed rest in bilateral split counterpoised traction, then application of hip abduction splint or cast, or weight bearing while a splint, cast, or brace holds the leg in abduction. Braces may remain in place for 6 to 18 months. Analgesics help relieve pain. Physical therapy with passive and active ROM exercises after cast removal helps restore motion.

For a young child in the early stages of the disease, osteotomy and subtrochanteric derotation provide maximum confinement of the epiphysis within the acetabulum to allow return of the femoral head to normal shape and full ROM. Proper placement of the epiphysis thus allows remolding with ambulation. Postoperatively, the patient requires a spica cast for about 2 months.

Special Considerations

When caring for the hospitalized child, do the following.

- ◆ Monitor the patient's fluid intake and output. Maintain sufficient fluid balance. Provide a diet sufficient for growth without causing excessive weight gain, which might necessitate cast change and loss of the corrective position.
- ◆ Provide cast care. Turn the child every 2 to 3 hours to expose the cast to air. When the cast is still wet, turn the child with your palms because depressions in the plaster may lead to pressure ulcers. After the cast dries, petal it with pieces of adhesive tape or moleskin, changing them as they become soiled. Protect the cast with a plastic covering during each bowel movement.

- ◆ Watch for circulatory or neurologic changes in the leg. Check toes for color, temperature, swelling, sensation, and motion; report dusky, cool, numb toes immediately. Check the skin under the cast with a flashlight every 4 hours while the patient is awake. Follow a consistent plan of skin care to prevent skin breakdown. *Never* use oils or powders under the cast because they increase skin breakdown and soften the cast. Check under the cast daily for odors, particularly after surgery, to detect skin breakdown or wound problems. Report persistent soreness.
- ◆ Relieve itching by using a hair dryer (set on cool) at the cast edges; this also decreases dampness from perspiration. If itching becomes excessive, get an order for an antipruritic. *Never* insert an object under the cast to scratch.
- ◆ Provide continuous emotional support. Explain all procedures and the need for bed rest, cast, or braces to the child; encourage the child to verbalize fears and anxiety. Encourage parents to participate in their child's care. Teach them proper cast care and how to recognize signs of skin breakdown. Offer tips for making home management of the bedridden child easier. Tell them what special supplies are needed: pajamas and trousers a size larger (open the side seam, and attach Velcro fasteners to close it), bedpan, adhesive tape, moleskin and, possibly, a hospital bed.
- ◆ When the cast is removed, debride dry, scaly skin *gradually* by applying lotion after bathing.
- ◆ Stress the need for follow-up care to monitor rehabilitation. Also stress home tutoring and socialization to promote normal mental and emotional growth and development.

OSGOOD–SCHLATTER DISEASE

Osgood–Schlatter disease, also called *osteochondrosis*, is a painful, incomplete separation of the epiphysis of the tibial tubercle from the tibial shaft. This is the common cause of knee pain in an adolescent. Severe disease may cause permanent tubercle enlargement.

Causes and Incidence

Osgood–Schlatter disease probably results from trauma before the complete fusion of the epiphysis to the main bone has occurred (between ages 10 and 15). Other causes include locally deficient blood supply and genetic factors.

It's most common in active adolescent boys, generally affecting one or both knees. It may occur in girls, typically between ages 10 and 11.

Pathophysiology

The proximal tibia has two ossification centers, the proximal tibial epiphysis and the tibial tuberosity, which are separated by a cartilage bridge. Before ossification, the tibial tuberosity is composed of fibrocartilage that has good tensile strength. However, during ossification, columnated cartilaginous cells with poor tensile strength replace the fibrocartilage, and it is within this small window between fibrocartilage and ossified matrix that the tibial tuberosity is at risk of avulsion fractures.

Complications

- ◆ Irregular growth
- ◆ Partial avascular necrosis of proximal tibial epiphysis

Signs and Symptoms

The patient complains of constant aching and pain and tenderness over the tibial tubercle, which worsens during any activity that causes forceful contraction of the patellar tendon on the tubercle, such as ascending or descending stairs, running, squatting, jumping, or forced flexion. The pain may be associated with some obvious soft-tissue swelling and localized heat and tenderness.

Diagnosis

Physical examination supports the diagnosis: the examiner forces the tibia into internal rotation while slowly extending the patient's knee from 90 degrees of flexion; at about 30 degrees, flexion produces pain that subsides immediately with external rotation of the tibia. Visible soft-tissue edema may be present over proximal tibial tuberosity with tenderness to palpation. Some patients' quadriceps may atrophy.

X-rays may be normal or show epiphyseal separation and soft-tissue swelling for up to 6 months after onset; eventually, they may show bone fragmentation. Bone scan may show increased uptake in the tibial tuberosity—even greater than the typical increased uptake in the normal epiphysis of the unaffected side.

Treatment

Osteochondrosis is usually self-limiting, and conservative treatment designed to reduce pain and decrease stress to the affected knee is usually adequate. Avoid strenuous exercises that involve the knee; use frequent ice applications after exercise for pain. Rest and quadriceps strengthening, hip extension, adductor strengthening, and hamstring and quadriceps-stretching exercises are recommended. Knee immobilization in extension for 6 to 8 weeks may be necessary. Analgesics and NSAIDs may be given for pain relief and reduction of local swelling.

Rarely, conservative measures fail, and surgery may be necessary. Such surgery includes removal or fixation of the epiphysis or drilling holes through the tubercle to the main bone to form channels for rapid revascularization.

Special Considerations

The following special considerations should be observed for patients with Osgood–Schlatter disease:

- ◆ Monitor the patient's circulation, sensation, and pain, and watch for excessive bleeding after surgery.
- ◆ Assess daily for limitation of motion. Administer analgesics as needed.
- ◆ Make sure knee support or splint isn't too tight. Keep the cast dry and clean, and petal it around the top and bottom margins to avoid skin irritation. Teach proper use of crutches. Tell the patient to protect the injured knee with padding and to avoid trauma and repeated flexion (running, contact sports).
- ◆ Monitor for muscle atrophy.
- ◆ Give reassurance and emotional support because disruption of normal activities is difficult for an active teenager. Emphasize that restrictions are temporary.

PAGET DISEASE

Paget disease, also called *osteitis deformans*, is a slowly progressive metabolic bone disease characterized by an initial phase of excessive bone resorption (osteoclastic phase), followed by a reactive phase of excessive abnormal bone formation (osteoblastic phase). The new bone structure, which is chaotic, fragile, and weak, causes painful deformities of both

external contour and internal structure. Paget disease usually localizes in one or more areas of the skeleton, but occasionally skeletal deformity is widely distributed. The bones most frequently affected are pelvis, leg, spine, arm, collar, and skull. It can be fatal, particularly when it's associated with heart failure (widespread disease creates a continuous need for high cardiac output), bone sarcoma, or giant-cell tumors.

Causes and Incidence

The disease occurs worldwide, but is more common in Europe, Australia, and New Zealand, where it's seen in up to 5% of the elderly population. The incidence is higher in men than in women and usually occurs in patients older than 40 years. Although its exact cause is unknown, one theory holds that early viral infection causes a dormant skeletal infection that erupts many years later as Paget disease. Genetic factors are also suspected.

Pathophysiology

Repeated episodes of accelerated osteoclastic resorption of spongy bone occur. The trabeculae diminish, and vascular fibrous tissue replaces marrow. This is followed by short periods of rapid, abnormal bone formation. The collagen fibers in this new bone are disorganized, and glycoprotein levels in the matrix decrease. The partially resorbed trabeculae thicken and enlarge because of excessive bone formation, and the bone becomes soft and weak.

Eventually, Paget disease progresses to an inactive phase in which abnormal remodeling is minimal or absent.

Complications

- ◆ Fractures
- ◆ Vertebral collapse
- ◆ Paraplegia
- ◆ Blindness and hearing loss (impingement on cranial nerves)
- ◆ Osteoarthritis
- ◆ Sarcoma
- ◆ Hypertension
- ◆ Renal calculi
- ◆ Hypercalcemia
- ◆ Gout

Signs and Symptoms

Clinical effects of Paget disease vary. Early stages may be asymptomatic, but when pain does develop, it's usually severe and persistent and may coexist with impaired movement resulting from impingement of abnormal bone on the spinal cord or sensory nerve root. Such pain intensifies with weight bearing.

The patient with skull involvement shows characteristic cranial enlargement over frontal and occipital areas (hat size may increase) and may complain of headaches. Other deformities include kyphosis (spinal curvature due to compression fractures of pagetic vertebrae), accompanied by a barrel-shaped chest and asymmetrical bowing of the tibia and femur, which commonly reduces height. Pagetic sites are warm and tender and are susceptible to pathologic fractures after minor trauma. Pagetic fractures heal slowly and usually incompletely.

Bony impingement on the cranial nerves may cause blindness and hearing loss with tinnitus and vertigo. Other complications include hypertension, renal calculi, hypercalcemia, gout, heart failure, a waddling gait (from softening of pelvic bones), and hearing loss.

Diagnosis

X-rays taken before overt symptoms develop show increased bone expansion and density. A bone scan, which is more sensitive than X-rays, clearly shows early pagetic lesions (radioisotope collects around areas of active disease). CT scan or MRI shows extra bony extension if sarcomatous degeneration occurs. Bone biopsy reveals characteristic mosaic pattern.

Other laboratory findings include:

- ◆ elevated serum alkaline phosphatase levels (an index of osteoblastic activity and bone formation)
- ◆ elevated serum calcium

Increasing use of routine chemistry screens (including serum alkaline phosphatase) is making early diagnosis more common. Serum osteocalcin and N-telopeptide are usually increased.

Treatment

Primary treatment consists of drug therapy and includes one of the following:

- ◆ Calcitonin (subcutaneously or intranasally) is used to retard bone resorption (which relieves bone lesions) and reduce levels of serum alkaline phosphate and urinary hydroxyproline secretion. Although calcitonin therapy requires long-term maintenance, improvement is noticeable after the first few weeks of treatment.
- ◆ Bisphosphonates, such as alendronate, ibandronate, pamidronate, risedronate, and zoledronic acid, produce rapid reduction in bone turnover and relieve pain. They also reduce serum alkaline phosphate and urinary hydroxyproline secretion. Therapy produces noticeable improvement after 1 to 3 months.
- ◆ Plicamycin, a cytotoxic antibiotic, is used to decrease calcium, urinary hydroxyproline, and serum alkaline phosphatase. It produces remission of symptoms within 2 weeks and biochemical improvement in 1 to 2 months. Plicamycin is used to control the disease and is reserved for severe cases with neurologic compromise and for those resistant to other therapies. However, it may destroy platelets or compromise renal function.

Orthopedic surgery is used to correct specific deformities in severe cases, reduce or prevent pathologic fractures, correct secondary deformities, or relieve neurologic impairment. Joint replacement is difficult because bonding material (methyl methacrylate) doesn't set properly on pagetic bone.

Other treatment varies according to symptoms. Analgesics or NSAIDs may be given to control pain.

Special Considerations

Patients with Paget disease require the following special considerations:

- ◆ To evaluate the effectiveness of analgesics, assess level of pain daily. Watch for new areas of pain or restricted movements, which may indicate new fracture sites, and sensory or motor disturbances, such as difficulty in hearing, seeing, or walking.
- ◆ Monitor serum calcium and alkaline phosphatase levels.
- ◆ If the patient is confined to prolonged bed rest, prevent pressure ulcers by providing good skin care. Reposition the patient frequently and use a flotation mattress. Provide high-topped sneakers to prevent footdrop.

- ◆ Monitor intake and output. Encourage adequate fluid intake to minimize renal calculi formation.
- ◆ Demonstrate how to inject calcitonin and rotate injection sites properly or how to perform nasal inhalation of the drug if that's the form prescribed. Warn the patient that adverse effects may occur (nausea, vomiting, local inflammatory reaction at injection site, facial flushing, itching of hands, and fever). Give reassurance that these adverse effects are usually mild and infrequent.
- ◆ To help the patient adjust to the changes in lifestyle imposed by this disease, teach how to pace activities and, if necessary, how to use assistive devices. Encourage the patient to follow a recommended exercise program, avoiding both immobilization and excessive activity. Suggest a firm mattress or a bed board to minimize spinal deformities. Warn against imprudent use of analgesics because diminished sensitivity to pain resulting from analgesic use may make the patient unaware of new fractures. To prevent falls at home, advise removal of throw rugs and other obstacles.
- ◆ Help the patient and family make use of community support resources, such as a visiting nurse or home health agency. For more information, refer them to the Paget's Disease Foundation.

HALLUX VALGUS

Hallux valgus is a lateral deviation of the great toe at the metatarsophalangeal joint. It occurs with medial enlargement of the first metatarsal head and bunion formation (bursa and callus formation at the bony prominence).

Causes and Incidence

Hallux valgus may be acquired or congenital. Acquired hallux valgus results from degenerative arthritis or prolonged pressure on the foot, especially from narrow-toed or high-heeled shoes that compress the forefoot. Bony alignment is normal at the outset of the disorder. This form typically occurs more frequently in women.

In congenital hallux valgus, abnormal bony alignment—increased space between first and second metatarsal (metatarsus primus varus)—causes bunion formation. This form is usually first observed in childhood.

Pathophysiology

During the gait cycle, the hallux and digits generally remain parallel to the long axis of the foot, regardless of the degree of forefoot abduction (or pronation) occurring. This is because of the pull of the conjoined adductor tendon, extensor hallucis longus, and flexor hallucis longus tendons. The tendons gain greater mechanical advantage the further the joint is displaced, with tension created in the medial aspect of the joint and compression laterally.

Line of pull of extensor hallucis longus causing metatarsal to deviate medially and hallux to deviate laterally.

Medial tension causes the medial collateral ligaments to pull on the dorsomedial aspect of the first metatarsal head, causing bone proliferation. Lateral tension causes the sesamoid apparatus to fixate in a laterally dislocated position. Remodeling also occurs laterally in addition to medially, as evidenced by the increase in the proximal articular set angle or structural remodeling of the cartilage. Therefore, without correction of the biomechanical factors, excessive pronation continues, with propagation of the deformity.

Complications

- ◆ Foot deformity
- ◆ Difficulty walking

Signs and Symptoms

Hallux valgus characteristically begins as a tender bunion covered by deformed, hard, erythematous skin and palpable bursa, typically distended with fluid. The first indication of hallux valgus may be pain over the bunion from shoe pressure. Pain can also stem from traumatic arthritis, bursitis, or abnormal stresses on the foot because hallux valgus changes the body's weight-bearing pattern. In an advanced stage, a flat, splayed forefoot may occur, with severely curled toes (*hammer toes*) and formation of a small bunion on the fifth metatarsal. (See *Hammer toe*.)

Hammer Toe

In hammer toe, the toe assumes a clawlike appearance from hyperextension of the metatarsophalangeal joint, flexion of the proximal interphalangeal joint, and hyperextension of the distal interphalangeal joint, usually under pressure from hallux valgus displacement. A painful corn forms on the back of the interphalangeal joint and on the bone end, and a callus forms on the sole of the foot, both of which make walking painful. Hammer toe may be mild or severe and can affect one toe or all five, as in clawfoot (which also causes a very high arch).

Hammer toe can be congenital (and familial) or acquired from constantly wearing short, narrow shoes, which put pressure on the end of the long toe. Acquired hammer toe is commonly bilateral and often develops in children who rapidly outgrow shoes and socks.

In young children, or adults with early deformity, repeated foot manipulation and splinting of the affected toe relieve discomfort and may correct the deformity. Other treatment includes protection of protruding joints with felt pads, corrective footwear (open-toed shoes and sandals or special shoes that conform to the shape of the foot), the use of a metatarsal arch support, and exercises, such as passive manual stretching of the proximal interphalangeal joint. Severe deformity requires surgical fusion of the proximal interphalangeal joint in a straight position.

Diagnosis



CONFIRMING DIAGNOSIS *A red, tender bunion makes hallux valgus obvious. X-rays confirm the diagnosis by showing medial deviation of the first metatarsal and lateral deviation of the great toe.*

Treatment

In the very early stages of acquired hallux valgus, good foot care and wide-toed shoes may eliminate the need for further treatment. Other useful measures for early management include felt pads to protect the bunion, foam pads or other devices to separate the first and second toes at night, and a supportive pad and exercises to strengthen the metatarsal arch. Early treatment is vital in patients predisposed to foot problems, such as those

with rheumatoid arthritis or diabetes mellitus. If the disease progresses to severe deformity with disabling pain, bunionectomy is necessary.

After surgery, the toe is immobilized in its corrected position in one of two ways: with a soft compression dressing that may cover the entire foot or just the great toe and the second toe, thereby serving as a splint, or with a short cast such as a light slipper spica cast.

The patient may need crutches or controlled weight bearing. Depending on the extent of the surgery, some patients walk on their heels a few days after surgery; others must wait 4 to 6 weeks to bear weight on the affected foot. Supportive treatment may include physical therapy, such as warm compresses, soaks, and exercises, and analgesics to relieve pain and stiffness.

Special Considerations

Before surgery, obtain a patient history and assess the neurovascular status of the foot (temperature, color, sensation, and blanching sign). If necessary, teach the patient how to walk with crutches.

After bunionectomy:

- ◆ Apply ice to reduce swelling. Support the patient's foot with pillows, elevate the foot of the bed, or put the bed in Trendelenburg position.
- ◆ Record the neurovascular status of the toes, including the patient's ability to move the toes (dressing may inhibit movement), every hour for the first 24 hours and then every 4 hours. Report any change in neurovascular status to the surgeon immediately.
- ◆ Prepare the patient for walking by instructing to dangle the affected foot over the side of the bed for a short time before standing, allowing a gradual increase in venous pressure. If crutches are needed, supervise the patient in using them, and make sure this skill is mastered before discharge. The patient should have a proper cast shoe or boot to protect the cast or dressing.
- ◆ Before discharge, instruct the patient to limit activities, to rest frequently with feet elevated, to elevate feet whenever pain or swelling is experienced, and to wear wide-toed shoes and sandals after the dressings are removed. Urge female patients not to resume wearing high-heeled, pointy-toed shoes.

- ◆ Teach proper foot care, such as cleanliness, massages, and cutting toenails straight across to prevent ingrown nails and infection.
- ◆ Suggest exercises to do at home to strengthen foot muscles, such as standing at the edge of a step on the heel and then raising and inverting the top of the foot.
- ◆ Stress the importance of follow-up care and prompt medical attention for painful bunions, corns, and calluses.

KYPHOSIS

Kyphosis, also called *roundback* or *hunchback*, is an anteroposterior curving of the spine that causes a bowing of the back, commonly at the thoracic, but sometimes at the thoracolumbar or sacral, level. The normal spine displays some convexity, but excessive thoracic kyphosis is pathologic.

Causes and Incidence

Kyphosis occurs in children and adults. Although congenital kyphosis is rare, it's usually severe, with resultant cosmetic deformity and reduced pulmonary function.

Adolescent kyphosis (also called *Scheuermann disease*, *juvenile kyphosis*, and *vertebral epiphysitis*), the most common form of this disorder, may result from growth retardation or a vascular disturbance in the vertebral epiphysis (usually at the thoracic level) during periods of rapid growth or from congenital deficiency in the thickness of the vertebral plates. Other causes include infection, inflammation, aseptic necrosis, and disk degeneration. The subsequent stress of weight bearing on the compromised vertebrae may result in the thoracic hump commonly seen in adolescents with kyphosis. Symptomatic adolescent kyphosis is more prevalent in girls than in boys and occurs most commonly between ages 12 and 16.

Adult kyphosis (adult roundback) may result from aging and associated degeneration of intervertebral disks, atrophy, and osteoporotic collapse of the vertebrae; from endocrine disorders, such as hyperparathyroidism and Cushing disease; and from prolonged steroid therapy. Adult kyphosis may also result from conditions such as arthritis, Paget disease, polio, compression fracture of the thoracic vertebrae, metastatic tumor, plasma

cell myeloma, or tuberculosis (TB). In both children and adults, kyphosis may also result from poor posture.

Disk lesions called *Schmorl nodes* may develop in anteroposterior curving of the spine and are localized protrusions of nuclear material through the cartilage plates and into the spongy bone of the vertebral bodies. If the anterior portions of the cartilage are destroyed, bridges of new bone may transverse the intervertebral space, causing ankylosis.

Pathophysiology

The pathophysiology of kyphosis depends on the etiologic factor. The exact cause of Scheuermann disease is still imprecisely defined. Scheuermann postulated that the condition resulted from avascular necrosis of the apophyseal ring. Other theories include histologic abnormalities at the endplate, osteoporosis, and mechanical factors that affect spinal growth. A Danish study demonstrated an important genetic component to the entity.

Postural kyphosis is present when accentuated kyphosis is observed without the characteristic 5 degrees of wedging over three consecutive vertebral segments that defines Scheuermann kyphosis. This is felt to be due to muscular imbalance leading to the roundback appearance of these individuals.

When focal kyphosis occurs after a fracture, more height is lost in the anterior aspect than in the posterior aspect; this is the typical fracture pattern. The angulation can increase as the fracture heals, placing pressure on the spinal cord. Patients with fractures have historically been treated with laminectomy alone, especially in the thoracic spine, and they often had progressive kyphosis at the fracture site.

Postinfectious kyphosis occurs in a manner similar to that just described. The mechanical integrity of the anterior column is lost as a consequence of the infectious process. Bending forces then accentuate the normal sagittal contour.

Complications

- ◆ Debilitating back pain
- ◆ Leg weakness or paralysis
- ◆ Decreased lung capacity

Signs and Symptoms

Development of adolescent kyphosis is usually insidious and may be asymptomatic except for the obvious curving of the back (sometimes more than 90 degrees). In some adolescents, kyphosis may produce mild pain at the apex of the curve (about 50% of patients), fatigue, tenderness or stiffness in the involved area or along the entire spine, and prominent vertebral spinous processes at the lower dorsal and upper lumbar levels, with compensatory increased lumbar lordosis, and hamstring tightness. Rarely, kyphosis may cause neurologic damage: spastic paraparesis secondary to spinal cord compression or herniated nucleus pulposus. In both adolescent and adult forms of kyphosis that aren't due to poor posture alone, the spine won't straighten when the patient assumes a recumbent position.

Adult kyphosis produces a characteristic roundback appearance, possibly associated with pain, weakness of the back, and generalized fatigue. Unlike the adolescent form, adult kyphosis rarely produces local tenderness, except in osteoporosis with a recent compression fracture.

Diagnosis

Physical examination reveals curvature of the thoracic spine in varying degrees of severity. X-rays may show vertebral wedging, Schmorl nodes, irregular end plates, and possibly mild scoliosis of 10 to 20 degrees. MRI should be used to distinguish adolescent kyphosis from TB and other inflammatory or neoplastic diseases that cause vertebral collapse; the severe pain, bone destruction, or systemic symptoms associated with these diseases help rule out a diagnosis of kyphosis. Other sites of bone disease, primary sites of malignancy, and infection must also be evaluated, possibly through vertebral biopsy.

Treatment

For kyphosis caused by poor posture alone, treatment may consist of therapeutic exercises, bed rest on a firm mattress (with or without traction), and a brace to straighten the kyphotic curve until spinal growth is complete. Corrective exercises include pelvic tilt to decrease lumbar lordosis, hamstring stretch to overcome muscle contractures, and thoracic hyperextension to flatten the kyphotic curve. These exercises may be performed in or out of the brace. Lateral X-rays taken every 4 months evaluate correction. Gradual weaning from the brace can begin after

maximum correction of the kyphotic curve and after vertebral wedging has decreased and the spine has reached full skeletal maturity. Loss of correction indicates that weaning from the brace has been too rapid, and time out of the brace is decreased accordingly.

Treatment for both adolescent and adult kyphosis also includes appropriate measures for the underlying cause and, possibly, spinal arthrodesis for relief of symptoms. Although rarely necessary, surgery may be recommended when kyphosis causes neurologic damage, a spinal curve greater than 60 degrees, or intractable and disabling back pain in a patient with full skeletal maturity. Preoperative measures may include halo-femoral traction. Corrective surgery includes a posterior spinal fusion with spinal instrumentation, iliac bone grafting, and plaster immobilization. Anterior spinal fusion followed by immobilization in plaster may be necessary when kyphosis produces a spinal curve greater than 70 degrees.

Special Considerations

Effective management of kyphosis necessitates first-rate supportive care for patients in traction or a brace, skillful patient teaching, and sensitive emotional support.

- ◆ Teach the patient with adolescent kyphosis caused by poor posture alone the prescribed therapeutic exercises and the fundamentals of good posture. Suggest bed rest when pain is severe. Encourage use of a firm mattress, preferably with a bed board. If the patient needs a brace, explain its purpose, how and when to wear it.
- ◆ Teach good skin care. Tell the patient not to use lotions, ointments, or powders where the brace contacts the skin. Provide instructions that only the physician or orthotist should adjust the brace.
- ◆ If corrective surgery is needed, explain all preoperative tests thoroughly as well as the need for postoperative traction or casting, if applicable. After surgery, check neurovascular status every 2 to 4 hours for the first 48 hours, and report any changes immediately. Turn the patient often by logrolling and teach the patient how to logroll independently. Provide meticulous skin care. Check the skin at the cast edges several times a day; use heel and elbow protectors to prevent skin breakdown. Remove antiembolism stockings, if ordered, at least three times a day for at least 30 minutes. Change dressings as ordered.

- ◆ Provide emotional support. The adolescent patient is likely to exhibit mood changes and periods of depression. Maintain communication and offer frequent encouragement and reassurance.
- ◆ Assist during removal of sutures and application of a new cast (usually about 10 days after surgery). Encourage gradual ambulation (usually with the use of a tilt table in the physical therapy department).
- ◆ At discharge, provide detailed, written cast care instructions. Tell the patient to immediately report pain, burning, skin breakdown, loss of feeling, tingling, numbness, or cast odor. Advise the patient to drink plenty of liquids to avoid constipation and to report any illness immediately. Arrange for home visits by a social worker and a home care nurse, as needed.

HERNIATED DISK

Herniated disk, also called *ruptured* or *slipped disk* and *herniated nucleus pulposus*, occurs when all or part of the nucleus pulposus—the soft, gelatinous, central portion of an intervertebral disk—is forced through the disk's weakened or torn outer ring (*anulus fibrosus*). When this happens, the extruded disk may impinge on spinal nerve roots as they exit from the spinal canal or on the spinal cord itself, resulting in back pain and other signs of nerve root irritation.

Causes and Incidence

Herniated disks may result from severe trauma or strain or may be related to intervertebral joint degeneration. Although herniated disks usually occur in adults (mostly men) younger than 45 years old, elderly people are also at risk because minor trauma may cause herniation in disks that have begun to deteriorate due to age. Ninety percent of herniation occurs in the lumbar and lumbosacral regions of the spine; 8% in the cervical region; and 1% to 2% in the thoracic region. Patients with a congenitally small lumbar spinal canal or with osteophyte formation on the vertebrae may be more susceptible to nerve root compression by a herniated disk and more likely to have neurologic symptoms.

Pathophysiology

An intervertebral disk has two parts: the soft center called the *nucleus pulposus* and the tough, fibrous surrounding ring called the *anulus fibrosus*.

The nucleus pulposus acts as a shock absorber, distributing the mechanical stress applied to the spine when the body moves.

Physical stress, usually a twisting motion, can tear or rupture the anulus fibrosus so that the nucleus pulposus herniates into the spinal canal. When this happens, the extruded disk may impinge on spinal nerve roots as they exit from the spinal canal or on the spinal cord itself, resulting in back pain and other signs of nerve root irritation. The vertebrae move closer together and in turn exert pressure on the nerve roots as they exit between the vertebrae. Pain and possibly sensory and motor loss follow. A herniated disk can also follow intervertebral joint degeneration; minor trauma may cause herniation.

Herniation occurs in three steps:

- ◆ *protrusion*—nucleus pulposus presses against the anulus fibrosus
- ◆ *extrusion*—nucleus pulposus bulges forcibly through the anulus fibrosus, pushing against the nerve root
- ◆ *sequestration*—anulus fibrosis gives way as the disk's core bursts and presses against the nerve root.

Complications

- ◆ Long-term back pain
- ◆ Rarely, spinal cord injuries, resulting in loss of movement or sensation in legs and feet or loss of bowel and bladder function

Signs and Symptoms

The overriding symptom of lumbar herniated disk is severe low back pain that radiates to the buttocks, legs, and feet, usually unilaterally. When herniation follows trauma, the pain may begin suddenly, subside in a few days, and then recur at shorter intervals and with progressive intensity. Sciatic pain follows, beginning as a dull pain in the buttocks. Valsalva's maneuver, coughing, sneezing, or bending intensifies the pain, which is commonly accompanied by muscle spasms. Herniated disk may also cause paresthesias or hyperesthesia, as well as sensory and motor loss in the area innervated by the compressed spinal nerve root and, in later stages, weakness and atrophy of leg muscles.

Diagnosis

Obtaining a careful patient history is vital because the events that intensify disk pain are diagnostically significant. The straight-leg-raising test and its variants are perhaps the best tests for herniated disk but may still be negative.

For the straight-leg-raising test, the patient lies in a supine position while the examiner places one hand on the patient's ilium, to stabilize the pelvis, and the other hand under the ankle, then slowly raises the patient's leg. The test is positive only if the patient complains of posterior leg (sciatic) pain, not back pain. In Lasègue's test, the patient lies flat while the thigh and knee are flexed to a 90-degree angle. Resistance and pain as well as loss of ankle or knee-jerk reflex indicate spinal root compression.

X-rays of the spine are essential to rule out other abnormalities but may not diagnose herniated disk because marked disk prolapse can be present despite a normal X-ray. A thorough check of the patient's peripheral vascular status—including posterior tibial and dorsalis pedis pulses and skin temperature of limbs—helps rule out ischemic disease, another cause of leg pain or numbness. After physical examination and X-rays, myelography, CT scans, and MRI provide the most specific diagnostic information, showing spinal canal compression by herniated disk material. MRI is the method of choice to confirm the diagnosis and determine the exact level of herniation. A myelogram can define the size and location of disk herniation. An electromyogram can determine the exact nerve root involved. A nerve conduction velocity test may also be performed.

Treatment

Unless neurologic impairment progresses rapidly, treatment is initially conservative and consists of several weeks of bed rest (possibly with pelvic traction), administration of NSAIDs, heat applications, and an exercise program. Epidural corticosteroids, short-term oral corticosteroids, nerve root blocks, or physical therapy may be used to decrease pain. Muscle relaxants, such as diazepam, methocarbamol, or cyclobenzaprine, may relieve associated muscle spasms.

A herniated disk that fails to respond to conservative treatment may necessitate surgery. The most common procedure, laminectomy, involves excision of a portion of the lamina and removal of the protruding disk. If laminectomy doesn't alleviate pain and disability, a spinal fusion may be necessary to overcome segmental instability. Laminectomy and spinal

fusion are sometimes performed concurrently to stabilize the spine. Microdiscectomy can also be used to remove fragments of nucleus pulposus.

Injection of the enzyme chymopapain into the herniated disk produces a loss of water and proteoglycans from the disk, thereby reducing both the disk's size and the pressure in the nerve root.

Special Considerations

Herniated disk requires supportive care, careful patient teaching, and strong emotional support to help the patient cope with the discomfort and frustration of chronic low back pain.

- ◆ If the patient requires myelography, question carefully about allergies to iodides, iodine-containing substances, or seafood because such allergies may indicate sensitivity to the test's radiopaque dye. Reinforce previous explanations of the need for this test and tell the patient to expect some pain. Provide assurance that a sedative will be provided before the test, if needed, to promote comfort and lessen anxiety. After the test, urge the patient to remain in bed with the head elevated (especially if metrizamide was used) and to drink plenty of fluids. Monitor intake and output. Watch for seizures and allergic reaction.
- ◆ During conservative treatment, watch for any deterioration in neurologic status (especially during the first 24 hours after admission), which may indicate an urgent need for surgery. Use antiembolism stockings as prescribed, and encourage the patient to move the legs, as allowed. Provide high-topped sneakers to prevent footdrop. Work closely with the physical therapy department to ensure a consistent regimen of leg- and back-strengthening exercises. Give plenty of fluids to prevent renal stasis, and remind the patient to cough, deep breathe, and use blow bottles or an incentive spirometer to preclude pulmonary complications. Provide good skin care. Assess for bowel and bladder functions. Use a fracture bedpan for the patient on complete bed rest.
- ◆ After laminectomy, microdiscectomy, or spinal fusion, enforce bed rest, as indicated. If a blood drainage system (Hemovac or Jackson-Pratt drain) is in use, check the tubing frequently for kinks and a secure vacuum. Empty the Hemovac at the end of each shift and record the amount and color of drainage. Report colorless moisture on dressings (possible cerebrospinal fluid leakage) or excessive drainage immediately.

Observe neurovascular status of the legs (color, motion, temperature, and sensation).

- ◆ Monitor vital signs and check for bowel sounds and abdominal distention. Use logrolling technique to turn the patient. Administer analgesics as ordered, especially 30 minutes before initial attempts at sitting or walking. Give the patient assistance during the first attempt to walk. Provide a straight-backed chair for limited sitting.
- ◆ Teach the patient who has undergone spinal fusion how to wear a brace. Assist with straight-leg-raising and toe-pointing exercises, as indicated. Before discharge, teach proper body mechanics—bending at the knees and hips (never at the waist), standing straight, and carrying objects close to the body. Advise the patient to lie down when tired and to sleep on either side (never on abdomen) on an extra-firm mattress or a bed board. Urge maintenance of proper weight to prevent lordosis caused by obesity.
- ◆ After chemonucleolysis, enforce bed rest as ordered. Administer analgesics and apply heat, as needed. Urge the patient to cough and deep breathe. Assist with physical therapy as necessary and advise the patient to continue these exercises after discharge.
- ◆ Tell the patient who must receive a muscle relaxant of possible adverse effects, especially drowsiness. Provide instruction to avoid activities that require alertness until the effects to the medication are known, and a tolerance has been built up.
- ◆ Provide emotional support. Try to cheer the patient up during periods of frustration and depression. Provide reassurance of progress, and offer encouragement. (See *Preventing a herniated disk*, page 321.)



PREVENTION PREVENTING A HERNIATED DISK

To prevent a herniated disk, tell your patient to follow these guidelines.

Exercise

Getting regular exercise can slow the degeneration of the disks related to aging. Muscle strength gained through exercising can

strengthen and stabilize the spine. If the patient has previously had a herniated disk, he should remember to avoid high-impact activities such as jogging, tennis, and high-impact aerobics for the first few months after a herniated disk.

Maintain Good Posture

Good posture reduces the pressure on the spine and disks. Keeping the back straight and aligned is essential, particularly when sitting for longer periods. Also, heavy objects should be lifted properly by letting the legs—not the back—do most of the work.

Maintain a Healthy Weight

Excess weight puts more pressure on the spine and disks, making them more susceptible to a herniation.

SCOLIOSIS

Scoliosis is a lateral curvature of the spine that may occur in the thoracic, lumbar, or thoracolumbar spinal segment. The curve may be convex to the right (more common in thoracic curves) or to the left (more common in lumbar curves). Rotation of the vertebral column around its axis occurs and may cause rib cage deformity. Scoliosis is commonly associated with kyphosis (*roundback*) and lordosis (*swayback*).

Causes and Incidence

Scoliosis may be functional, structural, or idiopathic. Functional (postural) scoliosis usually results from a discrepancy in leg lengths rather than from a fixed deformity of the spinal column; it corrects when the patient bends toward the convex side. Structural scoliosis results from a deformity of the vertebral bodies, and it doesn't correct when the patient bends to the side. Structural scoliosis may be:

- ◆ *congenital*: usually related to a congenital defect, such as wedge vertebrae, fused ribs or vertebrae, or hemivertebrae; may result from trauma to zygote or embryo
- ◆ *paralytic or musculoskeletal*: develops several months after asymmetrical paralysis of the trunk muscles due to polio, cerebral palsy,

or muscular dystrophy

- ◆ *idiopathic (the most common form)*: may be transmitted as an autosomal dominant or multifactorial trait. This form appears in a previously straight spine during the growing years. Brainstem dysfunction, possibly due to a lesion of the posterior columns or the inner ear, may be the cause



PEDIATRIC TIP *Idiopathic scoliosis can be classified as infantile, which affects mostly male infants between birth and age 3 and causes left thoracic and right lumbar curves; juvenile, which affects both sexes between ages 4 and 10 and causes varying types of curvature; or adolescent, which generally affects girls between age 10 and achievement of skeletal maturity and causes varying types of curvature.*

Pathophysiology

Differential stress on vertebral bone causes an imbalance of osteoblastic activity; thus, the curve progresses rapidly during adolescent growth spurt. Without treatment, the imbalance continues into adulthood.

Complications

- ◆ Debilitating back pain
- ◆ Reduced pulmonary function
- ◆ Cor pulmonale

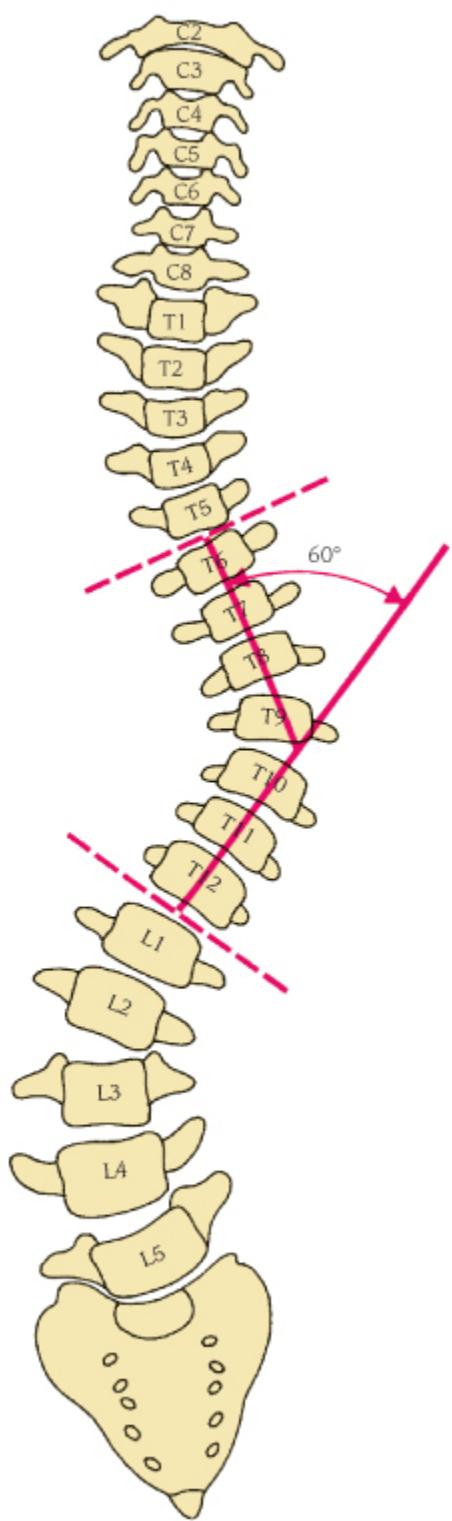
Signs and Symptoms

The most common curve in functional or structural scoliosis arises in the thoracic segment, with convexity to the right, and compensatory curves (S curves) in the cervical segment above and the lumbar segment below, both with convexity to the left. (See *Cobb method for measuring angle of curvature*, page 322.) As the spine curves laterally, compensatory curves develop to maintain body balance and mark the deformity. Scoliosis rarely produces subjective symptoms until it's well established; when symptoms do occur, they include backache, fatigue, and dyspnea. Because many teenagers are shy about their bodies, their parents suspect that something is wrong only after they notice uneven hemlines, pant legs that appear unequal in length, or subtle physical signs like one hip appearing higher than the

other. Untreated scoliosis may result in pulmonary insufficiency (curvature may decrease lung capacity), back pain, degenerative arthritis of the spine, disk disease, and sciatica.

Cobb Method for Measuring Angle of Curvature

The Cobb method measures the angle of curvature in scoliosis. The top vertebra in the curve (T6 in the illustration) is the uppermost vertebra whose upper face tilts toward the curve's concave side. The bottom vertebra in the curve (T12) is the lowest vertebra whose lower face tilts toward the curve's concave side. The angle at which perpendicular lines drawn from the upper face of the top vertebra and the lower face of the bottom vertebra intersect is the angle of the curve.



Diagnosis



CONFIRMING DIAGNOSIS *Anterior, posterior, and lateral spinal X-rays, taken with the patient standing upright and bending, confirm scoliosis and determine the degree of curvature (Cobb method) and flexibility of the spine.*

A scoliometer can also be used to measure the angle of trunk rotation. Physical examination reveals unequal shoulder heights, elbow levels, and heights of the iliac crests. Muscles on the convex side of the curve may be rounded and those on the concave side flattened, producing asymmetry of paraspinal muscles.

Treatment

Only two treatments effectively treat scoliosis: spinal bracing and surgery. If monitored closely, a properly constructed and fitted brace can successfully halt progression of a curve in approximately 70% of cooperative patients. Most braces should be worn over a long T-shirt or similar article of clothing for 23 hours a day. However, mild curvatures may require wearing the brace for fewer hours a day. Exercises must be done daily both in and out of the brace to maintain muscle strength. Patients should be seen for follow-up and brace adjustment every 3 months. Radiographs should be repeated at 6-month intervals. As the skeleton matures, as seen radiographically, brace wear should be gradually decreased until it's worn only at night.

The primary indications for surgery are relentless curve progression (usually curves over 40 degrees) or significant curve progression despite bracing. Surgery corrects lateral curvature by posterior spinal fusion and internal stabilization with metal rods. A distraction rod on the concave side of the curve “jacks” the spine into a straight position and provides an internal splint. An alternative procedure, anterior spinal fusion, corrects curvature with vertebral staples and an anterior stabilizing cable. Some spinal fusions may require postoperative immobilization in a brace. Postoperatively, periodic checkups are required for several months to monitor stability of the correction.

Special Considerations

It's important to provide emotional support in addition to meticulous skin care and patient teaching.

If the patient needs a brace:

- ◆ Enlist the help of a physical therapist, a social worker, and an orthotist. Before the patient goes home, explain what the brace does and how to care for it (how to check the screws for tightness and pad the uprights to prevent excessive wear on clothing). Suggest that loose-fitting, oversized clothes be worn for greater comfort.
- ◆ Tell the patient to wear the brace 23 hours/day and to remove it only for bathing and exercise. While still adjusting to the brace, instruct to lie down and rest several times per day.
- ◆ Suggest a soft mattress if a firm one is uncomfortable.
- ◆ To prevent skin breakdown, advise the patient not to use lotions, ointments, or powders on areas where the brace contacts the skin. Instruct keeping the skin dry and clean and to wear a snug T-shirt under the brace.
- ◆ Advise the patient to increase activities gradually and avoid vigorous sports. Emphasize the importance of conscientiously performing prescribed exercises.
- ◆ Instruct the patient to turn the whole body, instead of just the head, when looking to the side. To make reading easier, instruct to hold the book so it will be straight ahead at it instead of down. Prism glasses may be beneficial if this is difficult.

If the patient needs traction or a cast before surgery:

- ◆ Explain these procedures to the patient and the family. Remember that application of a body cast can be traumatic because it's done on a special frame and the patient's head and face are covered throughout the procedure.
- ◆ Check the skin around the cast edge daily. Keep the cast clean and dry and the edges of the cast petaled. Warn the patient not to insert or let anything get under the cast and to immediately report cracks in the cast, pain, burning, skin breakdown, numbness, or odor.

After corrective surgery:



ALERT *Check sensation, movement, color, and blood supply in all limbs every 2 to 4 hours for the first 48 hours and then several times a day, for signs of neurovascular deficit, a serious complication following spinal surgery. Logroll the patient often.*

- ◆ Measure intake, output, and urine specific gravity to monitor effects of blood loss, which is usually substantial.
- ◆ Monitor abdominal distention and bowel sounds.
- ◆ Encourage deep-breathing exercises to avoid pulmonary complications.
- ◆ Medicate for pain, especially before any activity.
- ◆ Promote active ROM arm exercises to help maintain muscle strength. Remember that any exercise, even brushing the hair or teeth, is helpful. Encourage the patient to perform quadriceps-setting, calf-pumping, and active ROM exercises of the ankles and feet.
- ◆ Watch for skin breakdown and signs of cast syndrome. Teach the patient how to recognize these signs. (See *Cast syndrome*.)

Cast Syndrome

Cast syndrome is a serious complication that sometimes follows spinal surgery and application of a body cast. Characterized by nausea, abdominal pressure, and vague abdominal pain, cast syndrome probably results from hyperextension of the spine. This hyperextension accentuates lumbar lordosis, compressing the third portion of the duodenum between the superior mesenteric artery anteriorly and the aorta and vertebral column posteriorly. High intestinal obstruction produces nausea, vomiting, and ischemic infarction of the mesentery.

After removal of the cast, treatment includes gastric decompression and I.V. fluids, with nothing by mouth. Antiemetics should be given sparingly because they may mask symptoms of cast syndrome, which, if untreated, may be fatal.

Teach patients who are discharged in body jackets, localizer casts, or high hip-spica casts how to recognize cast syndrome, which may manifest several weeks or months after application of the cast.

- ◆ Offer emotional support to help prevent depression that may result from altered body image and immobility. Encourage the patient to wear his or her own clothes, wash hair, and use makeup.
- ◆ If the patient is being discharged with a rod and cast and must have bed rest, arrange for a social worker and a visiting nurse to provide home care. Before discharge, check with the surgeon about activity limitations, and make sure the patient understands them.
- ◆ If you work in a school, screen children routinely for scoliosis during physical examinations.

Muscle and Connective Tissue

TENDINITIS AND BURSITIS

Tendinitis is a painful inflammation of tendons and of tendon-muscle attachments to bone, usually in the shoulder rotator cuff, hip, Achilles tendon, or hamstring. *Bursitis* is a painful inflammation of one or more of the bursae—closed sacs lubricated with small amounts of synovial fluid that facilitate the motion of muscles and tendons over bony prominences. Bursitis usually occurs in the subdeltoid, olecranon, trochanteric, calcaneal, or prepatellar bursae.

Causes and Incidence

Tendinitis commonly results from overuse or injury (such as strain during sports activity), another musculoskeletal disorder (such as rheumatic diseases or congenital defects), or aging.

Bursitis can occur at any age but usually occurs in older individuals due to an inflammatory joint disease (such as rheumatoid arthritis or gout) or recurring trauma that stresses or pressures a joint. Chronic bursitis follows attacks of acute bursitis or repeated trauma and infection. Septic bursitis may result from wound infection or from bacterial invasion of skin over the bursa.

Pathophysiology

In tendinitis, fluid from inflammation accumulates, causing swelling of the tendon and its enclosing sheath. Inflammatory changes cause thickening of the sheath, which limits movements and causes pain. Microtears cause

bleeding, edema, and pain in the involved tendon or tendons. At times, after repeated inflammations, calcium may be deposited in the tendon origin area, causing a calcific tendinitis.

The usual bursitis is an inflammation that is reactive to overuse or excessive pressure. The inflamed bursal sac becomes engorged, and the inflammation can spread to adjacent tissues. The inflammation may decrease with rest, heat, and aspiration of the fluid.

Complications

- ◆ Contractures of the tendon
- ◆ Scarring
- ◆ Muscle wasting
- ◆ Disability

Signs and Symptoms

The patient with tendinitis of the shoulder complains of restricted shoulder movement, especially abduction, and localized pain, which is most severe at night and usually interferes with sleep. The pain extends from the acromion (the shoulder's highest point) to the deltoid muscle insertion, predominantly in the so-called painful arc—that is, when the patient abducts the arm between 50 and 130 degrees. Fluid accumulation causes swelling. In calcific tendinitis, calcium deposits in the tendon cause proximal weakness and, if calcium erodes into adjacent bursae, acute calcific bursitis.

In bursitis, fluid accumulation in the bursae causes irritation, inflammation, sudden or gradual pain, and limited movement. Other symptoms vary according to the affected site. Subdeltoid bursitis impairs arm abduction, prepatellar bursitis (housemaid's knee) produces pain when the patient climbs stairs, and hip bursitis makes crossing the legs painful.

Diagnosis

In tendinitis, X-rays may be normal at first but later show bony fragments, osteophyte sclerosis, or calcium deposits. Arthrography is usually normal, with occasional small irregularities on the undersurface of the tendon. CT scan and MRI have replaced X-ray and even arthrography of the shoulder as diagnostic tools. An MRI will usually identify tears, partial tears, inflammation, or tumor but cannot reveal irregularities of the tendon sheath itself. Diagnosis of tendinitis must rule out other causes of shoulder pain,

such as myocardial infarction, cervical spondylosis, degenerative changes, and tendon tear or rupture. Significantly, in tendinitis, heat aggravates shoulder pain; in other painful joint disorders, heat usually provides relief.

Localized pain and inflammation and a history of unusual strain or injury 2 to 3 days before onset of pain are the bases for diagnosing bursitis. During early stages, X-rays are usually normal, except in calcific bursitis, where X-rays may show calcium deposits.

Treatment

Treatment to relieve pain includes resting the joint (by immobilization with a sling, splint, or cast, or via activity modification), NSAIDs, analgesics, application of cold or heat, ultrasound, or local injection of an anesthetic and corticosteroids to reduce inflammation. A mixture of a corticosteroid and an anesthetic such as lidocaine generally provides immediate pain relief. Extended-release injections of a corticosteroid, such as triamcinolone or prednisolone, offer long-term pain relief. Until the patient is free of pain and able to perform ROM exercises easily, treatment also includes oral NSAIDs, such as ibuprofen, naproxen, indomethacin, or oxaprozin. Short-term analgesics include propoxyphene, codeine, acetaminophen with codeine and, occasionally, oxycodone.

Supplementary treatment includes fluid removal by aspiration and heat therapy; for calcific tendinitis, ice packs, physical therapy, ultrasonography, or hydrotherapy generally helps maintain or regain ROM. It may be necessary to delay treatment until the acute attack is over to ensure maximum patient compliance. Rarely, calcific tendinitis requires surgical removal of calcium deposits. Long-term control of chronic bursitis and tendinitis may require changes in lifestyle to prevent recurring joint irritation.

Special Considerations

When treating patients with tendinitis or bursitis, remember to consider the following:

- ◆ Assess the severity of pain and the ROM to determine the effectiveness of the treatment.
- ◆ Before injecting corticosteroids or local anesthetics, ask the patient about drug allergies.

- ◆ Assist with intra-articular injection. Scrub the patient's skin thoroughly with povidone–iodine or a comparable solution. After the injection, massage the area to ensure penetration through the tissue and joint space. Apply ice intermittently for about 4 hours to minimize pain. Avoid applying heat to the area for 2 days.
- ◆ Tell the patient to take anti-inflammatory agents with milk to minimize GI distress and to report any signs of distress immediately.
- ◆ Advise the patient to perform strengthening exercises and avoid activities that aggravate the joint.
- ◆ Remind the patient to wear a splint or sling during the first few days of an attack of subdeltoid bursitis or tendinitis to support the arm and protect the shoulder, particularly at night. Demonstrate how to wear the sling so it won't put too much weight on the shoulder.
- ◆ Advise the patient to maintain joint mobility and prevent muscle atrophy by performing exercises or physical therapy when free of pain.



PEDIATRIC TIP *A common form of tendinitis in adolescents (both males and females) is patellar tendinitis associated with inflammation of the tibial epiphysis in Osgood–Schlatter disease.*

EPICONDYLITIS

Lateral epicondylitis of the elbow (tennis elbow) is inflammation of the extensor tendons of the forearm. Medial epicondylitis (golfer's elbow) is inflammation at the origin of the flexor muscles of the wrist.

Causes and Incidence

Epicondylitis probably begins as a partial tear and is common among tennis players or persons whose activities require a forceful grasp, wrist extension against resistance, or frequent rotation of the forearm, such as using a screwdriver. Untreated epicondylitis may become disabling as adherent fibers form between the tendons and the elbow capsule.

Pathophysiology

Epicondylitis is the result of irritation and inflammation where the tendon attaches to a bone. Overuse and excessive pressure are factors.

Complications

- ◆ Recurrence of injury
- ◆ Tendon rupture

Signs and Symptoms

The patient's initial symptom is elbow pain that gradually worsens and commonly radiates to the forearm and back of the hand whenever an object is grasped, or the elbow is twisted. Other associated signs and symptoms include tenderness over the involved lateral or medial epicondyle or over the head of the radius and a weak grasp. In rare instances, epicondylitis may cause local heat, swelling, or restricted ROM.

Diagnosis

Because X-rays are almost always negative, diagnosis typically depends on clinical signs and symptoms and a patient history of playing tennis or engaging in similar activities. The pain can be reproduced by wrist extension and supination with lateral epicondyle involvement or by flexion and pronation with medial epicondyle involvement.

Treatment

Treatment aims to relieve pain, usually by NSAIDs or local injection of corticosteroids and an anesthetic. Supportive treatment includes an immobilizing splint from the distal forearm to the elbow or wrist splint, which generally relieves pain in 2 to 3 weeks; heat therapy, such as warm compresses, short-wave diathermy, and ultrasound (alone or in combination with diathermy); and physical therapy, such as manipulation and massage to detach the tendon from the chronically inflamed periosteum. A "tennis elbow strap" or counterface brace has helped many patients. This strap, which is wrapped snugly around the forearm approximately 1 (2.5 cm) below the epicondyle, helps relieve the strain on affected forearm muscles and tendons. If these measures prove ineffective, surgical release of the tendon at the epicondyle may be necessary.

Special Considerations

The following special considerations accompany diagnosis and treatment of epicondylitis:

- ◆ Assess the patient's level of pain, ROM, and sensory function. Monitor heat therapy to prevent burns.
- ◆ Advise the patient to take anti-inflammatory drugs with food to avoid GI irritation.
- ◆ Instruct the patient to rest the elbow, wrist, or both until inflammation subsides.
- ◆ Remove the support daily, and gently move the arm to prevent stiffness and contracture.
- ◆ Instruct the patient to follow the prescribed exercise program. For example, the arm may be stretched and the wrist flexed to the maximum then press the back of the hand against a wall until a pull can be felt in the forearm and hold this position for 1 minute.
- ◆ Advise the patient to warm up for 15 to 20 minutes before beginning any sports activity.
- ◆ Urge the patient to wear an elastic support or splint during any activity that stresses the forearm or elbow.
- ◆ Tell the patient to check the equipment. For example, a tennis racquet may not be the right size or weight. Also, changing surfaces may help to reduce stress.

ACHILLES TENDON CONTRACTURE

Achilles tendon contracture is a shortening of the Achilles tendon (*tendo calcaneus* or *heel cord*) that causes foot pain and strain and limits ankle dorsiflexion.

Causes and Incidence

Achilles tendon contracture may reflect a congenital structural anomaly or a muscular reaction to chronic poor posture, especially in women who wear high-heeled shoes or joggers who land on the balls of their feet instead of their heels. Other causes include paralytic conditions of the legs, such as poliomyelitis or cerebral palsy.

Pathophysiology

The Achilles tendon spans two joints and connects the calcaneus to the gastrocnemius and soleus muscles, comprising the largest and strongest muscle complex in the calf. The tendon is vulnerable to injury because of its limited blood supply, especially when subjected to strong forces.

The blood supply to the tendon is provided by longitudinal arteries that run the length of the muscle complex. The area of the tendon with the poorest blood supply is approximately 2 to 6 cm above the insertion into the calcaneus. The blood supply diminishes with age, predisposing this area of the tendon to chronic inflammation and possible rupture.

Complication

- ◆ Permanent weakness

Signs and Symptoms

Sharp, spasmoid pain during dorsiflexion of the foot characterizes the reflex type of Achilles tendon contracture. In footdrop (fixed equinus), contracture of the flexor foot muscle prevents placing the heel on the ground.

Diagnosis

Physical examination and patient history suggest Achilles tendon contracture.



CONFIRMING DIAGNOSIS *A simple test confirms Achilles tendon contracture: While the patient keeps the knee flexed, the examiner places the foot in dorsiflexion; gradual knee extension forces the foot into plantar flexion.*

Treatment

Conservative treatment aims to correct Achilles tendon contracture by raising the inside heel of the shoe in the reflex type; by gradually lowering the heels of shoes (sudden lowering can aggravate the problem) and stretching exercises if the cause is high heels; or by using support braces or casting to prevent footdrop in a paralyzed patient. Alternative therapy includes using wedged plaster casts or stretching the tendon by manipulation. Analgesics may be given to relieve pain.

With fixed footdrop, treatment may include surgery. Although this procedure may weaken the tendon, it allows further stretching by cutting the tendon. After surgery, a short leg cast maintains the foot in 90-degree dorsiflexion for 6 weeks. Some surgeons allow partial weight bearing on a walking cast after 2 weeks.

Special Considerations

After surgery to lengthen the Achilles tendon:

- ◆ Elevate the casted foot to decrease venous pressure and edema by raising the foot of the bed or supporting the foot with pillows.
- ◆ Record the neurovascular status of the toes (temperature, color, sensation, capillary refill time, and toe mobility) every hour for the first 24 hours and then every 4 hours. If any changes are detected, increase the elevation of the patient's legs and notify the surgeon immediately.
- ◆ Prepare the patient for ambulation by dangling the foot over the side of the bed for short periods (5 to 15 minutes) before getting out of bed, allowing for a gradual increase in venous pressure. Assist the patient in walking, as ordered (usually within 24 hours of surgery), using crutches and a nonweight-bearing or touch-down gait.
- ◆ Protect the patient's skin with moleskin or by petaling the edges of the cast. Before discharge, teach the patient how to care for the cast, and advise to elevate the foot regularly when sitting or whenever the foot throbs or becomes edematous. Also, make sure the patient understands how much exercise and walking are recommended after discharge.
- ◆ To prevent Achilles tendon contracture in paralyzed patients, apply support braces, universal splints, casts, or high-topped sneakers. Make sure the weight of the sheets doesn't keep paralyzed feet in plantar flexion. For other patients, teach good foot care and urge them to seek immediate medical care for foot problems. Warn women against wearing high heels constantly and suggest regular foot (dorsiflexion) exercises.

CARPAL TUNNEL SYNDROME

Carpal tunnel syndrome, a form of repetitive stress injury, is the most common of the nerve entrapment syndromes. It results from compression of the median nerve at the wrist, within the carpal tunnel. This compression neuropathy causes sensory and motor changes in the median distribution of the hand.

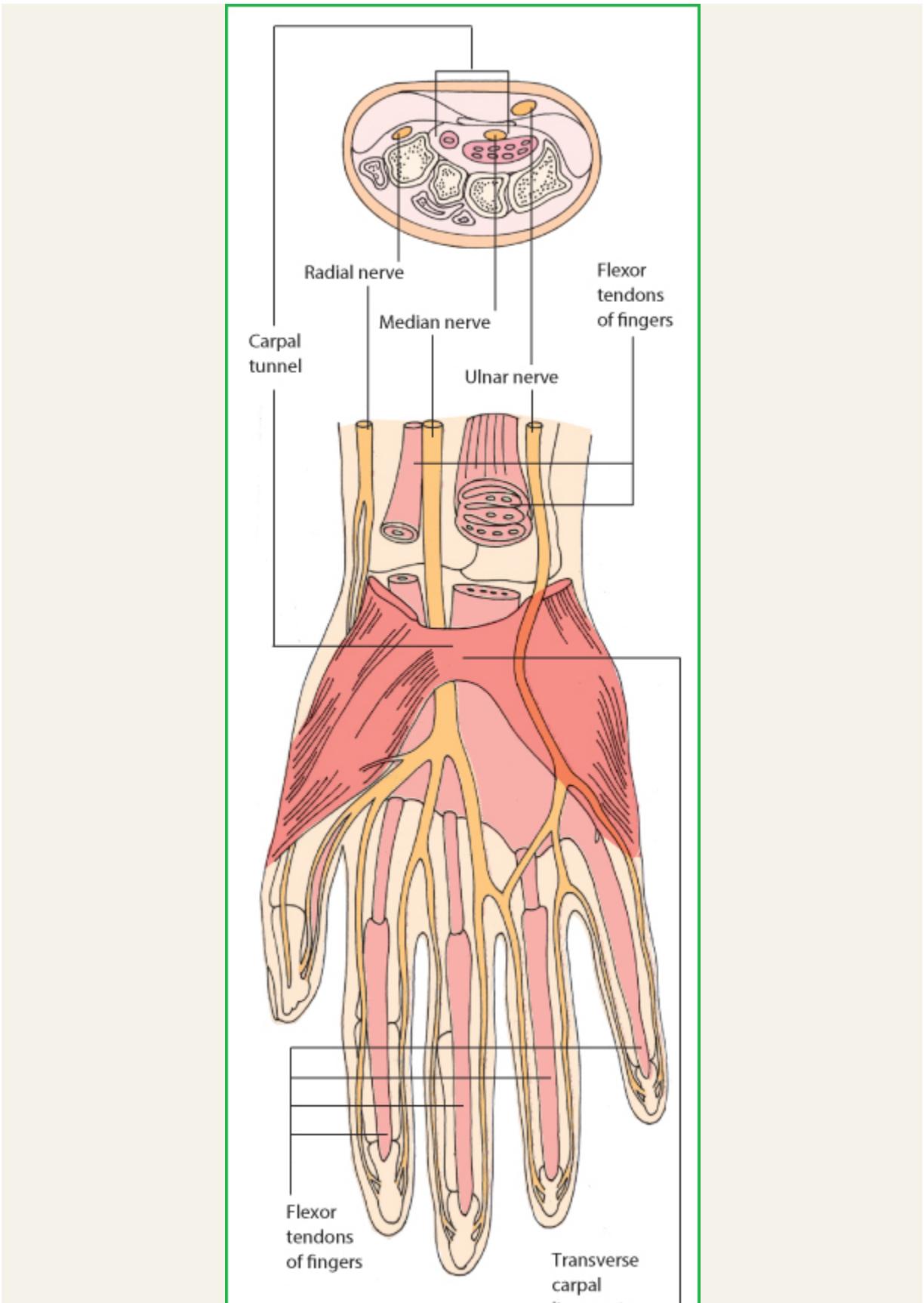
Causes and Incidence

The carpal tunnel is formed by the carpal bones and the transverse carpal ligament. (See *The carpal tunnel*.) Inflammation or fibrosis of the tendon sheaths that pass through the carpal tunnel commonly causes edema and

compression of the median nerve. Many conditions can cause the contents or structure of the carpal tunnel to swell and press the median nerve against the transverse carpal ligament, including rheumatoid arthritis, flexor tenosynovitis (commonly associated with rheumatic disease), nerve compression, pregnancy, renal failure, menopause, diabetes mellitus, acromegaly, edema following Colles fracture, hypothyroidism, amyloidosis, myxedema, benign tumors, TB, and other granulomatous diseases. Another source of damage to the median nerve is dislocation or acute sprain of the wrist.

The Carpal Tunnel

The carpal tunnel is clearly visible in this palmar view and cross section of a right hand. Note the median nerve, flexor tendons of fingers, and blood vessels passing through the tunnel on their way from the forearm to the hand.



Carpal tunnel injury is five times more common in women than in men. It usually occurs in women between ages 30 and 60 and poses a serious occupational health problem. Assembly-line workers and packers and people who repeatedly use poorly designed tools are most likely to develop this disorder. Any strenuous use of the hands—sustained grasping, twisting, or flexing—aggravates this condition. (See *Preventing carpal tunnel syndrome.*)



PREVENTION PREVENTING CARPAL TUNNEL SYNDROME

To prevent carpal tunnel syndrome, advise your patients to make these lifestyle changes.

Take Frequent Breaks

Gently stretching and bending the hands and wrists every 15 to 20 minutes gives the hands and wrists a break, especially when using equipment that vibrates or exerts a great amount of force. Tasks should also be alternated to avoid repetitive movements, which can contribute to tendinitis and carpal tunnel syndrome.

Watch Hand and Wrist Positioning

When using a keyboard, bending the wrist all the way up or down should be avoided. A relaxed middle position is best. The keyboard should be kept at elbow height or slightly lower.

Improve Posture

Poor posture can cause the shoulders to roll forward, allowing the neck and shoulder muscles to shorten, which can compress the nerves in the neck. This position can affect the wrists, hands, and fingers.

Keep Hands Warm

Hand stiffness and pain develops more frequently in a cold environment. Using fingerless gloves may help if the temperature can't

be adjusted at work.

Pathophysiology

The carpal bones and the transverse carpal ligament form the carpal tunnel. Inflammation or fibrosis of the tendon sheaths that pass through the carpal tunnel usually causes edema and compression of the median nerve. (See *Cross section of the wrist with carpal tunnel syndrome*, page 327.) This compression neuropathy causes sensory and motor changes in the median distribution of the hands, initially impairing sensory transmission to the thumb, index finger, second finger, and inner aspect of the third finger.

Complications

- ◆ Decreased wrist function
- ◆ Permanent nerve damage
- ◆ Loss of movement and sensation

Signs and Symptoms

The patient with carpal tunnel syndrome usually complains of weakness, pain, burning, numbness, or tingling in one or both hands. This paresthesia affects the thumb, forefinger, middle finger, and half of the fourth finger. The patient is unable to clench the hand into a fist; the nails may be atrophic, the skin dry and shiny.

Because of vasodilatation and venous stasis, symptoms are typically worse at night and in the morning. The pain may spread to the forearm and, in severe cases, as far as the shoulder or neck. The patient can usually relieve such pain by shaking or rubbing hands vigorously or dangling his arms.

Diagnosis

Physical examination reveals decreased sensation to light touch or pinpricks in the affected fingers. Thenar muscle atrophy occurs in about half of all cases of carpal tunnel syndrome, but it's usually a late sign. The patient exhibits a positive Tinel's sign (tingling over the median nerve on light percussion) and responds positively to Phalen's wrist-flexion test (holding the forearms vertically and allowing both hands to drop into complete flexion at the wrists for 1 minute reproduces symptoms of carpal tunnel

syndrome). A compression test supports this diagnosis: A blood pressure cuff inflated above systolic pressure on the forearm for 1 to 2 minutes provokes pain and paresthesia along the distribution of the median nerve.

Electromyography and nerve conduction velocity detect a median nerve motor conduction delay of more than 5 ms. Other laboratory tests may identify the underlying disease.

Treatment

Conservative treatment should be tried first, including resting the hands by splinting the wrist in neutral extension for 1 to 2 weeks. NSAIDs usually provide symptomatic relief. Injection of the carpal tunnel with hydrocortisone and lidocaine may provide significant but temporary relief. If a definite link has been established between the patient's occupation and the development of repetitive stress injury, seeking another type of work may be recommended. Effective treatment may also require correction of an underlying disorder. When conservative treatment fails, the only alternative is surgical decompression of the nerve by resecting the entire transverse carpal tunnel ligament or by using endoscopic surgical techniques. Neurolysis (freeing of the nerve fibers) may also be necessary.

Special Considerations

Patient care for carpal tunnel syndrome includes the following:

- ◆ Administer mild analgesics as needed. Encourage the patient to use the hands as much as possible. If the dominant hand has been impaired, you may have to help with eating and bathing.
- ◆ Teach the patient how to apply a splint. Instruct to not to make it too tight. Demonstrate how to remove the splint to perform gentle ROM exercises, which should be done daily. Make sure the patient knows how to do these exercises before discharge.
- ◆ After surgery, monitor vital signs, and regularly check the color, sensation, and motion of the affected hand.
- ◆ Advise the patient who's about to be discharged to occasionally exercise the hands in warm water. If the arm is in a sling, instruct to remove the sling several times a day to do exercises for the elbow and shoulder.
- ◆ Suggest occupational counseling for the patient who must change jobs because of repetitive stress injury.

TORTICOLLIS

Torticollis, sometimes called *wryneck*, is a neck deformity in which the sternocleidomastoid (SCM) neck muscles are spastic or shortened, causing bending of the head to the affected side and rotation of the chin to the opposite side.

Causes and Incidence

Torticollis may be congenital or acquired. The three types of acquired torticollis—acute, spasmotic, and hysterical—have differing causes. The acute form results from muscular damage caused by inflammatory diseases, such as myositis, lymphadenitis, or TB; from cervical spinal injuries that produce scar tissue contracture; and, less commonly, from tumor or medication. The spasmotic form results from rhythmic muscle spasms caused by an organic central nervous system disorder (probably due to irritation of the nerve root by arthritis or osteomyelitis). Hysterical torticollis is due to a psychogenic inability to control neck muscles.

Acquired torticollis usually develops during the first 10 years of life or between ages 30 and 60. The incidence of congenital (muscular) torticollis is highest in infants after difficult delivery (breech presentation), in firstborn infants, and in girls. Possible causes of congenital torticollis include malposition of the head in utero, prenatal injury, fibroma, interruption of blood supply, or fibrotic rupture of the SCM muscle, with hematoma and scar formation.

Pathophysiology

Congenital Torticollis

Congenital muscular torticollis is rare (<2%) and is believed to be caused by local trauma to the soft tissues of the neck just before or during delivery. The most common explanation involves birth trauma to the SCM muscle, resulting in fibrosis or that intrauterine malpositioning leads to unilateral shortening of the SCM. There may be resultant hematoma formation followed by muscular contracture. These children often have undergone breech or difficult forceps delivery.

The fibrosis in the muscle may be due to venous occlusion and pressure on the neck in the birth canal because of cervical and skull position. Another hypothesis includes malposition in utero resulting in intrauterine or

perinatal compartment syndrome. Other causes of congenital torticollis include postural torticollis, pterygium colli (webbed neck), SCM cysts, vertebral anomalies, odontoid hyperplasia, spina bifida, hypertrophy or absence of cervical musculature, and Arnold–Chiari syndrome. It can also be seen with clavicular fractures, especially in neonates secondary to birth trauma. Up to 20% of children with congenital muscular torticollis have congenital dysplasia of the hip as well.

Acquired Torticollis

The pathophysiology of acquired torticollis depends on the underlying disease process. Cervical muscle spasm causing torticollis can result from any injury or inflammation of the cervical muscles or cranial nerves from different disease processes.

Acute torticollis can be the result of blunt trauma to head and neck, or from simply sleeping in an awkward position. Acute torticollis may be self-limited in days to weeks or the result of idiosyncrasy to certain medications (e.g., traditional dopamine receptor blockers, metoclopramide, phenytoin, or carbamazepine). After stopping medication, it quickly resolves without further action. After the resolution of acute traumatic torticollis, a chronic or persistent form may reappear after days or weeks of a quiescent interval. This situation often has legal implications regarding liability associated with the acute traumatic incident.

Complication

- ◆ Permanent contracture

Signs and Symptoms



PEDIATRIC TIP *The first sign of congenital torticollis is commonly a firm, nontender, palpable enlargement of the SCM muscle that's visible at birth and for several weeks afterward. It slowly regresses during a period of 6 months, although incomplete regression can cause permanent contracture. If the deformity is severe, the infant's face and head flatten from sleeping on the affected side; this asymmetry gradually worsens. The infant's chin turns away from the side of the shortened muscle, and the head*

tilts to the shortened side. The shoulder may elevate on the affected side, restricting neck movement.

The first sign of acquired torticollis is usually recurring unilateral stiffness of neck muscles followed by a drawing sensation and a momentary twitching or contraction that pulls the head to the affected side. This type of torticollis commonly produces severe neuralgic pain throughout the head and neck.

Diagnosis

A history of painless neck deformity from birth suggests congenital torticollis; gradual onset of painful neck deformity suggests acquired torticollis. Diagnosis must rule out TB of the cervical spine, pharyngeal or tonsillar inflammations, spinal accessory nerve damage, ruptured transverse ligaments, subdural hematoma, tumors of soft tissue or bone, dislocations and fractures, scoliosis, congenital abnormalities of the cervical spine and base of the skull, rheumatoid arthritis, and osteomyelitis. In acquired torticollis, cervical spine X-rays are negative for bone or joint disease but may reveal an associated disorder (such as TB, scar tissue formation, tumor, deformities, or arthritis). CT scan or MRI may help rule out pathogenic causes.

Treatment

Treatment of congenital torticollis aims to stretch the shortened muscle. Nonsurgical treatment includes passive neck stretching and proper positioning during sleep for an infant and active stretching exercises for an older child—for example, touching the ear opposite the affected side to the shoulder and touching the chin to the same shoulder.

Surgical correction involves sectioning the SCM muscle; this should be done during preschool years and only if other therapies fail.

Treatment of acquired torticollis aims to control pain and correct the underlying cause of the disease. In the acute form, application of heat, cervical traction, and gentle massage may help relieve pain; analgesics may also be helpful. Stretching exercises and a neck brace may relieve symptoms of the spasmodic and hysterical forms. Drug treatment includes anticholinergic drugs such as baclofen. Botulinum toxin injections are effective in temporarily relieving torticollis, but injections must be repeated every 3 months.

Special Considerations

Patient care for torticollis includes the following:

- ◆ To aid early diagnosis of congenital torticollis, observe the infant for limited neck movement, and thoroughly assess the degree of discomfort.
- ◆ Teach the parents of an affected child how to perform stretching exercises with the infant. Suggest placing toys or mobiles on the side of the crib opposite the affected side of the child's neck to encourage the child to move the head and stretch the neck.
- ◆ If surgery is necessary, prepare the patient by shaving the neck to the hairline on the affected side.

After corrective surgery:

- ◆ Monitor the patient closely for nausea or signs of respiratory complications, especially if in cervical traction. Keep suction equipment available to prevent aspiration.
- ◆ The patient may be in a cast or in traction day and night or at night only. Monitor the skin around the chin, ears, and back of the head if the patient is in cervical traction. Monitor for problems related to clenching of teeth. If the patient is in a cast, give meticulous cast care, including the monitoring of circulation, sensation, and color around the cast. Protect the cast around the patient's chin and mouth with waterproof material. Check for skin irritation, pressure areas, or softening of cast pad.
- ◆ Provide emotional support for the patient and the family to relieve their anxiety due to fear, pain, limitations from the brace or traction, and an altered body image.
- ◆ Begin stretching exercises as soon as the patient can tolerate them.
- ◆ Before discharge, explain to the patient or the parents the importance of continuing daily heat applications, massages, and stretching exercises, as prescribed, and of keeping the cast clean and dry. Emphasize that physical therapy is essential for a successful rehabilitation after the cast is removed.

RHABDOMYOLYSIS

Rhabdomyolysis is the breakdown of muscle fibers that results in the release of muscle fiber content into the circulation. It results from the toxicity of destroyed muscle cells, causing kidney damage or failure.

Predisposing factors include trauma, ischemia, polymyositis, and drug overdose. Toxins and environmental, infectious, and metabolic factors may induce it. Rhabdomyolysis accounts for 8% to 15% of cases of acute renal failure; about 5% of cases result in death.

Causes and Incidence

Rhabdomyolysis follows direct injury to the skeletal muscle fibers, specifically the sarcolemma, which then release myoglobin into the bloodstream. Myoglobin is an oxygen-binding protein pigment found in skeletal muscle. When this muscle is damaged, myoglobin is released into the bloodstream. It's then filtered by the kidneys.

Myoglobin may occlude the structures of the kidney causing damage, such as acute tubular necrosis or kidney failure. Myoglobin can also cause kidney failure because it breaks down into potentially toxic compounds. Necrotic skeletal muscle may cause massive fluid shifts from the bloodstream into the muscle, reducing the relative fluid volume of the body and leading to shock and reduced blood flow to the kidneys.

The disorder may be caused by any condition that results in damage to skeletal muscle. Rhabdomyolysis may result from blunt trauma; extensive burn injury; viral, bacterial, or fungal infection (such as Legionnaires' disease or, especially, influenza type A or B); prolonged immobilization; near electrocution or near drowning; metabolic or genetic factors; drug therapy; or toxins. Heavy exercise may result in rhabdomyolysis. Other causes include shaken baby syndrome, exposure to extreme cold, heatstroke, and snakebite.

In the United States, rhabdomyolysis affects about 8% to 15% of people with acute renal failure and has a slightly higher incidence in men than in women. The overall mortality rate is 5%. It can occur in infants, toddlers, and adolescents who inherited enzyme deficiencies of carbohydrate and lipid metabolism or those with inherited myopathies, such as Duchenne muscular dystrophy, and malignant hyperthermia.

Pathophysiology

Muscle trauma that compresses tissue causes ischemia and necrosis. The ensuing local edema further increases compartment pressure and tamponade; pressure from severe swelling causes blood vessels to collapse, leading to tissue hypoxia, muscle infarction, neural damage in the area of

the fracture, and release of myoglobin from the necrotic muscle fibers into the circulation. Myoglobin may occlude the structures of the kidney, causing such damage as acute tubular necrosis or kidney failure. Myoglobin can also cause kidney failure because it breaks down into potentially toxic compounds.

Complications

- ◆ Acute tubular necrosis
- ◆ Kidney failure

Signs and Symptoms

Signs and symptoms of rhabdomyolysis include myalgias or muscle pain (especially in the thighs, calves, or lower back), weakness, tenderness, malaise, fever, dark urine, nausea, and vomiting. The patient may also experience weight gain, seizures, joint pain, and fatigue. Symptoms may be subtle initially. Rhabdomyolysis can result in acute renal failure.

Diagnosis

A serum or urine myoglobin test is positive. Creatine kinase levels 100 times above normal or higher suggest rhabdomyolysis. A urinalysis may reveal casts and may be positive for hemoglobin without evidence of red blood cells on microscopic examination. Serum potassium may be very high (potassium is released from cells into the bloodstream when cell breakdown occurs).

Treatment

Early, aggressive hydration may prevent complications from rhabdomyolysis by rapidly eliminating the myoglobin from the kidneys. I.V. hydration and diuretics promote diuresis. Diuretic medications, such as mannitol or furosemide, may aid in flushing the pigment out of the kidneys. If urine output is sufficient, bicarbonate may be given to maintain an alkaline urine state, thereby helping to prevent the dissociation of myoglobin into toxic compounds. Hyperkalemia should be treated if present. Kidney failure should be treated as appropriate. Dialysis may be necessary and, in severe cases, kidney transplantation.

Special Considerations

- ♦ Monitor the patient's intake and output, vital signs, electrolyte levels, daily weight, and laboratory results.



ALERT *Watch for signs of renal failure (such as decreasing urine output and increasing urine specific gravity), fluid overload (such as dyspnea and tachycardia), pulmonary edema, and electrolyte imbalances (such as serum potassium).*

- ♦ Provide reassurance and emotional support for the patient and the family.
- ♦ To help prevent rhabdomyolysis from occurring, ensure adequate hydration, monitor the patient for adverse reactions to any prescribed medications, and monitor blood transfusion administration carefully.

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13

Ear, Nose, and Throat Disorders

Introduction

Ear, nose, and throat disorders rarely prove fatal (except for those resulting from neoplasms, epiglottitis, and neck trauma), but they may cause serious social, cosmetic, and communication problems. Untreated hearing loss or deafness can drastically impair the ability to interact with society. Ear disorders also can cause impaired equilibrium. Nasal disorders can cause changes in facial features and interfere with breathing and tasting. Diseases arising in the throat may threaten airway patency and interfere with speech. In addition, these disorders can cause considerable discomfort and pain for the patient and require thorough assessment and prompt treatment.

THE EAR

Hearing begins when sound waves reach the tympanic membrane, which then vibrates the ossicles, incus, malleus, and stapes in the middle ear cavity. The stapes transmits these vibrations to the perilymphatic fluid in the inner ear by vibrating against the oval window. The vibrations then pass across the cochlea's fluid receptor cells in the basilar membrane, stimulating movement of the hair cells of the organ of Corti. The axons of the cochlear nerve terminate around the bases of those hair cells. Sound waves, which initiate impulses, travel over the auditory nerve (made up of

the cochlear nerve and the vestibular nerve) to the temporal lobe of the brain.

The inner ear structures also maintain the body's equilibrium and balance through the fluid in the semicircular canals. This fluid is set in motion by body movement and stimulates nerve cells that line the canals. These cells, in turn, transmit impulses to the cerebellum of the brain by way of the vestibular branch of the eighth cranial nerve (the acoustic nerve).

Although the ear can respond to sounds that vibrate at frequencies from 20 to 20,000 Hz, the range of normal speech is from 250 to 4,000 Hz, with 70% falling between 500 and 2,000 Hz. The ratio between sound intensities, the decibel (dB) is the unit for expressing the relative intensity (loudness) of sounds. A faint whisper registers 10 to 15 dB; average conversation, 50 to 60 dB; a shout, 85 to 90 dB. Hearing damage may follow exposure to sounds louder than 90 dB.

ASSESSMENT

After obtaining a thorough patient history of any ear disease, inspect the auricle and surrounding tissue for deformities, lumps, and skin lesions. (See *Structures of the external ear*, page 626.) Ask the patient if they have ear pain. If you see inflammation, check for tenderness by moving the auricle and pressing on the tragus and the mastoid process. Check the ear canal for excessive cerumen, discharge, or foreign bodies.

Structures of the External Ear

The structures of the external ear are depicted below.

EXTERNAL EAR

Helix

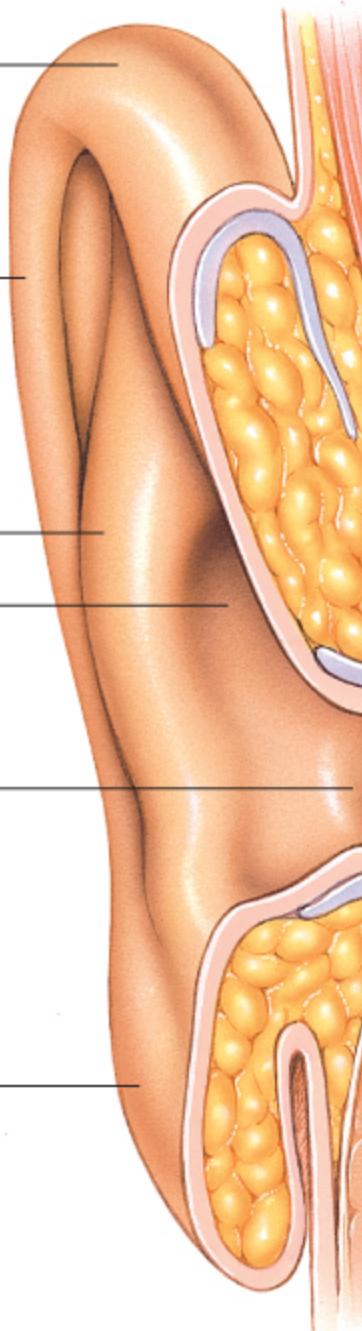
Darwinian
tubercle

Anthelix

Concha

External
acoustic
meatus

Lobule of
auricle



Ask the patient if they have had episodes of vertigo or blurred vision. To test for vertigo, have the patient stand on one foot and close their eyes, or

have them walk a straight line with their eyes closed. Ask them if they always fall to the same side and if the room seems to be spinning.

AUDIOMETRIC TESTING

Audiometric testing evaluates hearing and determines the type and extent of hearing loss. The simplest but least reliable method for judging hearing acuity consists of covering one of the patient's ears, standing 18" to 24" (46 to 61 cm) from the uncovered ear, and whispering a short phrase or series of numbers. (Block the patient's vision to prevent lip reading.) Then ask the patient to repeat the phrase or series of numbers. To test hearing at both high and low frequencies, repeat the test in a normal speaking voice. (As an alternative, you can hold a ticking watch to the patient's ear.)

If you identify a hearing loss, further testing is necessary to determine if the loss is conductive or sensorineural. A conductive loss can result from faulty bone conduction (inability of the eighth cranial nerve to respond to sound waves traveling through the skull) or faulty air conduction (impaired transmission of sound through ear structures to the auditory nerve and, ultimately, the temporal lobe of the brain).

Sensorineural hearing loss results from damage to the cochlear or vestibulocochlear nerve, which can result from aging and prolonged exposure to high frequency or loud noises.

The following tests assess bone and air conduction:

- ◆ Impedance audiometry detects middle ear pathology, precisely determining the degree of tympanic membrane and middle ear mobility. One end of the impedance audiometer, a probe with three small tubes, is inserted into the external canal; the other end is attached to an oscillator. One tube delivers a low tone of variable intensity, the second contains a microphone, and the third, an air pump. A mobile tympanic membrane reflects minimal sound waves and produces a low-voltage curve on the graph. A tympanic membrane with decreased mobility reflects maximal sound waves and produces a high-voltage curve.
- ◆ Pure tone audiometry uses an audiometer to produce a series of pure tones of calibrated decibels of loudness at different frequencies (125 to 8,000 Hz). These test tones are conveyed to the patient's ears through headphones or a bone conduction (sound) vibrator. Speech threshold represents the loudness at which a person with normal hearing can

perceive the tone. Both air conduction and bone conduction are measured for each ear, and the results are plotted on a graph. If hearing is normal, the line is plotted at 0 dB. In adults, normal hearing may range from 0 to 25 dB.

- ◆ In the Rinne test, the base of a lightly vibrating tuning fork is placed on the mastoid process (bone conduction). Then the fork is moved to the front of the meatus, where the patient should continue to hear the vibrations (air conduction). The patient must determine which sounds are heard longer. In a positive Rinne test, sounds heard through air conduction are heard relatively longer than those heard through bone conduction. This may suggest sensorineural hearing loss. In a negative Rinne test, sounds heard through bone conduction are heard longer than those heard through air conduction, which may suggest a conductive loss.
- ◆ Speech audiometry uses the same technique as pure tone audiometry, but with speech, instead of pure tones, transmitted through the headset. (A person with normal hearing can hear and repeat 88% to 100% of transmitted words.)
- ◆ Tympanometry, using the impedance audiometer, measures tympanic membrane compliance with air pressure variations in the external canal and determines the degree of negative pressure in the middle ear.
- ◆ In Weber test (used for testing unilateral hearing loss), the handle of a lightly vibrating tuning fork is placed on the midline of the forehead. Normally, the patient should hear sounds equally in both ears. With conductive hearing loss, sound lateralizes (localizes) to the ear with the poorest hearing. With sensorineural loss, sound lateralizes to the better functioning ear.

THE NOSE

As air travels between the septum and the turbinates, it touches sensory hairs (cilia) in the mucosal surface, which then add, retain, or remove moisture and particles in the air to ensure delivery of humid, bacteria-free air to the pharynx and lungs. In addition, when air touches the mucosal cilia, the resultant stimulation of the first cranial nerve sends nerve impulses to the olfactory area of the frontal cortex, providing the sense of smell.

ASSESSMENT

Check the external nose for redness, edema, masses, or poor alignment. Marked septal cartilage depression may indicate saddle deformity because of septal destruction from trauma or congenital syphilis; extreme lateral deviation may result from injury. Red nostrils may indicate frequent nose blowing caused by allergies or infectious rhinitis. Dilated, engorged blood vessels may suggest alcoholism or constant exposure to the elements. A bulbous, discolored nose may be a sign of rosacea.

With a nasal speculum and adequate lighting, check nasal mucosa for pallor and edema or redness and inflammation, dried mucous plugs, furuncles, and polyps. Also, look for abnormal appearance of the capillaries, boggy turbinates, and a deviated or perforated septum. Check for nasal discharge (assess color, consistency, and odor) and blood. Profuse, thin, watery discharge may indicate allergy or cold; excessive, thin, purulent discharge may indicate cold or chronic sinus infection.

Check for sinus inflammation by applying pressure to the nostrils, orbital rims, and cheeks. Pain after pressure applied above the upper orbital rims indicates frontal sinus irritation; pain after pressure applied to the cheeks, maxillary sinus irritation.

THE THROAT

Parts of the throat include the pharynx, epiglottis, and larynx. The pharynx is the passageway for food to the esophagus and air to the larynx. The epiglottis (the lid of the larynx) diverts material away from the glottis during swallowing. The larynx produces sounds by vibrating expired air through the vocal cords. Changes in vocal cord length and air pressure affect pitch and voice intensity. The larynx also stimulates the vital cough reflex when a foreign body touches its sensitive mucosa.

ASSESSMENT

Using a bright light and a tongue blade, inspect the patient's mouth and throat. Look for inflammation or white patches, and any irregularities on the tongue or throat. Make sure the patient's airway isn't compromised and also assess vital signs. Watch for and immediately report signs of respiratory distress (dyspnea, tachycardia, tachypnea, inspiratory stridor, restlessness, and nasal flaring) and changes in voice or in skin color, such as circumoral or nail bed cyanosis. Assess symmetry of the tongue as well as function of

the soft palate. The main diagnostic test used in throat assessment is a culture to identify the infective organism.

External Ear

OTITIS EXTERNA

Otitis externa, inflammation of the skin of the external ear canal and auricle, may be acute or chronic. Also known as *external otitis* and *swimmer's ear*, it's most common in the summer. With treatment, acute otitis externa usually subsides within 7 days—although it may become chronic—and tends to recur.

Causes and Incidence

Otitis externa usually results from bacteria, such as *Pseudomonas*, *Proteus vulgaris*, *Staphylococcus aureus*, and streptococci and, sometimes, from fungi, such as *Aspergillus niger* and *Candida albicans* (fungal otitis externa is most common in tropical regions). Occasionally, chronic otitis externa results from dermatologic conditions, such as seborrhea or psoriasis. Allergic reactions stemming from nickel or chromium earrings, chemicals in hair spray, cosmetics, hearing aids, and medications (such as sulfonamide and neomycin, which is commonly used to treat otitis externa) can also cause otitis externa.

Predisposing factors include:

- ◆ Swimming in contaminated water. (Cerumen creates a culture medium for the waterborne organism.)
- ◆ Cleaning the ear canal with a cotton swab, bobby pin, finger, or other foreign object. (This irritates the ear canal and, possibly, introduces the infecting microorganism.)
- ◆ Exposure to dust or hair-care products (such as hair spray or other irritants), which causes the patient to scratch the ear, excoriating the auricle and canal.
- ◆ Regular use of earphones, earplugs, or earmuffs, which trap moisture in the ear canal, creating a culture medium for infection (especially if earplugs don't fit properly).
- ◆ Chronic drainage from a perforated tympanic membrane.

- ◆ Perfumes or self-administered eardrops.

Pathophysiology

Otitis externa can take an acute or a chronic form. Acute disease commonly results from bacterial or fungal overgrowth in an ear canal subjected to excess moisture or to local trauma. Chronic disease often is part of a more generalized dermatologic or allergic problem. Symptoms of early acute and most chronic disease include pruritus and local discomfort. If left untreated, acute disease can be followed by canal edema, discharge, and pain, and eventually by extra-canal manifestations. Topical application of an acidifying solution is usually adequate in treating early disease. An antimicrobial-containing ototopical is the preferred treatment for later-stage acute disease, and oral antibiotic therapy is reserved for advanced disease or those who are immunocompromised. Preventive measures reduce recurrences and typically involve minimizing ear canal moisture, trauma, or exposure to materials that incite local irritation or contact dermatitis.

Complications

- ◆ Complete closure of the ear canal
- ◆ Significant hearing loss
- ◆ Otitis media
- ◆ Cellulitis
- ◆ Abscesses
- ◆ Stenosis

Signs and Symptoms

Acute otitis externa characteristically produces moderate to severe pain that's exacerbated by manipulating the auricle or tragus, clenching the teeth, opening the mouth, or chewing. Its other clinical effects may include fever, foul-smelling discharge, crusting in the external ear, regional cellulitis, partial hearing loss, and itching. It's usually difficult to view the tympanic membrane because of pain in the external canal. Hearing acuity is normal unless complete occlusion has occurred.

Fungal otitis externa may be asymptomatic, although *A. niger* produces a black or gray, blotting, paper-like growth in the ear canal. In chronic otitis externa, pruritus replaces pain, and scratching may lead to scaling and skin thickening. Aural discharge may also occur.

Diagnosis



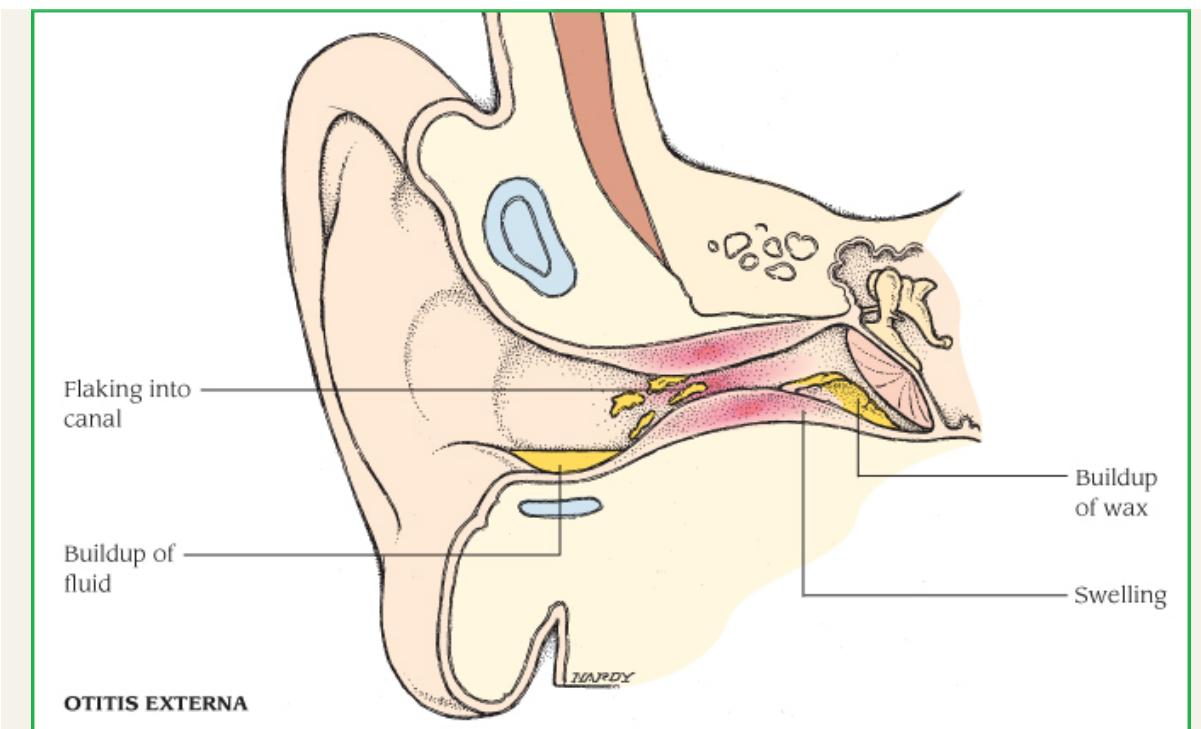
CONFIRMING DIAGNOSIS

Physical examination confirms otitis externa. In acute otitis externa, otoscopy reveals a swollen external ear canal (sometimes to the point of complete closure), preauricular lymphadenopathy (tender nodes anterior to the tragus, posterior to the ear, or in the upper neck), and, occasionally, regional cellulitis.

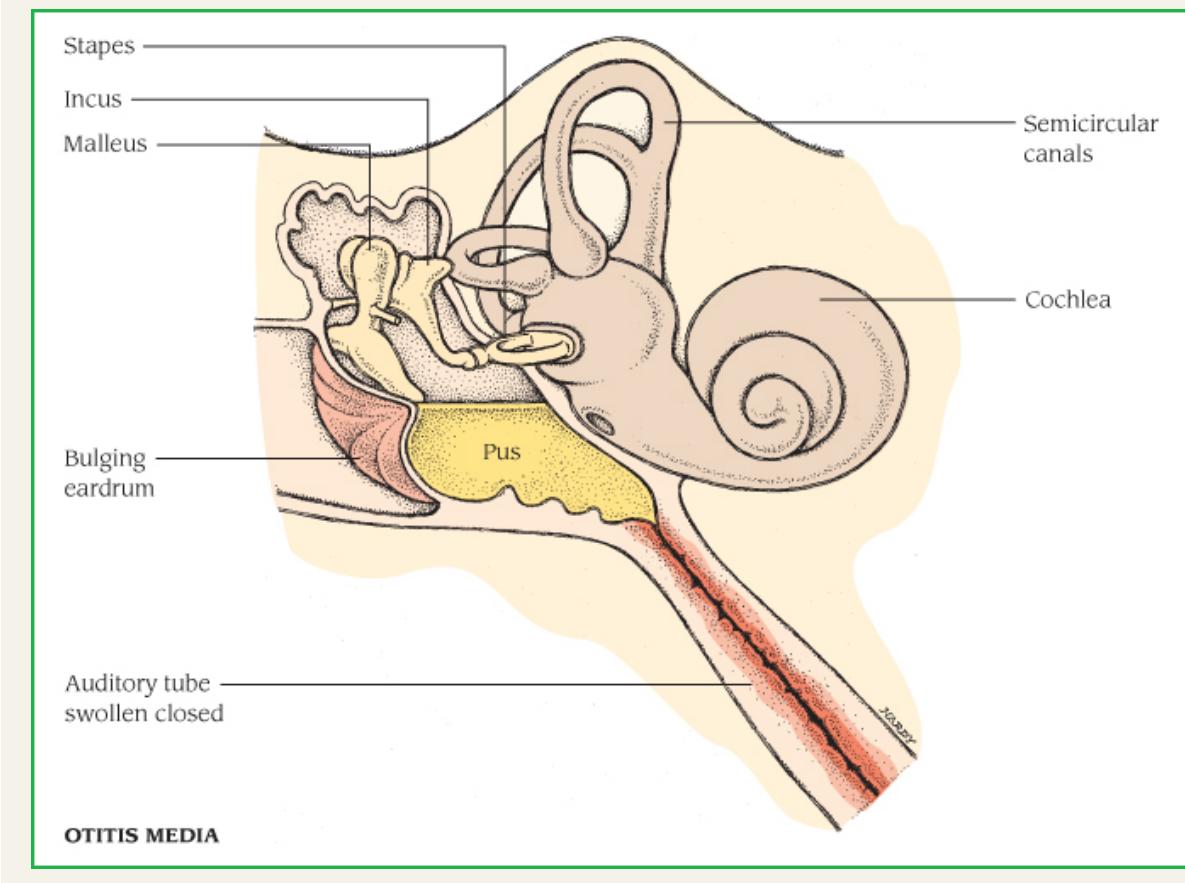
In fungal otitis externa, removal of the growth reveals thick red epithelium. Microscopic examination or culture and sensitivity tests can identify the causative organism and determine antibiotic treatment. Pain on palpation of the tragus or auricle distinguishes acute otitis externa from acute otitis media. (See *Differentiating acute otitis externa from acute otitis media*, page 629.)

Differentiating Acute Otitis Externa From Acute Otitis Media

Use the assessment findings shown below to help differentiate acute otitis externa from acute otitis media.



Acute Otitis Externa (Occurs Primarily In Summer)



Acute Otitis Media (Occurs Primarily In Winter)

In chronic otitis externa, physical examination reveals thick red epithelium in the ear canal. Severe chronic otitis externa may reflect underlying diabetes mellitus, hypothyroidism, or nephritis. Microscopic examination or culture and sensitivity tests can identify the causative organism and help in the determination of antibiotic treatment.

Treatment

To relieve the pain of acute otitis externa, treatment includes heat therapy to the preauricular region (heat lamp; hot, damp compresses; or a heating pad), aspirin or acetaminophen, and codeine. Instillation of antibiotic eardrops (with or without hydrocortisone) follows cleaning of the ear and removal of debris. However, a corticosteroid helps reduce the inflammatory response. If fever persists or regional cellulitis or tender postauricular adenopathy develops, a systemic antibiotic is necessary.

If the ear canal is too edematous for the instillation of eardrops, an ear wick may be used for the first few days.

Topical treatment is generally required for otitis externa, as systemic antibiotics alone aren't sufficient. Analgesics, such as acetaminophen or ibuprofen, may be required temporarily.

As with other forms of this disorder, fungal otitis externa necessitates careful cleaning of the ear. Application of a keratolytic or 2% salicylic acid in cream-containing nystatin may help treat otitis externa resulting from candidal organisms. Instillation of slightly acidic eardrops creates an unfavorable environment in the ear canal for most fungi as well as *Pseudomonas*. No specific treatment exists for otitis externa caused by *A. niger*, except repeated cleaning of the ear canal with baby oil.

In chronic otitis externa, primary treatment consists of cleaning the ear and removing debris. Supplemental therapy includes instillation of antibiotic eardrops or application of antibiotic ointment or cream (neomycin, bacitracin, or polymyxin B, possibly combined with hydrocortisone). Another ointment contains phenol, salicylic acid, precipitated sulfur, and petroleum jelly and produces exfoliative and antipruritic effects.

For mild chronic otitis externa, treatment may include instillation of antibiotic eardrops once or twice weekly and wearing of specially fitted earplugs while the patient is showering, shampooing, or swimming.

Special Considerations

If the patient has acute otitis externa:

- ◆ The patient shouldn't participate in any swimming activity.
- ◆ Have the patient return to the clinic in 1 week for evaluation of the tympanic membrane to make sure it's intact.
- ◆ Monitor vital signs, particularly temperature. Watch for and record the type and amount of aural drainage.
- ◆ Remove debris and gently clean the ear canal with mild Burow solution (aluminum acetate). Place a wisp of cotton soaked with solution into the ear, and apply a saturated compress directly to the auricle. Afterward, dry the ear gently but thoroughly. (In severe otitis externa, such cleaning may be delayed until after initial treatment with antibiotic eardrops.)
- ◆ To instill eardrops in an adult, grasp the helix and pull upward and backward to straighten the canal.
- ◆ Tell the patient to notify the physician if they develop an allergic reaction to the antibiotic drops or ointment, which may be indicated by increased swelling and discomfort of the area and worsening of other symptoms.



PEDIATRIC TIP *To instill eardrops in a child, pull the earlobe downward and backward. To ensure that the drops reach the epithelium, insert a wisp of cotton moistened with eardrops.*

If the patient has chronic otitis externa, clean the ear thoroughly. Use wet soaks intermittently on oozing or infected skin. If the patient has a chronic fungal infection, clean the ear canal well, and then apply an exfoliative ointment.

- ◆ Urge prompt treatment for otitis media to prevent perforation of the tympanic membrane. (See *Preventing otitis externa*.)



PREVENTION

PREVENTING OTITIS EXTERNA

Any patient who has experienced otitis externa should be taught to prevent a recurrence by avoiding irritants, such as hair-care products and earrings, and by avoiding cleaning the ears with cotton-tipped applicators or other objects. Encourage the patient to keep water out of the ears when showering or shampooing by using lamb's wool earplugs, coated with petroleum jelly. Also, parents of young children should be told that modeling clay makes a tight seal to prevent water from getting into the external ear canal.

In addition, when the patient goes swimming, keep their head above water or wear earplugs. After swimming, the patient should instill one or two drops of a mixture that is one-half 70% alcohol and one-half white vinegar to toughen the skin of the external ear canal.



ELDER TIP *If the patient is an elderly person or has diabetes, evaluate for malignant otitis externa.*



PEDIATRIC TIP *Children who have an intact tympanic membrane but are predisposed to otitis externa from swimming should instill two to three drops of a 1:1 solution of white vinegar and 70% ethyl alcohol into their ears before and after swimming.*

BENIGN TUMORS OF THE EAR CANAL

Benign tumors may develop anywhere in the ear canal. Common types include keloids, osteomas, and sebaceous cysts; their causes vary. (See *Causes and characteristics of benign ear tumors*.) These tumors seldom become malignant; with proper treatment, the prognosis is excellent.

Causes and Characteristics of Benign Ear Tumors

Tumor	Causes and incidence	Characteristics
Keloid	<ul style="list-style-type: none"> ◆ Surgery or trauma such as ear piercing ◆ Most common in blacks 	<ul style="list-style-type: none"> ◆ Hypertrophy and fibrosis of scar tissue ◆ Commonly recurs
Osteoma	<ul style="list-style-type: none"> ◆ Idiopathic growth ◆ Predisposing factor: swimming in cold water ◆ Three times more common in males than in females ◆ Seldom occurs before adolescence 	<ul style="list-style-type: none"> ◆ Bony outgrowth from wall of external auditory meatus ◆ Usually bilateral and multiple (exostoses) ◆ May be circumscribed or diffuse, nondisplaceable, nontender
Sebaceous cyst	<ul style="list-style-type: none"> ◆ Obstruction of a sebaceous gland 	<ul style="list-style-type: none"> ◆ Painless, circumscribed, round mass of variable size filled with oily, fatty, glandular secretions ◆ May occur on external ear and outer third of external auditory canal

Signs and Symptoms

A benign ear tumor is usually asymptomatic, unless it becomes infected, in which case pain, fever, or inflammation may result. (Pain is usually a sign of a malignant tumor.) If the tumor grows large enough to obstruct the ear canal by itself or through accumulated cerumen and debris, it may cause hearing loss and the sensation of pressure.

Diagnosis

 **CONFIRMING DIAGNOSIS** *Clinical features and patient history suggest a benign tumor of the ear canal; otoscopy confirms it. To rule out cancer, a biopsy may be necessary.*

Treatment

Generally, a benign tumor requires surgical excision if it obstructs the ear canal, is cosmetically undesirable, or becomes malignant.

Treatment for keloids may include surgery followed by repeated injections of long-acting steroids into the suture line. Excision must be complete, but even this may not prevent recurrence.

Surgical excision of an osteoma consists of elevating the skin from the surface of the bony growth and shaving the osteoma with a mechanical burr or drill.

Before surgery, a sebaceous cyst requires preliminary treatment with antibiotics, to reduce inflammation. To prevent recurrence, excision must be complete, including the sac or capsule of the cyst.

Special Considerations

Because treatment for benign ear tumors generally doesn't require hospitalization, focus care on emotional support and on providing appropriate patient education so that the patient follows the therapeutic plan properly when he's at home.

- ◆ Thoroughly explain diagnostic procedures and treatment to the patient and family. Reassure them and answer any questions they may have.
- ◆ After surgery, instruct the patient in good aural hygiene. Until the ear is completely healed, advise the patient not to insert anything into their ear or allow water to get into it. Suggest that they cover their ears with a cap when showering.
- ◆ Teach the patient how to recognize signs of infection, such as pain, fever, localized redness, and swelling. If the patient detects any of these signs, instruct to report them immediately.

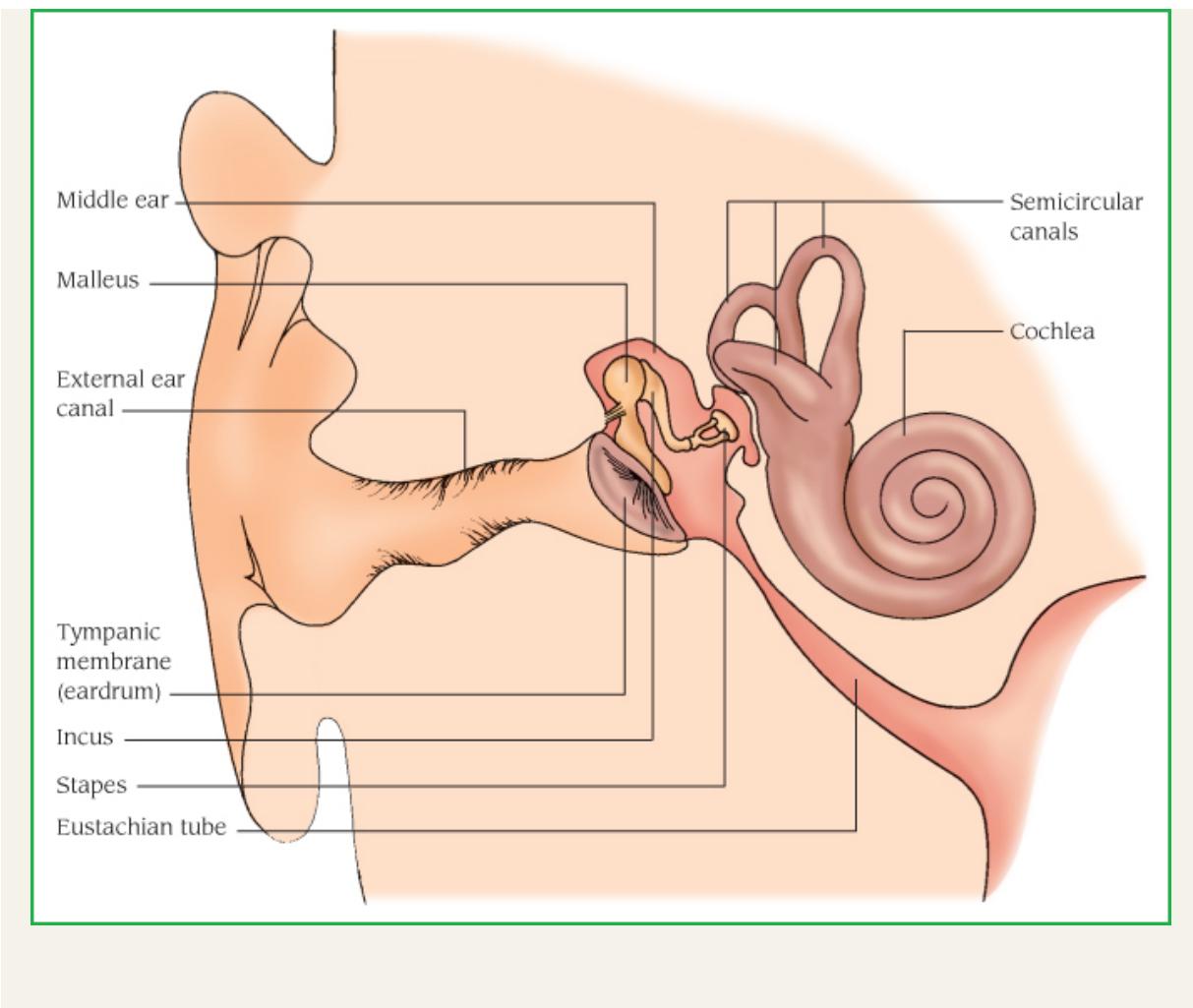
Middle Ear

OTITIS MEDIA

Otitis media, inflammation of the middle ear, may be suppurative or secretory, acute, persistent, unresponsive, or chronic. With prompt treatment, the prognosis for acute otitis media is excellent; however, prolonged accumulation of fluid within the middle ear cavity causes chronic otitis media and, possibly, perforation of the tympanic membrane. (See *Site of otitis media*.)

Site of Otitis Media

The common site of otitis media is shown below.



Chronic suppurative otitis media may lead to scarring, adhesions, and severe structural or functional ear damage. Chronic secretory otitis media, with its persistent inflammation and pressure, may cause conductive hearing loss.

Recurrent otitis media is defined as three near-acute otitis media episodes within 6 months or four episodes of acute otitis media within 1 year.

Otitis media with complications involves damage to middle ear structures (such as adhesions, retraction, pockets, cholesteatoma, and intratemporal and intracranial complications).

Causes and Incidence

Otitis media results from disruption of eustachian tube patency. In the suppurative form, respiratory tract infection, allergic reaction, nasotracheal intubation, or positional changes allow nasopharyngeal flora to reflux

through the eustachian tube and colonize the middle ear. Suppurative otitis media usually results from bacterial infection with pneumococcus, *Haemophilus influenzae* (the most common cause in children younger than age 6), *Moraxella catarrhalis*, beta-hemolytic streptococci, staphylococci (most common cause in children age 6 or older), or gram-negative bacteria. Predisposing factors include the normally wider, shorter, more horizontal eustachian tubes and increased lymphoid tissue in children, as well as anatomic anomalies. Chronic suppurative otitis media results from inadequate treatment for acute otitis episodes or from infection by resistant strains of bacteria or, rarely, tuberculosis.

Secretory otitis media results from obstruction of the eustachian tube. This causes a buildup of negative pressure in the middle ear that promotes transudation of sterile serous fluid from blood vessels in the membrane of the middle ear. Such effusion may be secondary to eustachian tube dysfunction from viral infection or allergy. It may also follow barotrauma (pressure injury caused by the inability to equalize pressures between the environment and the middle ear), as occurs during rapid aircraft descent in a person with an upper respiratory tract infection (URTI) or during rapid underwater ascent in scuba diving (barotitis media).

Chronic secretory otitis media follows persistent eustachian tube dysfunction from mechanical obstruction (adenoidal tissue overgrowth or tumors), edema (allergic rhinitis or chronic sinus infection), or inadequate treatment for acute suppurative otitis media.

Acute otitis media is common in children; its incidence rises during the winter months, paralleling the seasonal rise in nonbacterial respiratory tract infections. Chronic secretory otitis media most commonly occurs in children with tympanostomy tubes or those with a perforated tympanic membrane.

Pathophysiology

Acute otitis media is an acute infection of the middle ear, usually lasting less than 6 weeks. The primary cause of acute otitis media is usually *Streptococcus pneumoniae*, *H. influenzae*, and *M. catarrhalis*, which enter the middle ear after eustachian tube dysfunction caused by obstruction related to upper respiratory infections, inflammation of surrounding structures (e.g., sinusitis, adenoid hypertrophy), or allergic reactions (e.g., allergic rhinitis). Bacteria can enter the eustachian tube from contaminated

secretions in the nasopharynx and the middle ear from a tympanic membrane perforation. A purulent exudate is usually present in the middle ear, resulting in a conductive hearing loss.

Complications

- ◆ Spontaneous rupture of the tympanic membrane
- ◆ Persistent perforation
- ◆ Chronic otitis media
- ◆ Mastoiditis
- ◆ Abscesses
- ◆ Vertigo
- ◆ Permanent hearing loss

Signs and Symptoms

Clinical features of acute suppurative otitis media include severe, deep, throbbing pain (from pressure behind the tympanic membrane); signs of URTI (sneezing or coughing); mild to very high fever; hearing loss (usually mild and conductive); tinnitus; dizziness; nausea; and vomiting. Other possible effects include bulging of the tympanic membrane, with concomitant erythema, and purulent drainage in the ear canal from tympanic membrane rupture. However, many patients are asymptomatic.

Acute secretory otitis media produces a severe conductive hearing loss—which varies from 15 to 35 dB, depending on the thickness and amount of fluid in the middle ear cavity—and, possibly, a sensation of fullness in the ear and popping, crackling, or clicking sounds on swallowing or with jaw movement. Accumulation of fluid may also cause the patient to hear an echo when the patient speaks and to experience a vague feeling of top-heaviness.

The cumulative effects of chronic otitis media include thickening and scarring of the tympanic membrane, decreased or absent tympanic membrane mobility, cholesteatoma (a cystlike mass in the middle ear), and, in chronic suppurative otitis media, a painless, purulent discharge. The extent of associated conductive hearing loss varies with the size and type of tympanic membrane perforation and ossicular destruction.

If the tympanic membrane has ruptured, the patient may state that the pain has suddenly stopped. Complications may include abscesses (brain, subperiosteal, and epidural), sigmoid sinus or jugular vein thrombosis,

septicemia, meningitis, suppurative labyrinthitis, facial paralysis, and otitis externa.



PEDIATRIC TIP *The following factors increase a child's risk of developing otitis media:*

- ◆ *acute otitis media in the first year after birth (recurrent otitis media)*
- ◆ *day care*
- ◆ *family history of middle ear disease*
- ◆ *formula feeding*
- ◆ *male gender*
- ◆ *sibling history of otitis media*
- ◆ *smoking in the household*

Acute otitis media may not produce any symptoms in the first few months of life; irritability may be the only indication of earache.

Diagnosis

In acute suppurative otitis media, otoscopy reveals obscured or distorted bony landmarks of the tympanic membrane. Pneumatoscopy can show decreased tympanic membrane mobility, but this procedure is painful with an obviously bulging, erythematous tympanic membrane. The pain pattern is diagnostically significant: For example, in acute suppurative otitis media, pulling the auricle *doesn't* exacerbate the pain. A culture of the ear drainage identifies the causative organism.

In acute secretory otitis media, otoscopic examination reveals tympanic membrane retraction, which causes the bony landmarks to appear more prominent.

Examination also detects clear or amber fluid behind the tympanic membrane. If hemorrhage into the middle ear has occurred, as in barotrauma, the tympanic membrane appears blue-black.

In chronic otitis media, patient history discloses recurrent or unresolved otitis media. Otoscopy shows thickening, sometimes scarring, and decreased mobility of the tympanic membrane; pneumatoscopy shows decreased or absent tympanic membrane movement. A history of recent air travel or scuba diving suggests barotitis media.

Tympanocentesis for microbiologic diagnosis is recommended for treatment failures and may be followed by myringotomy. Tympanometry, acoustic reflex measurement, or acoustic reflexometry may be needed to document the presence of fluid in the middle ear. White blood cell count is higher in bacterial otitis media than in sterile otitis media. Mastoid X-rays or computed tomography (CT) scan of the head or mastoids may show the spreading of the infection beyond the middle ear.

Treatment

In acute suppurative otitis media, antibiotic therapy includes amoxicillin. In areas with a high incidence of beta-lactamase-producing *H. influenzae* and in patients who aren't responding to ampicillin or amoxicillin, amoxicillin/clavulanate potassium may be used. For those who are allergic to penicillin derivatives, therapy may include cefaclor or trimethoprim and sulfamethoxazole. Severe, painful bulging of the tympanic membrane usually necessitates myringotomy. Broad-spectrum antibiotics can help prevent acute suppurative otitis media in high-risk patients. A single dose of ceftriaxone 50 mg/kg is effective against major pathogens but is expensive and is reserved for very sick infants. In the patient with recurring otitis media, antibiotics must be used with discretion to prevent the development of resistant strains of bacteria.

In acute secretory otitis media, inflation of the eustachian tube using Valsalva maneuver several times a day may be the only treatment required. Otherwise, nasopharyngeal decongestant therapy may be helpful. It should continue for at least 2 weeks and, sometimes, indefinitely, with periodic evaluation. If decongestant therapy fails, myringotomy and aspiration of middle ear fluid are necessary, followed by insertion of a polyethylene tube into the tympanic membrane, for immediate and prolonged equalization of pressure. The tube falls out spontaneously after 9 to 12 months. Concomitant treatment for the underlying cause (such as elimination of allergens, or adenoidectomy for hypertrophied adenoids) may also be helpful in correcting this disorder.

Treatment for chronic otitis media includes broad-spectrum antibiotics, such as amoxicillin/clavulanate potassium or cefuroxime, for exacerbations of acute otitis media; elimination of eustachian tube obstruction; treatment for otitis externa; myringoplasty and tympanoplasty to reconstruct middle

ear structures when thickening and scarring are present; and, possibly, mastoidectomy. Cholesteatoma requires excision.

Special Considerations

- ◆ Explain all diagnostic tests and procedures. After myringotomy, maintain drainage flow. Don't place cotton or plugs deeply into the ear canal; however, sterile cotton may be placed loosely in the external ear to absorb drainage. To prevent infection, change the cotton whenever it gets damp, and wash hands before and after giving ear care. Watch for and report headache, fever, severe pain, or disorientation.
- ◆ After tympanoplasty, reinforce dressings and observe for excessive bleeding from the ear canal. Administer analgesics as needed. Warn the patient against blowing the nose or getting the ear wet when bathing.
- ◆ Encourage the patient to complete the prescribed course of antibiotic treatment. If nasopharyngeal decongestants are ordered, teach correct instillation.
- ◆ Suggest application of heat to the ear to relieve pain. (See *Preventing otitis media*, page 635.)



PREVENTION PREVENTING OTITIS MEDIA

For a patient recovering from otitis media at home, teach these guidelines to help prevent a recurrence.

Instruct the patient how to recognize upper respiratory infections and encourage early treatment. Encourage the patient to get a pneumococcal vaccine to prevent infections that can cause respiratory and aural infections.

Tell parents to wash children's toys and promote frequent hand washing. For infants, tell parents to avoid the use of pacifiers and encourage breast-feeding for at least the first 6 months of the child's life. It has been shown that breast milk contains antibodies that protect the infant from ear infections. If the child is bottle-fed, instruct the parents not to feed the infant in a supine position and not to put the child to bed with a bottle. Explain that doing so could

cause reflux of nasopharyngeal flora. Also, teach the parent to keep the child away from secondhand smoke.

To promote eustachian tube patency, instruct the patient to perform Valsalva maneuver several times a day, especially during airplane travel. Also, explain adverse reactions to the prescribed medications, emphasizing those that require immediate medical attention.

- ◆ Advise the patient with acute secretory otitis media to watch for and immediately report pain and fever—signs of secondary infection.
- ◆ Identify and treat allergies.

MASTOIDITIS

Mastoiditis is a bacterial infection and inflammation of the air cells of the mastoid antrum. Although the prognosis is good with early treatment, possible complications include meningitis, facial paralysis, brain abscess, and suppurative labyrinthitis.

Causes and Incidence

Bacteria that cause mastoiditis include pneumococci, *H. influenzae*, *M. catarrhalis*, beta-hemolytic streptococci, staphylococci, and gram-negative organisms. Mastoiditis is usually a complication of chronic otitis media; less frequently, it develops after acute otitis media. An accumulation of pus under pressure in the middle ear cavity results in necrosis of adjacent tissue and extension of the infection into the mastoid cells. Chronic systemic diseases or immunosuppression may also lead to mastoiditis. Anaerobic organisms play a role in chronic mastoiditis.



PREVENTION PREVENTING OTITIS EXTERNA

Any patient who has experienced otitis externa should be taught to prevent a recurrence by avoiding irritants, such as hair-care products and earrings, and by avoiding cleaning the ears with cotton-tipped applicators or other objects. Encourage the patient to keep water out of the ears when showering or shampooing by using lamb's wool earplugs, coated with petroleum jelly. Also, parents of young children should be

told that modeling clay makes a tight seal to prevent water from getting into the external ear canal.

In addition, when the patient goes swimming, keep their head above water or wear earplugs. After swimming, the patient should instill one or two drops of a mixture that is one-half 70% alcohol and one-half white vinegar to toughen the skin of the external ear canal.



PEDIATRIC TIP *Acute otitis media increases a child's risk of developing mastoiditis. If mastoiditis does occur in infants younger than age 1, the swelling occurs superior to the ear and pushes the auricle downward instead of outward. I.V. antibiotic treatment choice includes ampicillin or cefuroxime. Before antibiotics, mastoiditis was one of the leading causes of death in children; now, it's uncommon and less dangerous.*

Pathophysiology

Mastoiditis, inflammation of the mastoid process, a projection of the temporal bone just behind the ear. Mastoiditis, which primarily affects children, usually results from an infection of the middle ear (otitis media). Symptoms include pain and swelling behind the ear and over the side of the head and fever. An abscess may develop; this indicates that the infection has eroded the bone and destroyed its outer layer. Mastoiditis may affect other structures within the cranium and produce complications including meningitis, abscesses of the dura mater covering the brain; infection or blood clots of the lateral sinus (the large blood channel emptying into the internal jugular vein); and infection of the labyrinth (the inner ear) containing the balance and hearing apparatus. Mastoiditis is a rare condition that is treated by the early administration of antibiotics. Surgical drainage and removal of diseased bone may be necessary if antibiotics are not successful.

Complications

- ◆ Destruction of the mastoid bone
- ◆ Facial paralysis
- ◆ Meningitis
- ◆ Partial or complete hearing loss

Signs and Symptoms

Primary clinical features include a dull ache and tenderness in the area of the mastoid process, low-grade fever, headache, and a thick, purulent discharge that gradually becomes more profuse, possibly leading to otitis externa. Postauricular erythema and edema may push the auricle out from the head; pressure within the edematous mastoid antrum may produce swelling and obstruction of the external ear canal, causing conductive hearing loss.

Diagnosis

X-rays or CT scan of the mastoid area reveal hazy mastoid air cells; the bony walls between the cells appear decalcified. Audiometric testing may reveal a conductive hearing loss. Physical examination shows a dull, thickened, and edematous tympanic membrane, if the membrane isn't concealed by obstruction. During examination, the external ear canal is cleaned; persistent oozing into the canal indicates perforation of the tympanic membrane.

Treatment

Treatment for mastoiditis consists of intense parenteral antibiotic therapy. Reasonable initial antibiotic choices include ceftriaxone with nafcillin or clindamycin. If bone damage is minimal, myringotomy or tympanocentesis drains purulent fluid and provides a specimen of discharge for culture and sensitivity testing. Recurrent or persistent infection or signs of intracranial complications necessitate simple mastoidectomy. This procedure involves removal of the diseased bone and cleaning of the affected area, after which a drain is inserted.

A chronically inflamed mastoid requires radical mastoidectomy (excision of the posterior wall of the ear canal, remnants of the tympanic membrane, and the malleus and incus, although these bones are usually destroyed by infection before surgery). The stapes and facial nerve remain intact. Radical mastoidectomy, which is seldom necessary because of antibiotic therapy, doesn't drastically affect the patient's hearing because significant hearing loss precedes surgery. With either surgical procedure, the patient continues oral antibiotic therapy for several weeks after surgery and facility discharge. The prognosis is good if treatment is started early.

Indications for immediate surgical intervention include meningitis, brain abscess, cavernous sinus thrombosis, acute suppurative labyrinthitis, and facial palsy.

Special Considerations

- ◆ After simple mastoidectomy, give pain medication as needed. Check wound drainage and reinforce dressings (the surgeon usually changes the dressing daily and removes the drain in 72 hours). Check the patient's hearing, and watch for signs of complications, especially infection (either localized or extending to the brain); facial nerve paralysis, with unilateral facial drooping; bleeding; and vertigo, especially when the patient stands.
- ◆ After radical mastoidectomy, the wound is packed with petroleum gauze or gauze treated with an antibiotic ointment. Give pain medication before the packing is removed, on the fourth or fifth postoperative day.
- ◆ Because of stimulation to the inner ear during surgery, the patient may feel dizzy and nauseated for several days afterward. Keep the side rails up, and assist the patient with ambulation. Also, give antiemetics as needed.
- ◆ Before discharge, teach the patient and family how to change and care for the dressing. Urge compliance with the prescribed antibiotic treatment and promote regular follow-up care.
- ◆ If the patient is an elderly person or diabetic, evaluate for malignant otitis externa.



ELDER TIP Encourage the patient to seek early treatment for ear infections.

OTOSCLEROSIS

The most common cause of chronic, progressive conductive hearing loss, otosclerosis is the slow formation of spongy bone in the otic capsule, particularly at the oval window. With surgery, the prognosis is good.

Causes and Incidence

Otosclerosis appears to result from a genetic factor transmitted as an autosomal dominant trait; many patients report family histories of hearing loss (excluding presbycusis). Pregnancy may trigger onset of this condition.

Otosclerosis occurs in at least 10% of the U.S. population. It's three times more prevalent in females than in males, usually affecting people between ages 15 and 30. Whites are most susceptible.

Pathophysiology

Otosclerosis is a localized disease of bone remodeling within the otic capsule of the human temporal bone. Unlike other similar bone diseases, it does not occur outside of the temporal bone. These lesions seem to begin by resorption of stable otic capsule bone in adults, followed by a reparative phase with bone deposition. There are clearly genetic factors that lead to this disease, but measles virus infection and autoimmunity also may play contributing roles. Surgical correction of the conductive hearing loss is highly effective, but nonsurgical intervention has not yet been shown to prevent or slow the disease.

Complications

- ◆ Bilateral conductive hearing loss
- ◆ Taste disturbance

Signs and Symptoms

Spongy bone in the otic capsule immobilizes the footplate of the normally mobile stapes, disrupting the conduction of vibrations from the tympanic membrane to the cochlea. This causes progressive unilateral hearing loss, which may advance to bilateral deafness. Other symptoms include tinnitus and paracusis of Willis (hearing conversation better in a noisy environment than in a quiet one).

Diagnosis

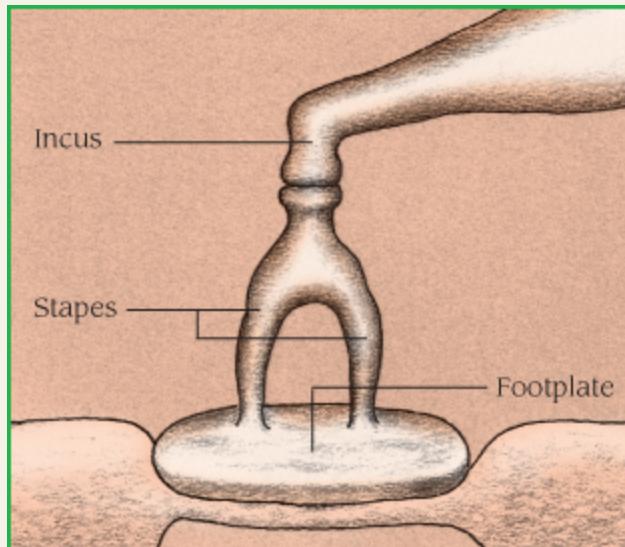
Early diagnosis is based on a Rinne test that shows bone conduction lasting longer than air conduction (normally, the reverse is true). As otosclerosis progresses, bone conduction also deteriorates. Audiometric testing reveals hearing loss ranging from 60 dB in early stages to total loss. Weber test detects sound lateralizing to the more affected ear. Physical examination reveals a normal tympanic membrane. Head CT scan and X-ray help distinguish otosclerosis from other causes of hearing loss.

Treatment

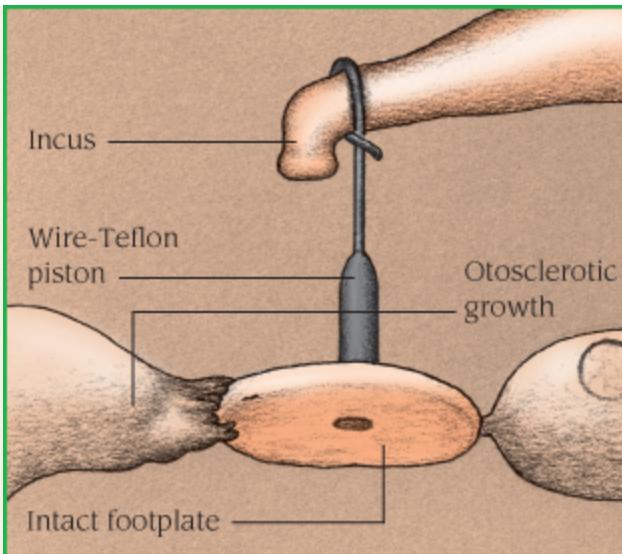
Treatment consists of stapedectomy (removal of the stapes) and insertion of a prosthesis to restore partial or total hearing. This procedure is performed on only one ear at a time, beginning with the ear that has suffered greater damage. Alternative surgery includes stapedotomy (creation of a small hole in the stapes' footplate), through which a wire and piston are inserted. (See *Types of stapedectomy*.) Recent procedural innovations involve laser surgery. Postoperatively, treatment includes antibiotics to prevent infection. If surgery isn't possible, a hearing aid (air conduction aid with molded ear insert receiver) enables the patient to hear conversation in normal surroundings, although this therapy isn't as effective as stapedectomy.

Types of Stapedectomy

Surgery may remove part or all of the stapes, depending on the extent of otosclerotic growth. It may be performed using various techniques. Two techniques used to implant prostheses are depicted below.

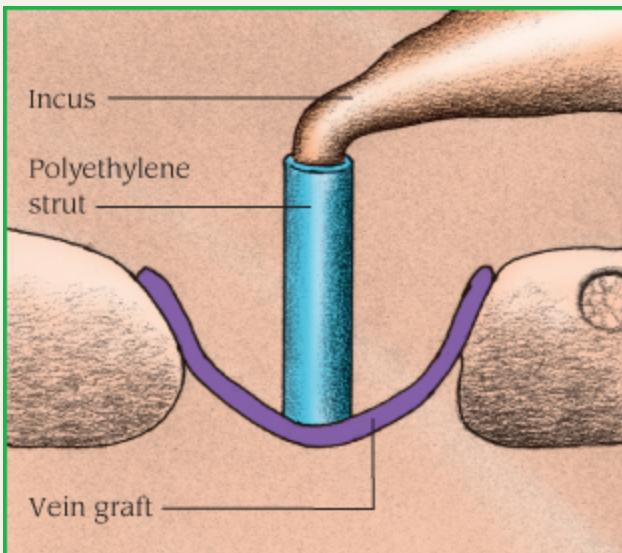


Normal Middle Ear



Partial Stapedectomy

Wire-Teflon Prosthesis



Total Stapedectomy

Vein Graft and Strut Prosthesis

Special Considerations

- During the first 24 hours after surgery, keep the patient supine, with the affected ear facing upward (to maintain the position of the graft). Enforce bed rest with bathroom privileges for 48 hours. Because the

patient may be dizzy, keep the side rails up, and assist them with ambulation. Assess for pain and vertigo, which may be relieved with repositioning or prescribed medication.

- ◆ Tell the patient that hearing won't return until edema subsides and packing is removed.



ALERT *Watch for and report postoperative facial drooping, which may indicate swelling of or around the facial nerve.*

- ◆ Before discharge, instruct the patient to avoid loud noises and sudden pressure changes (such as those that occur while diving or flying) until healing is complete (usually 6 months). Advise the patient not to blow their nose for at least 1 week to prevent contaminated air and bacteria from entering the eustachian tube.
- ◆ Stress the importance of protecting the ears against cold; avoiding any activities that provoke dizziness, such as straining, bending, or heavy lifting and, if possible, avoiding contact with anyone who has an URTI. Teach the patient and family how to change the external ear dressing (eye or gauze pad) and care for the incision. Emphasize the need to complete the prescribed antibiotic regimen and to return for scheduled follow-up care.

INFECTIOUS MYRINGITIS

Acute infectious myringitis is characterized by inflammation, hemorrhage, and effusion of fluid into the tissue at the end of the external ear canal and the tympanic membrane. This self-limiting disorder (resolving spontaneously within 3 days to 2 weeks) commonly follows acute otitis media or URTI.

Chronic granular myringitis, a rare inflammation of the squamous layer of the tympanic membrane, causes gradual hearing loss. Without specific treatment, this condition can lead to stenosis of the ear canal, as granulation extends from the tympanic membrane to the external ear.

Causes and Incidence

Acute infectious myringitis usually follows viral infection but may also result from infection with bacteria (pneumococcus, *H. influenzae*, beta-hemolytic streptococci, staphylococci) or any other organism that can cause

acute otitis media. Myringitis is a rare sequela of atypical pneumonia caused by *Mycoplasma pneumoniae*. The cause of chronic granular myringitis is unknown.

Acute infectious myringitis frequently occurs epidemically in children.

Pathophysiology

Bullous myringitis is a common condition characterized by vesicular eruptions of the tympanic membrane. In the majority of cases the condition is self-limited, although serious complications have been reported. The disease is primarily one of childhood, but is frequently seen in adults. Bullous myringitis is generally thought to be of viral origin, although several investigations have failed to establish this. Recent studies suggest a relationship to influenza virus and the Eaton agent, a pleuropneumonia-like organism (*M. pneumoniae*) known to be capable of producing primary atypical pneumonia.

Complications

- ◆ Gradual hearing loss
- ◆ Stenosis of the ear canal

Signs and Symptoms

Acute infectious myringitis begins with severe ear pain, commonly accompanied by tenderness over the mastoid process. Small, reddened, inflamed blebs form in the canal, on the tympanic membrane, and, with bacterial invasion, in the middle ear. Fever and hearing loss are rare unless fluid accumulates in the middle ear or a large bleb totally obstructs the external auditory meatus. Spontaneous rupture of these blebs may cause bloody discharge. Chronic granular myringitis produces pruritus, purulent discharge, and gradual hearing loss.

Diagnosis

 **CONFIRMING DIAGNOSIS** *Diagnosis of acute infectious myringitis is based on physical examination showing characteristic blebs and a typical patient history. Culture and sensitivity testing of exudate identifies secondary infection. In chronic granular myringitis, physical examination*

may reveal granulation extending from the tympanic membrane to the external ear.

Treatment

Hospitalization usually isn't required for acute infectious myringitis. Treatment consists of measures to relieve pain: analgesics, such as aspirin or acetaminophen, and application of heat to the external ear are usually sufficient, but severe pain may necessitate the use of codeine.



ALERT *Aspirin and combination aspirin products aren't recommended for people younger than age 19 during episodes of fever-causing illnesses because the use of aspirin has been linked to Reye syndrome.*

Systemic or topical antibiotics prevent or treat secondary infection. Incision of blebs and evacuation of serum and blood may relieve pressure and help drain exudate but don't speed recovery.

Treatment for chronic granular myringitis consists of systemic antibiotics or local anti-inflammatory/antibiotic combination eardrops, and surgical excision and cautery. If stenosis is present, surgical reconstruction is necessary.

Special Considerations

- ◆ Stress the importance of completing the prescribed antibiotic therapy.
- ◆ Teach the patient how to instill topical antibiotics (eardrops). When necessary, explain incision of blebs.



PREVENTION *Advise early treatment for acute otitis media.*

Inner Ear

MÉNIÈRE DISEASE

Ménière disease, a labyrinthine dysfunction also known as *endolymphatic hydrops*, produces severe vertigo, sensorineural hearing loss, and tinnitus. After multiple attacks over several years, this disorder leads to residual tinnitus and hearing loss. Usually, only one ear is involved.

Causes and Incidence

The exact cause of Ménière disease is unknown. It may result from overproduction or decreased absorption of endolymph, which causes endolymphatic hydrops or endolymphatic hypertension, with consequent degeneration of the vestibular and cochlear hair cells. This condition may also stem from autonomic nervous system dysfunction that produces a temporary constriction of blood vessels supplying the inner ear. In some cases, Ménière disease may be related to otitis media, syphilis, or head injury. Risk factors include recent viral illness, respiratory infection, stress, fatigue, use of prescription or nonprescription drugs (such as aspirin), and a history of allergies, smoking, and alcohol use. There also may be genetic risk factors: In some women, premenstrual edema may precipitate attacks of Ménière disease.

In the United States, about 100,000 people per year develop Ménière disease.

Pathophysiology

The pathophysiology of Ménière disease is not clearly understood. It was previously thought that Ménière was closely correlated with endolymphatic hydrops, a condition in which endolymph builds up because of an obstruction in the endolymphatic sac. Other possible origins of the disease are perisaccular fibrosis, atrophy of the endolymphatic sac and loss of epithelial integrity, hypoplasia of the vestibular aqueduct, and narrowing of the lumen of the endolymphatic duct.

Complications

- ◆ Tinnitus
- ◆ Partial to total hearing loss
- ◆ Permanent balance disability

Signs and Symptoms

Ménière disease produces three characteristic effects: severe episodic vertigo, tinnitus, and sensorineural hearing loss. A feeling of fullness or blockage in the ear is also common. Violent paroxysmal attacks last from 10 minutes to several hours. During an acute attack, other symptoms include severe nausea, vomiting, sweating, giddiness, and nystagmus.

Vertigo may cause loss of balance and falling to the affected side. Symptoms tend to wax and wane as the endolymphatic pressure rises and falls. To lessen these symptoms, the patient may assume a characteristic posture—lying on the side of the unaffected ear and looking in the direction of the affected ear.

Initially, the patient may be asymptomatic between attacks, except for residual tinnitus that worsens during an attack. Such attacks may occur several times a year, or remissions may last as long as several years. These attacks become less frequent as hearing loss progresses (usually unilaterally); they may cease when hearing loss is total. All symptoms are aggravated by motion.

Diagnosis

The presence of all three typical symptoms suggests Ménière disease. Audiometric studies indicate a sensorineural hearing loss and loss of discrimination and recruitment. Selected studies such as electronystagmography, electrocochleography, CT scan, magnetic resonance imaging, or X-rays of the internal meatus may be necessary for differential diagnosis.

Laboratory studies, including thyroid and lipid studies, may be performed to rule out other conditions such as *Treponema pallidum*.

Caloric testing may reveal loss or impairment of thermally induced nystagmus on the involved side. However, it's important not to overlook an acoustic tumor, which produces an identical clinical picture.

Treatment

Treatment with atropine may stop an attack in 20 to 30 minutes. Epinephrine or diphenhydramine may be necessary in a severe attack; dimenhydrinate, meclizine, diphenhydramine, or diazepam may be effective in a milder attack.

Long-term management includes use of a diuretic or vasodilator and restricted sodium intake (<2 g/day). A typical diuretic regime is hydrochlorothiazide 50 to 100 mg daily. Prophylactic antihistamines or mild sedatives (phenobarbital, diazepam) may also be helpful. If Ménière disease persists after 2 years of treatment, produces incapacitating vertigo, or resists medical management, surgery may be necessary. Destruction of the affected labyrinth permanently relieves symptoms but results in

irreversible hearing loss. Systemic streptomycin is reserved for the patient with bilateral disease for whom no other treatment can be considered. If a patient fails medical therapy and remains disabled by vertigo, surgical decompression of the endolymphatic sac may bring relief.

Special Considerations

If the patient is in the hospital during an attack of Ménière disease:

- ◆ Advise the patient against reading and exposure to glaring lights, to reduce dizziness.
- ◆ Keep the side rails of the patient's bed up to prevent falls. Tell the patient not to get out of bed or walk without assistance.
- ◆ Instruct the patient to avoid sudden position changes and any tasks that vertigo makes hazardous because an attack can begin quite rapidly. Hazardous activities, such as driving and climbing, should be avoided until 1 week after symptoms disappear.
- ◆ Before surgery, if the patient is vomiting, record fluid intake and output and characteristics of vomitus. Administer antiemetics as needed, and give small amounts of fluid frequently.
- ◆ After surgery, record intake and output carefully. Tell the patient to expect dizziness and nausea for 1 or 2 days after surgery. Give prophylactic antibiotics and antiemetics, as ordered.

LABYRINTHITIS

Labyrinthitis, an inflammation of the labyrinth of the inner ear, frequently incapacitates the patient by producing severe vertigo that lasts for 3 to 5 days; symptoms gradually subside over a 3- to 6-week period. Viral labyrinthitis is commonly associated with URTI.

Causes

Labyrinthitis is usually caused by viral infection. It may be a primary infection, the result of trauma, or a complication of influenza, otitis media, or meningitis. In chronic otitis media, cholesteatoma formation erodes the bone of the labyrinth, allowing bacteria to enter from the middle ear. Toxic drug ingestion is another possible cause of labyrinthitis and neuritis.

Pathophysiology

Labyrinthitis is an inflammatory response within the membranous inner ear structures in response to infection. It is a generally short-lived minor illness that has the potential to cause temporary or permanent disablement in terms of hearing loss. Other symptoms include nausea and vomiting, pain in the affected ear, vertigo, and fever.

Complications

- ◆ Meningitis
- ◆ Permanent hearing loss
- ◆ Permanent balance disability

Signs and Symptoms

Because the inner ear controls both hearing and balance, this infection typically produces severe vertigo (with any movement of the head) and sensorineural hearing loss. Vertigo begins gradually but peaks within 48 hours, causing loss of balance and falling in the direction of the affected ear. Other associated signs and symptoms include spontaneous nystagmus, with jerking movements of the eyes toward the unaffected ear, and nausea, vomiting, and giddiness. With cholesteatoma, signs of middle ear disease may appear. With severe bacterial infection, purulent drainage, increased salivation, generalized malaise, and perspiration can occur. To minimize symptoms such as giddiness and nystagmus, the patient may assume a characteristic posture—lying on the side of the unaffected ear and looking in the direction of the affected ear.

Diagnosis

A typical clinical picture and a history of URTI suggest labyrinthitis. Typical diagnostic measures include culture and sensitivity testing to identify the infecting organism, if purulent drainage is present, and audiometric testing. When an infectious etiology can't be found, additional testing must be done to rule out a brain lesion or Ménière disease.

Differentiation from other causes of dizziness or vertigo may include head CT scan or magnetic resonance imaging, audiology or audiometry testing, caloric stimulation tests, electronystagmography, electroencephalogram, and auditory-evoked potential studies.

Treatment

Symptomatic treatment includes bed rest, with the head immobilized between pillows, and antibiotics to combat diffuse purulent labyrinthitis. Oral fluids can prevent dehydration caused by vomiting. For severe nausea and vomiting, I.V. fluids may be necessary. Medications that help reduce symptoms include antihistamines, anticholinergics, sedative-hypnotics, and antiemetics; benzodiazepines help control vertigo.

When conservative management fails, treatment necessitates surgical excision of the cholesteatoma and drainage of the infected areas of the middle and inner ear. Prevention is possible by early and vigorous treatment for predisposing conditions, such as otitis media and any local or systemic infection.

Special Considerations

- ◆ Keep the side rails up to prevent falls. Tell the patient to keep still and rest during attacks and to avoid sudden position changes.
- ◆ If vomiting is severe, administer antiemetics as ordered. Record intake and output, and give I.V. fluids as ordered.
- ◆ During an attack, dim the lighting and tell the patient to avoid reading.
- ◆ Tell the patient that recovery may take as long as 6 weeks. During this time, they should limit activities that vertigo may make hazardous. Hazardous activities, such as driving and climbing, should be avoided until 1 week after symptoms disappear.
- ◆ If recovery doesn't occur within 4 to 6 weeks, a CT scan should be performed to rule out an intracranial lesion.

HEARING LOSS

Hearing loss results from a mechanical or nervous impediment to the transmission of sound waves. The major forms of hearing loss are classified as *conductive loss* (interrupted passage of sound from the external ear to the junction of the stapes and oval window), *sensorineural loss* (impaired cochlea or acoustic [eighth cranial] nerve dysfunction, causing failure of transmission of sound impulses within the inner ear or brain), or *mixed loss* (combined dysfunction of conduction and sensorineural transmission). Hearing loss may be partial or total and is calculated from this American Medical Association formula: Hearing is 1.5% impaired for every decibel that the pure tone average exceeds 25 dB.

Causes and Incidence

Congenital hearing loss may be transmitted as a dominant, autosomal dominant, autosomal recessive, or sex-linked recessive trait. Hearing loss in neonates may also result from trauma, toxicity, or infection during pregnancy or delivery. Predisposing factors include a family history of hearing loss or known hereditary disorders (e.g., otosclerosis), maternal exposure to rubella or syphilis during pregnancy, use of ototoxic drugs during pregnancy, prolonged fetal anoxia during delivery, and congenital abnormalities of the ears, nose, or throat. Premature or low-birth-weight neonates are most likely to have structural or functional hearing impairment; those with serum bilirubin levels above 20 mg/dL also risk hearing impairment from the toxic effect of high-serum bilirubin levels on the brain. In addition, trauma during delivery may cause intracranial hemorrhage and may damage the cochlea or the acoustic nerve.

Sudden deafness refers to sudden hearing loss in a person with no prior hearing impairment. This condition is considered a medical emergency because prompt treatment may restore full hearing. Its causes and predisposing factors may include:

- ◆ acute infections, especially mumps (most common cause of unilateral sensorineural hearing loss in children), and other bacterial and viral infections, such as rubella, rubeola, influenza, herpes zoster, and infectious mononucleosis; and mycoplasma infections
- ◆ blood dyscrasias (leukemia, hypercoagulation)
- ◆ head trauma or brain tumors
- ◆ metabolic disorders (diabetes mellitus, hypothyroidism, hyperlipoproteinemia)
- ◆ neurologic disorders (multiple sclerosis, neurosyphilis)
- ◆ ototoxic drugs (tobramycin, streptomycin, quinine, gentamicin, furosemide, ethacrynic acid)
- ◆ vascular disorders (hypertension, arteriosclerosis)

Noise-induced hearing loss, which may be transient or permanent, may follow prolonged exposure to loud noise (85 to 90 dB) or brief exposure to extremely loud noise (>90 dB). Such hearing loss is common in workers subjected to constant industrial noise and in military personnel, hunters, and rock musicians.

Presbycusis, an otologic effect of aging, results from a loss of hair cells in the organ of Corti. This disorder causes progressive, symmetrical, bilateral sensorineural hearing loss, usually of high-frequency tones.

Minor decreases in hearing are common after age 20. Some deafness due to nerve damage occurs in one of every five people by age 55.

Complications

- ◆ Tympanic membrane perforation
- ◆ Cholesteatoma
- ◆ Permanent hearing loss

Signs and Symptoms



PEDIATRIC TIP Although congenital hearing loss may produce no obvious signs of hearing impairment at birth, a deficient response to auditory stimuli generally becomes apparent within 2 to 3 days. As the child grows older, hearing loss impairs speech development.

Sudden deafness may be conductive, sensorineural, or mixed, depending on etiology. Associated clinical features depend on the underlying cause.

Noise-induced hearing loss causes sensorineural damage, the extent of which depends on the duration and intensity of the noise. Initially, the patient loses perception of certain frequencies (around 4,000 Hz) but, with continued exposure, eventually loses perception of all frequencies.



ELDER TIP Presbycusis usually produces tinnitus and the inability to understand the spoken word.



PEDIATRIC TIP The behavior of an infant who's deaf may appear normal and mislead the parents as well as the professional, especially if the infant has autosomal recessive deafness and is the first child of carrier parents.

Diagnosis



CONFIRMING DIAGNOSIS Patient, family, and occupational histories and a complete audiology examination usually provide ample evidence of

hearing loss and suggest possible causes or predisposing factors.

The Weber, Rinne, and specialized audiologic tests differentiate between conductive and sensorineural hearing loss.

Treatment

After the underlying cause is identified, therapy for congenital hearing loss refractory to surgery consists of developing the patient's ability to communicate through sign language, speech reading, or other effective means. Measures to prevent congenital hearing loss include aggressively immunizing children against rubella to reduce the risk of maternal exposure during pregnancy; educating pregnant women about the dangers of exposure to drugs, chemicals, or infection; and careful monitoring during labor and delivery to prevent fetal anoxia.

Treatment for sudden deafness requires prompt identification of the underlying cause. Prevention necessitates educating patients and healthcare professionals about the many causes of sudden deafness and the ways to recognize and treat them.

Hyperbilirubinemia can be controlled by phototherapy and exchange transfusions. Children need the appropriate immunizations. Medications that may be ototoxic should be used judiciously in children and monitored closely. Reduction of exposure to loud noises generally prevents high-frequency hearing loss.

In people with noise-induced hearing loss, overnight rest usually restores normal hearing in those who have been exposed to noise levels greater than 90 dB for several hours, but not in those who have been exposed to such noise repeatedly. As hearing deteriorates, treatment must include speech and hearing rehabilitation, because hearing aids are seldom helpful. Prevention of noise-induced hearing loss requires public recognition of the dangers of noise exposure and insistence on the use, as mandated by law, of protective devices such as earplugs during occupational exposure to noise.

Amplifying sound, as with a hearing aid, helps some patients with presbycusis, but many patients have an intolerance to loud noise and wouldn't be helped by a hearing aid.

Special Considerations

- ◆ When speaking to a patient with hearing loss who can read lips, stand directly in front of them, with the light on your face, and speak slowly

and distinctly. If possible, speak to the patient at eye level. Approach the patient within their visual range, and elicit their attention by raising your arm or waving; touching them may be unnecessarily startling.

- ◆ Make other staff members and facility personnel aware of the patient's disability and their established method of communication. Carefully explain diagnostic tests and facility procedures in a way the patient understands.
- ◆ Make sure the patient with a hearing loss is in an area where activity can be observed and approaching persons can be seen because such a patient depends totally on visual clues.
- ◆ When addressing an older patient, speak slowly and distinctly in a low tone; avoid shouting.
- ◆ Provide emotional support and encouragement to the patient learning to use a hearing aid. Teach them how the aid works and how to maintain it.
- ◆ Refer children with suspected hearing loss to an audiologist or otolaryngologist for further evaluation. Any child who fails a language screening examination should be referred to a speech pathologist for language evaluation. The child with a mild language delay may be involved with a home language-enrichment program.



PREVENTION *Watch for signs of hearing impairment in the patient receiving ototoxic drugs. Emphasize the danger of excessive exposure to noise; stress the danger to pregnant women of exposure to drugs, chemicals, and infection (especially rubella); and encourage the use of protective devices in a noisy environment.*

MOTION SICKNESS

Motion sickness is characterized by loss of equilibrium associated with nausea and vomiting that results from irregular or rhythmic movements or from the sensation of motion. Removal of the stimulus restores normal equilibrium. Motion sickness also can be induced when patterns of motion differ from what the patient has previously experienced.

Causes and Incidence

Motion sickness may result from excessive stimulation of the labyrinthine receptors of the inner ear by certain motions, such as those experienced in a

car, boat, plane, or swing. The disorder may also be caused by confusion in the cerebellum from conflicting sensory input—the visual stimulus (a moving horizon) conflicts with labyrinthine perception. Predisposing factors include tension or fear, offensive odors, or sights and sounds associated with a previous attack. Motion sickness from cars, elevators, trains, and swings is most common in children; from boats and airplanes, in adults. People who suffer from one kind of motion sickness aren't necessarily susceptible to other types.

Pathophysiology

Motion sickness is a syndrome that occurs when a patient is exposed to certain types of motion and usually resolves soon after its cessation. It is a common response to motion stimuli during travel. Although nausea is a hallmark symptom, the syndrome includes symptoms ranging from vague malaise to completely incapacitating illness. These symptoms, which can affect the patient's recreation, employment, and personal safety, can occur within minutes of experiencing motion and can last for several hours after its cessation.

Signs and Symptoms

Typically, motion sickness induces nausea, vomiting, headache, dizziness, fatigue, diaphoresis, and, occasionally, difficulty in breathing, leading to a sensation of suffocation. These symptoms usually subside when the precipitating stimulus is removed, but they may persist for several hours or days.

Treatment

The best way to treat the disorder is to stop the motion that's causing it. If this isn't possible, the patient will benefit from lying down, closing their eyes, and trying to sleep. Antiemetics, such as dimenhydrinate, cyclizine, meclizine, and scopolamine (transdermal patch), may prevent or relieve motion sickness.

Special Considerations



PEDIATRIC TIP *An elevated car seat may help prevent motion sickness in a child by allowing the patient to see out of the front window.*

- ◆ Tell the patient to avoid exposure to precipitating motion whenever possible.
- ◆ Instruct the patient to avoid eating or drinking for at least 4 hours before traveling and to take an antiemetic 30 to 60 minutes before traveling or to apply a transdermal scopolamine patch at least 4 hours before traveling. Tell the patient with prostate enlargement or glaucoma to consult a physician or pharmacist before taking antiemetics.



PREVENTION *The traveler can minimize motion sickness by sitting where motion is least apparent (near the wing section in an aircraft, in the center of a boat, or in the front seat of an automobile). Instruct the patient to keep the head still and eyes closed or focused on a distant and stationary object.*

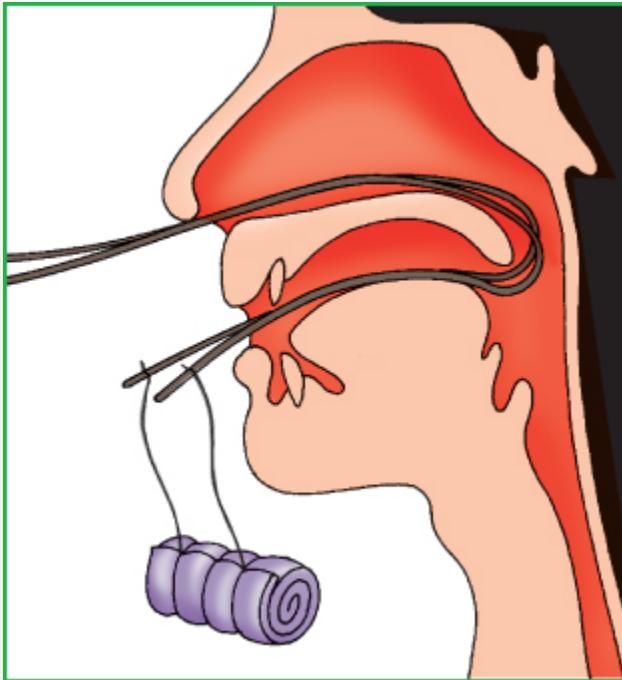
Nose

EPISTAXIS

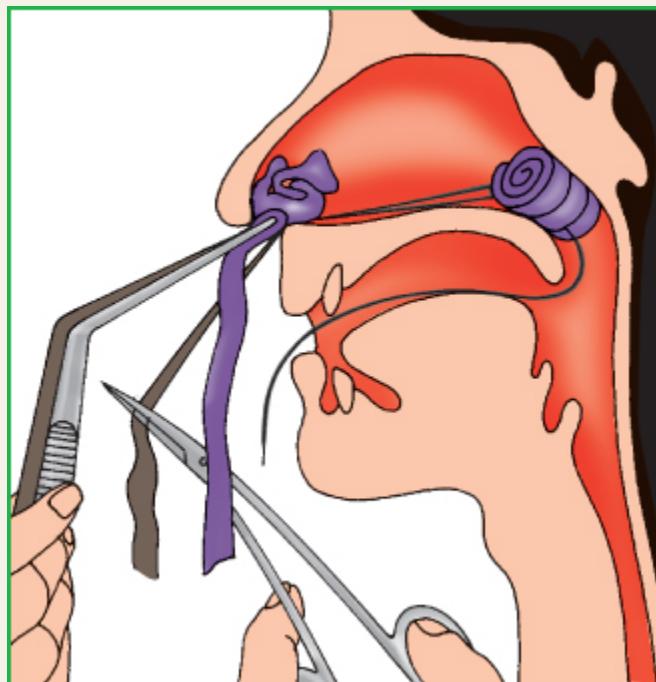
Epistaxis, commonly known as a *nosebleed*, may be a primary disorder or may occur secondary to another condition. Such bleeding in children generally originates in the anterior nasal septum and tends to be mild. In adults, such bleeding is most likely to originate in the posterior septum and can be severe enough to warrant nasal packing. (See *Inserting an anterior–posterior nasal pack*, pages 644 and 645.) Epistaxis is twice as common in children as in adults.

Inserting an Anterior–Posterior Nasal Pack

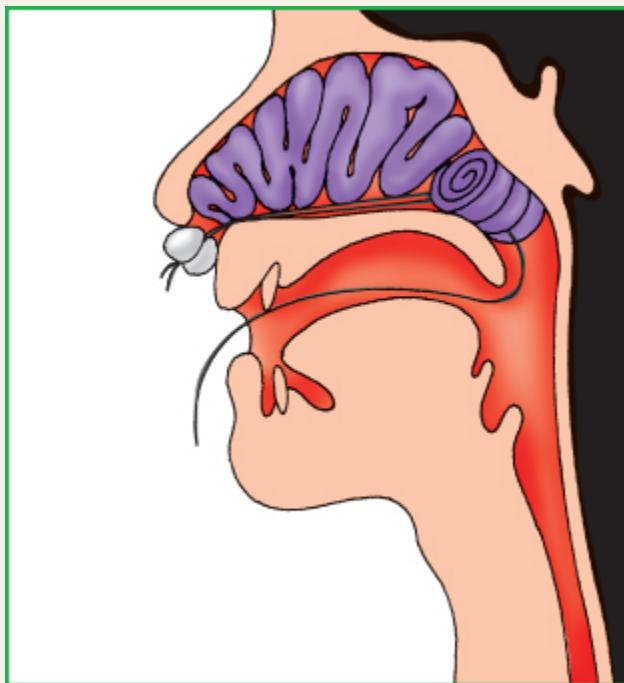
The first step in the insertion of an anterior–posterior nasal pack is the insertion of catheters into the nostrils. After the catheters are drawn through the mouth, a suture from the pack is tied to each (as shown above).



This positions the pack in place as the catheters are drawn back through the nostrils. Although the sutures are held tightly, packing is inserted into the anterior nose (as shown above).



The sutures are then secured around a dental roll; the middle suture extends from the mouth (as shown above) and is taped to the cheek.



Causes

Epistaxis usually follows trauma from external or internal causes: a blow to the nose, nose picking, or insertion of a foreign body; low humidity; or allergies, colds, or sinusitis. Less commonly, it follows polyps; acute or chronic infections such as sinusitis or rhinitis, which cause congestion and eventual bleeding of the capillary blood vessels; or inhalation of chemicals that irritate the nasal mucosa.

Predisposing factors include anticoagulant therapy, hypertension, long-term use of aspirin, overuse of decongestant nasal sprays, high altitudes and dry climates, sclerotic vessel disease, Hodgkin disease, hereditary hemorrhagic telangiectasia, neoplastic disorders (such as juvenile nasopharyngeal angiofibromas [JNAs]), scurvy, vitamin K deficiency, rheumatic fever, and blood dyscrasias (hemophilia, purpura, leukemia, and anemias).

Pathophysiology

Nosebleeds are due to the rupture of a blood vessel within the richly perfused nasal mucosa. Rupture may be spontaneous or initiated by trauma. An increase in blood pressure (e.g., due to general hypertension) tends to increase the duration of spontaneous epistaxis. Anticoagulant medication and disorders of blood clotting can promote and prolong bleeding. Spontaneous epistaxis is more common in the elderly as the nasal mucosa (lining) becomes dry and thin and blood pressure tends to be higher. The elderly are also more prone to prolonged nose bleeds as their blood vessels are less able to constrict and control the bleeding. Sometimes blood flowing from other sources of bleeding passes through the nasal cavity and exits the nostrils. It is thus blood coming from the nose but is not a true nosebleed, that is, not truly originating from the nasal cavity.

Complications

- ◆ Aspiration
- ◆ Shock

Signs and Symptoms

Blood oozing from the nostrils usually originates in the anterior nose and is bright red. Blood from the back of the throat originates in the posterior area and may be dark or bright red (commonly mistaken for hemoptysis due to expectoration). Epistaxis is generally unilateral, except when it's because of dyscrasia or severe trauma. In severe epistaxis, blood may seep behind the nasal septum; it may also appear in the middle ear and in the corners of the eyes.

Associated clinical effects depend on the severity of bleeding. Moderate blood loss may produce light-headedness, dizziness, and slight respiratory difficulty; severe hemorrhage causes hypotension, rapid and bounding pulse, dyspnea, and pallor. Bleeding is considered severe if it persists longer than 10 minutes after pressure is applied and causes blood loss as great as 1 L/hour in adults. Exsanguination (bleeding to death) from epistaxis is rare.

Diagnosis



CONFIRMING DIAGNOSIS *Although simple observation confirms epistaxis, inspection with a bright light and a nasal speculum is necessary to locate the site of bleeding.*

Relevant laboratory values include:

- ◆ gradual reduction in hemoglobin levels and hematocrit (HCT; usually inaccurate immediately following epistaxis because of hemoconcentration)
- ◆ decreased platelet count in the patient with blood dyscrasia
- ◆ prothrombin time and partial thromboplastin time showing a coagulation time twice the control, because of a bleeding disorder or anticoagulant therapy

Diagnosis must rule out underlying systemic causes of epistaxis, especially disseminated intravascular coagulation and rheumatic fever. Bruises or concomitant bleeding elsewhere probably indicates a hematologic disorder.



PEDIATRIC TIP *Bleeding tests are indicated if any of the following are present:*

- ◆ family history of a bleeding disorder
- ◆ medical history of easy bleeding
- ◆ spontaneous bleeding at other sites
- ◆ onset before age 2 or a drop in HCT due to epistaxis
- ◆ bleeding that won't clot with direct pressure by the physician
- ◆ bleeding that lasts longer than 30 minutes

Treatment

Mild nosebleeds that occur spontaneously may be treated by gently squeezing the soft portion of the nose between the thumb and finger for 5 to 10 minutes while the patient leans forward slightly (to avoid swallowing the blood) and breathes through the mouth.

For anterior bleeding, treatment consists of application to the bleeding site of a cotton ball saturated with epinephrine, and external pressure, followed by cauterization with electrocautery or a silver nitrate stick. If these measures don't control the bleeding, petroleum gauze nasal packing may be needed.

For posterior bleeding, therapy includes gauze packing inserted through the nose, or postnasal packing inserted through the mouth, depending on the

bleeding site. (Gauze packing generally remains in place for 24 to 48 hours; postnasal packing, 3 to 5 days.) An alternate method, the nasal balloon catheter, also controls bleeding effectively. Antibiotics may be appropriate if packing must remain in place for longer than 24 hours. If local measures fail to control bleeding, additional treatment may include supplemental vitamin K and, for severe bleeding, blood transfusions and surgical ligation or embolization of a bleeding artery.

Special Considerations

To control epistaxis:

- ◆ Elevate the patient's head to 45 degrees.
- ◆ Continuously compress the soft portion of the nares against the septum for 5 to 10 minutes. Apply an ice collar or cold, wet compresses to the nose. If bleeding continues after 10 minutes of pressure, notify the physician.
- ◆ Administer oxygen as needed, and monitor saturation levels.
- ◆ Monitor vital signs and skin color; record blood loss.
- ◆ Tell the patient to breathe through their mouth and not to swallow blood, talk, or blow their nose.
- ◆ Keep vasoconstrictors, such as phenylephrine, handy.
- ◆ Reassure the patient and their family that epistaxis usually looks worse than it is.



PREVENTION

- ◆ *Instruct the patient not to pick their nose or insert foreign objects into it, and to avoid bending or lifting. Emphasize the need for follow-up examinations and periodic blood studies after an episode of epistaxis. Advise prompt treatment for nasal infection or irritation.*
- ◆ *Suggest humidifiers for people who live in dry climates or at high elevations, or whose homes are heated with circulating hot air.*

SEPTAL PERFORATION AND DEVIATION

Perforated septum, a hole in the nasal septum between the two air passages, usually occurs in the anterior cartilaginous septum but may occur in the bony septum. Deviated septum, a shift from the midline, is common in most

adults. This condition may be severe enough to obstruct the passage of air through the nostrils. With surgical correction, the prognosis for either perforated or deviated septum is good.

Causes and Incidence

Generally, perforated septum is caused by traumatic irritation, most commonly resulting from excessive nose picking; less frequently, it results from repeated cauterization for epistaxis or from penetrating septal injury. It may also result from perichondritis, an infection that gradually erodes the perichondrial layer and cartilage, finally forming an ulcer that perforates the septum. Other causes of septal perforation include syphilis, tuberculosis, untreated septal hematoma, inhalation of irritating chemicals, cocaine snorting, use of nasal sprays, chronic nasal infections, nasal carcinoma, granuloma, and chronic sinusitis.

Deviated septum commonly develops during normal growth, as the septum shifts from one side to the other. Consequently, few adults have perfectly straight septa. Nasal trauma resulting from a fall, a blow to the nose, or surgery further exaggerates the deviation. Congenital deviated septum is rare.

Complications

- ◆ Hemorrhage
- ◆ Infections
- ◆ Deformity

Signs and Symptoms

A small septal perforation is usually asymptomatic but may produce a whistle on inspiration. A large perforation causes rhinitis, epistaxis, nasal crusting, and watery discharge.

The patient with a deviated septum may develop a crooked nose, as the midline deflects to one side. The predominant symptom of severe deflection, however, is nasal obstruction. Other manifestations include a sensation of fullness in the face, shortness of breath, stertor (snoring or laborious breathing), nasal discharge, recurring epistaxis, infection, sinusitis, and headache.

Diagnosis

Although clinical features suggest septal perforation or deviation, confirmation requires inspection of the nasal mucosa with a bright light and a nasal speculum.

Treatment

Symptomatic treatment for perforated septum includes decongestants to reduce nasal congestion by local vasoconstriction, local application of lanolin or petroleum jelly to prevent ulceration and crusting, and antibiotics to combat infection. Surgery may be necessary to graft part of the perichondrial layer over the perforation. Also, a plastic or Silastic “button” prosthesis may be used to close the perforation.

Symptomatic treatment for deviated septum usually includes analgesics to relieve headache, decongestants to minimize secretions, and, as necessary, vasoconstrictors, nasal packing, or cautery to control hemorrhage. Manipulation of the nasal septum at birth can correct congenital deviated septum.

Corrective surgical procedures include:

- ◆ reconstruction of the nasal septum by submucous resection to reposition the nasal septal cartilage and relieve nasal obstruction
- ◆ rhinoplasty to correct nasal structure deformity by intranasal incisions
- ◆ septoplasty to relieve nasal obstruction and enhance cosmetic appearance

Special Considerations

- ◆ In the patient with perforated septum, use a cotton applicator to apply petroleum jelly to the nasal mucosa to minimize crusting and ulceration.
- ◆ Warn the patient with perforation or severe deviation against blowing their nose. To relieve nasal congestion, instill saline nose drops and suggest use of a humidifier. Give decongestants as ordered.
- ◆ Prevention and patient education are the first lines of treatment for perforations caused by nasal sprays. Proper technique (aiming away from the nasal septum) should be reviewed. Medication should be withheld when scabs are noted on the septum.
- ◆ To treat epistaxis, have the patient sit upright, provide an emesis basin, and instruct the patient to expectorate any blood. Compress the outer portion of the nose against the septum for 10 to 15 minutes, and apply ice packs. If bleeding persists, notify the physician.

- ◆ If corrective surgery is scheduled, prepare the patient to expect postoperative facial edema, periorbital bruising, and nasal packing, which remains in place for 12 to 24 hours. The patient must breathe through the mouth. After surgery for deviated septum, the patient may also have a splint on their nose.
- ◆ To reduce or prevent edema and promote drainage, place the patient in semi-Fowler position, and use a cool-mist vaporizer to liquefy secretions and facilitate normal breathing. To lessen facial edema and pain, place crushed ice in a rubber glove or a small ice bag, and apply the glove or ice bag intermittently over the eyes and nose for 24 hours.
- ◆ Because the patient is breathing through the mouth, provide frequent mouth care.
- ◆ Change the mustache dressing or drip pad as needed. Record the color, consistency, and amount of drainage. While nasal packing is in place, expect slight, bright red drainage, with clots. After packing is removed, watch for purulent discharge, an indication of infection.
- ◆ Watch for and report excessive swallowing, hematoma, or a falling or flapping septum (depressed, or soft and unstable septum). Intranasal examination is necessary to detect hematoma formation. Any of these complications requires surgical correction.
- ◆ Administer sedatives and analgesics as needed. Because of its anticoagulant properties, aspirin is contraindicated after surgery for septal deviation or perforation.
- ◆ Nose blowing may cause bruising and swelling even after nasal packing is removed. After surgery, the patient must limit physical activity for 2 or 3 days and, if they are a smoker, they must stop smoking for at least 2 days.
- ◆ Instruct the patient to sneeze with their mouth open and to avoid bending over at the waist. (Advise the patient to stoop to pick up fallen objects.)

SINUSITIS

Sinusitis—*inflammation of the paranasal sinuses*—may be acute, subacute, chronic, allergic, or hyperplastic. Acute sinusitis usually results from the common cold and lingers in subacute form in only about 10% of patients. Chronic sinusitis follows persistent bacterial infection; allergic sinusitis accompanies allergic rhinitis; hyperplastic sinusitis is a combination of

purulent acute sinusitis and allergic sinusitis or rhinitis. The prognosis is good for all types.

Causes and Incidence

Sinusitis usually results from viral or bacterial infection. The bacteria responsible for acute sinusitis are usually pneumococci, other streptococci, *H. influenzae*, and *M. catarrhalis*. Staphylococci and gram-negative bacteria are more likely to cause sinusitis in chronic cases or in intensive care patients.

Predisposing factors include any condition that interferes with drainage and ventilation of the sinuses, such as chronic nasal edema, deviated septum, viscous mucus, nasal polyps, allergic rhinitis, nasal intubation, or debilitation due to chemotherapy, malnutrition, diabetes, blood dyscrasias, cystic fibrosis, human immunodeficiency virus or other immunodeficiency disorders, or chronic use of steroids. Bacterial invasion commonly occurs as a result of the conditions listed above or after a viral infection. It may also result from swimming in contaminated water.

Other risk factors for developing sinusitis include a history of asthma, overuse of nasal decongestants, presence of a foreign body in the nose, frequent swimming or diving, dental work, pregnancy, changes in altitude (flying or climbing), air pollution and smoke, gastroesophageal reflux disease (GERD), and having a deviated nasal septum, nasal bone spur, or polyp.

Each year, more than 30 million adults and children get sinusitis.



PEDIATRIC TIP *The incidence of both acute and chronic sinusitis increases in later childhood. Sinusitis may be more prevalent in children who have had tonsils and adenoids removed.*

Complications

- ◆ Meningitis
- ◆ Cavernous and sinus thrombosis
- ◆ Bacteremia or septicemia
- ◆ Brain abscess
- ◆ Osteomyelitis
- ◆ Mucocele

- ◆ Orbital cellulitis abscess

Pathophysiology

The most common cause of acute sinusitis is an URTI of viral origin. The viral infection can lead to inflammation of the sinuses that usually resolves without treatment in less than 14 days. If symptoms worsen after 3 to 5 days or persist for longer than 10 days and are more severe than normally experienced with a viral infection, a secondary bacterial infection is diagnosed. The inflammation can predispose to the development of acute sinusitis by causing sinus ostial blockage. Although inflammation in any of the sinuses can lead to blockade of the sinus ostia, the most commonly involved sinuses in both acute and chronic sinusitis are the maxillary and the anterior ethmoid sinuses. The nasal mucosa responds to the virus by producing mucus and recruiting mediators of inflammation, such as white blood cells, to the lining of the nose, which cause congestion and swelling of the nasal passages. The resultant sinus cavity hypoxia and mucus retention cause the cilia—which move mucus and debris from the nose—to function less efficiently, creating an environment for bacterial growth. If the acute sinusitis does not resolve, chronic sinusitis can develop from mucus retention, hypoxia, and blockade of the ostia. This promotes mucosal hyperplasia, continued recruitment of inflammatory infiltrates, and the potential development of nasal polyps.

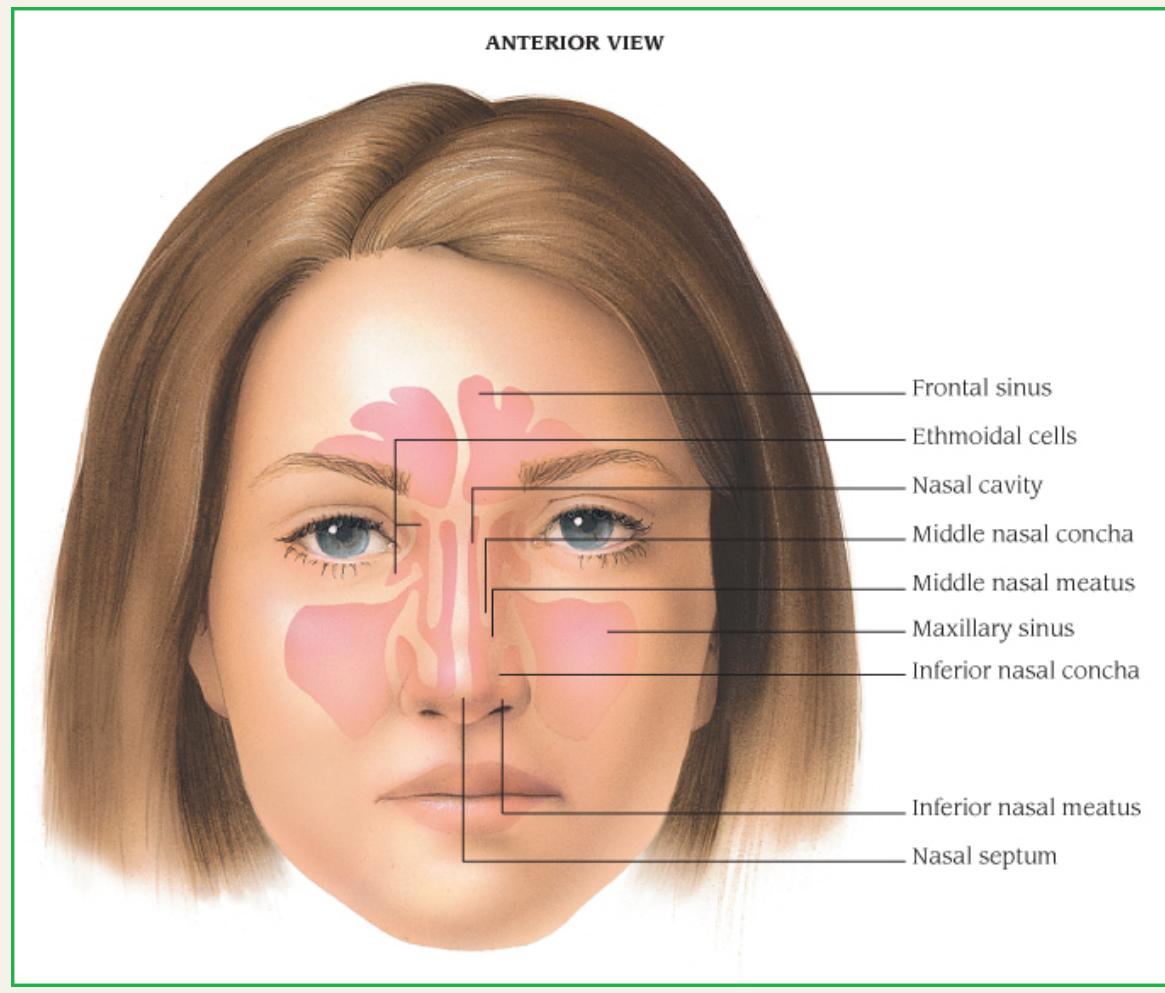
Signs and Symptoms

The primary indication of acute sinusitis is nasal congestion, followed by a gradual buildup of pressure in the affected sinus. For 24 to 48 hours after onset, nasal discharge may be present and later may become purulent. Associated symptoms include malaise, sore throat, headache, and low-grade fever of 99° to 99.5° F (37.2° to 37.5° C).

Characteristic pain depends on the affected sinus: maxillary sinusitis causes pain over the cheeks and upper teeth; ethmoid sinusitis, pain over the eyes; frontal sinusitis, pain over the eyebrows; and sphenoid sinusitis (rare), pain behind the eyes. (See *Locating the paranasal sinuses*, page 648.)

Locating The Paranasal Sinuses

The location of a patient's sinusitis pain indicates the affected sinus. For example, an infected maxillary sinus can cause tooth pain. (Note: The sphenoid sinus, which lies under the eye and above the soft palate, isn't depicted here.)



Purulent nasal drainage that continues for longer than 3 weeks after an acute infection subsides suggests *subacute sinusitis*. Other clinical features of the subacute form include nasal congestion, vague facial discomfort, fatigue, and a nonproductive cough.

Chronic sinusitis is defined as infection lasting longer than 8 weeks. The effects of chronic sinusitis are similar to those of acute sinusitis, but the chronic form causes continuous mucopurulent discharge.

The effects of *allergic sinusitis* are the same as those of allergic rhinitis. In both conditions, the prominent symptoms are sneezing, frontal headache, watery nasal discharge, and a stuffy, burning, itchy nose.

In *hyperplastic sinusitis*, bacterial growth on the diseased tissue causes pronounced tissue edema; thickening of the mucosal lining and the development of mucosal polyps combine to produce chronic stuffiness of the nose, in addition to headaches.

Diagnosis

The following measures are useful:

- ◆ Antral puncture promotes drainage of purulent material. It may also be used to provide a specimen for culture and sensitivity testing of the infecting organism, but it's seldom performed.
- ◆ Nasal examination reveals inflammation and pus.
- ◆ Palpation and percussion reveal tenderness of the frontal and maxillary sinuses.
- ◆ Sinus X-rays reveal cloudiness in the affected sinus, air and fluid, and any thickening of the mucosal lining.
- ◆ Transillumination is a simple diagnostic tool that involves shining a light into the patient's mouth with the lips closed around it. Infected sinuses look dark and normal sinuses transilluminate.
- ◆ Ultrasound, CT scan, magnetic resonance imaging, and X-rays aid in diagnosing suspected complications.

Treatment

Local decongestants usually are tried before systemic decongestants; steam inhalation may also be helpful. Antibiotics are necessary to combat purulent or persistent infection. Amoxicillin and amoxicillin/clavulanate potassium are usually the antibiotics of choice. Other possible therapy includes cefixime for responsive infections or if beta-lactamase-producing bacteria are present. Because sinusitis is a deep-seated infection, antibiotics should be given for 10 days to 2 weeks. Azithromycin is given for 5 days and may need to be repeated immediately. Local applications of heat may help to relieve pain and congestion. In subacute sinusitis, antibiotics and decongestants may be helpful.

Treatment for allergic sinusitis must include treatment for allergic rhinitis—avoidance measures, administration of antihistamines, identification of allergens by skin testing, and desensitization by immunotherapy. Severe allergic symptoms may require treatment with corticosteroids and epinephrine.

In both chronic sinusitis and hyperplastic sinusitis, using antihistamines, antibiotics, and a steroid nasal spray may relieve pain and congestion. If subacute infection persists, the sinuses may be irrigated. If irrigation fails to relieve symptoms, endoscopic sinus surgery may be required to obtain a histologic diagnosis, remove polyps, and provide adequate ventilation of the infected sinuses. Partial or total resection of the middle turbinate as well as more radical procedures, such as total sphenoethmoidectomy, may be performed.

Special Considerations

- ◆ Enforce bed rest, and encourage the patient to drink plenty of fluids to promote drainage. Don't elevate the head of the bed by more than 30 degrees.
- ◆ To relieve pain and promote drainage, apply warm compresses continuously, or four times daily for 2-hour intervals. Also, give analgesics and antihistamines as needed.
- ◆ Watch for and report complications, such as vomiting, chills, fever, edema of the forehead or eyelids, blurred or double vision, and personality changes.
- ◆ If surgery is necessary, tell the patient what to expect postoperatively: nasal packing will be in place for 12 to 24 hours following surgery; he'll have to breathe through the mouth and won't be able to blow the nose. After surgery, monitor for excessive drainage or bleeding and watch for complications.
- ◆ To prevent edema and promote drainage, place the patient in semi-Fowler position. To relieve edema and pain and to minimize bleeding, apply ice compresses or a rubber glove filled with ice chips over the nose and iced saline gauze over the eyes. Continue these measures for 24 hours.
- ◆ Frequently change the mustache dressing or drip pad, and record the consistency, amount, and color of drainage (expect scant, bright red, and clotty drainage).

- ◆ Because the patient will be breathing through their mouth, provide meticulous mouth care.
- ◆ Tell the patient that even after the packing is removed, nose blowing may cause bleeding and swelling. If the patient is a smoker, instruct them not to smoke for at least 2 or 3 days after surgery.
- ◆ Tell the patient to finish the prescribed antibiotics, even if their symptoms disappear.

NASAL POLYPS

Benign and edematous growths, nasal polyps are usually multiple, mobile, and bilateral. Nasal polyps may become large and numerous enough to cause nasal distention and enlargement of the bony framework, possibly occluding the airway.

Causes and Incidence

Nasal polyps are usually produced by the continuous pressure resulting from a chronic allergy that causes prolonged mucous membrane edema in the nose and sinuses. Other predisposing factors include chronic sinusitis, chronic rhinitis, and recurrent nasal infections.

Nasal polyps are more common in adults than in children and tend to recur. They're also commonly seen in patients with long-term allergic rhinitis and in patients with the aspirin triad (aspirin sensitivity, asthma, and nasal polyps). About 1 in 4 people with cystic fibrosis have nasal polyps.

Complication

- ◆ Airway obstruction

Signs and Symptoms

Nasal obstruction is the primary indication of nasal polyps. Such obstruction causes anosmia, a sensation of fullness in the face, nasal discharge, headache, and shortness of breath. Associated clinical features are usually the same as those of allergic rhinitis.

Diagnosis

Diagnosis of nasal polyps is aided by the following tests.

- ◆ Examination with a nasal speculum shows a dry, red surface, with clear or gray growths. Large growths may resemble tumors.
- ◆ X-rays of sinuses and nasal passages reveal soft-tissue shadows over the affected areas.



PEDIATRIC TIP Nasal polyps in children require further testing to rule out cystic fibrosis and Peutz–Jeghers syndrome.

Treatment

Intranasal glucocorticoids are the treatment of choice. Direct injection into the polyps may temporarily reduce the polyp. A short course of oral corticosteroids (such as prednisone) may be beneficial. Treatment for the underlying cause may include nasal antihistamines to control allergy, and antibiotic therapy if infection is present. Local application of an astringent shrinks hypertrophied tissue.

Surgical treatment should be considered after medical management has failed. A polypectomy is usually performed under a local anesthetic and the use of surgical lasers is becoming more popular; however, patients should be warned that nasal polyps have high recurrence rates. Continued recurrence may require surgical opening of the ethmoid, sphenoid, and maxillary sinuses and evacuation of diseased tissue.

Special Considerations

- ◆ Administer antihistamines, as ordered, for the patient with allergies. Prepare the patient for scheduled surgery by telling them what to expect postoperatively, such as nasal packing for 1 to 2 days after surgery.

After surgery:

- ◆ Watch for excessive bleeding or other drainage, and promote patient comfort.
- ◆ Elevate the head of the bed to facilitate breathing, reduce swelling, and promote adequate drainage. Change the mustache dressing or drip pad, as needed, and record the consistency, amount, and color of nasal drainage.
- ◆ Intermittently apply ice compresses over the nostrils to lessen swelling, prevent bleeding, and relieve pain.

- ◆ If nasal bleeding occurs—most likely after packing is removed—sit the patient upright, monitor vital signs, and advise not to swallow blood. Compress the outside of the nose against the septum for 10 to 15 minutes. If bleeding persists, nasal packing may be necessary.



PREVENTION *Instruct patients with allergies to avoid exposure to allergens and to take antihistamines at the first sign of an allergic reaction. Also, advise them to avoid overuse of nose drops and sprays.*

NASAL PAPILLOMAS

A papilloma is a benign epithelial tissue overgrowth within the intranasal mucosa. Inverted papillomas grow into the underlying tissue, usually at the junction of the antrum and the maxillary sinus; they generally occur singly but sometimes are associated with squamous cell cancer. Exophytic papillomas, which also tend to occur singly, arise from epithelial tissue, commonly on the surface of the nasal septum.

Pathophysiology

Inverted papilloma is a benign epithelial growth in the underlying stroma of the nasal cavity and paranasal sinuses. The pathogenesis of this lesion remains unclear, although allergy, chronic sinusitis, and viral infections have been suggested as possible causes.

Causes and Incidence

A papilloma may arise as a benign precursor of a neoplasm or as a response to tissue injury or viral infection, but its cause is unknown. Both types of papillomas are most prevalent in males. Recurrence is common, even after surgical excision.

Complications

- ◆ Severe respiratory distress (rare)
- ◆ Nasal drainage
- ◆ Infection

Signs and Symptoms

Both inverted and exophytic papillomas typically produce symptoms related to unilateral nasal obstruction—congestion, postnasal drip, headache, shortness of breath, dyspnea, and, rarely, severe respiratory distress, nasal drainage, and infection. Epistaxis is most likely to occur with exophytic papillomas. Occasionally hemorrhage may be the presenting symptom.

Diagnosis

On examination of the nasal mucosa, inverted papillomas usually appear large, bulky, highly vascular, and edematous; color varies from dark red to gray; and consistency, from firm to friable. Exophytic papillomas are usually raised, firm, and rubbery; pink to gray; and securely attached by a broad or pedunculated base to the mucous membrane.



PEDIATRIC TIP *Juvenile angiofibroma is a benign vascular tumor that arises in the nasopharynx and occurs most commonly in adolescent males. Nasal obstruction and hemorrhage may occur as with nasal papillomas. Any adolescent male who continues to have recurrent episodes of epistaxis should be assessed for juvenile angiofibroma. Medical management involves surgical excision, with preoperative embolization to reduce bleeding.*



CONFIRMING DIAGNOSIS *Tissue biopsy followed by histologic examination of excised tissue confirms the diagnosis.*

Treatment

The most effective treatment is wide surgical excision or diathermy, with careful inspection of adjacent tissues and sinuses to rule out extension. The use of surgical lasers is becoming more popular. Ibuprofen or acetaminophen and decongestants may relieve symptoms.

Special Considerations

- ◆ If bleeding occurs, have the patient sit upright, and expectorate blood into an emesis basin. Compress both sides of the nose against the septum for 10 to 15 minutes, and apply ice compresses to the nose. If the bleeding doesn't stop, notify the physician.



ALERT Check for airway obstruction. Place your hand under the patient's nostrils to assess air exchange and watch for signs of mild shortness of breath.

- ◆ If surgery is scheduled, tell the patient what to expect postoperatively. Instruct the patient not to blow the nose. (Packing is usually removed 12 to 24 hours after surgery.)
- ◆ Postoperatively, monitor vital signs and respiratory status. Use pulse oximetry to monitor oxygen saturation levels. As needed, administer analgesics and facilitate breathing with a cool-mist vaporizer. Provide mouth care.
- ◆ Frequently change the mustache dressing or drip pad, to ensure proper absorption of drainage. Record the type and amount of drainage. While the nasal packing is in place, expect scant, usually bright red, clotted drainage. Remember that the amount of drainage typically increases for a few hours after the packing is removed.
- ◆ Because papillomas tend to recur, tell the patient to seek medical attention at the first sign of nasal discomfort, discharge, or congestion that doesn't subside with conservative treatment.
- ◆ Encourage regular follow-up visits to detect early signs of recurrence.

ADENOID HYPERPLASIA

A fairly common childhood condition, adenoid hyperplasia (also known as *adenoid hypertrophy*) is enlargement of the lymphoid tissue of the nasopharynx. Normally, adenoidal tissue is small at birth ($\frac{3}{4}$ " to $1\frac{1}{4}$ " [2 to 3 cm]), grows until the child reaches adolescence, and then begins to slowly atrophy. In adenoid hyperplasia, however, this tissue continues to grow. Enlarged adenoids commonly accompany tonsillitis.

Causes and Incidence

The cause of adenoid hyperplasia is unknown, but contributing factors may include heredity, chronic infection, chronic nasal congestion, persistent allergy, insufficient aeration, and inefficient nasal breathing. Inflammation resulting from repeated infection increases the patient's risk of respiratory obstruction.

Complications

- ◆ Otitis media
- ◆ Conductive hearing loss
- ◆ Sinusitis
- ◆ Cor pulmonale
- ◆ Pulmonary arterial hypertension

Signs and Symptoms

Typically, adenoid hyperplasia produces symptoms of respiratory obstruction, especially mouth breathing, snoring at night, and frequent, prolonged nasal congestion. Persistent mouth breathing during the formative years produces voice alteration and distinctive changes in facial features—a slightly elongated face, open mouth, highly arched palate, shortened upper lip, and vacant expression.



PEDIATRIC TIP *Occasionally, the child is incapable of mouth breathing, snores loudly at night, and may eventually show effects of nocturnal respiratory insufficiency (sleep apnea), such as intercostal retractions and nasal flaring.*

Diagnosis



CONFIRMING DIAGNOSIS Nasopharyngoscopy or rhinoscopy confirms adenoid hyperplasia by allowing visualization of abnormal tissue. Lateral pharyngeal X-rays show an obliterated nasopharyngeal air column.

Treatment

Adenoidectomy is the treatment of choice for adenoid hyperplasia and is commonly recommended for the patient with prolonged mouth breathing, nasal speech, adenoid facies, recurrent otitis media, constant nasopharyngitis, and nocturnal respiratory distress. This procedure usually eliminates recurrent nasal infections and ear complications, and reverses any secondary hearing loss.

Special Considerations

Care requires sympathetic preoperative care and diligent postoperative monitoring.

Before surgery, do the following.

- ◆ Describe the facility routine, and arrange for the patient and their parents to tour relevant areas.
- ◆ Explain adenoidectomy to the child, using illustrations if necessary, and detail the recovery process. Advise them that they'll probably need to be hospitalized. If facility protocol allows, encourage one parent to stay with the child and participate in their care.

After surgery, take these steps.



ALERT *Maintain a patent airway. Position the child on their side, with their head down, to prevent aspiration of draining secretions.*

Frequently check the throat for bleeding. Be alert for vomiting of old, partially digested blood (coffee-ground vomitus). Closely monitor vital signs, and report excessive bleeding, rise in pulse rate, drop in blood pressure, tachypnea, and restlessness.

- ◆ If no bleeding occurs, offer cracked ice or water when the patient is fully awake.
- ◆ Tell the parents that their child may temporarily have a nasal voice.

VELOPHARYNGEAL INSUFFICIENCY

Velopharyngeal insufficiency results from failure of the velopharyngeal sphincter to close properly during speech, giving the voice a hypernasal quality and permitting nasal emission (air escape during pronunciation of consonants).

Causes and Incidence

Velopharyngeal insufficiency can result from an inherited palate abnormality, or it can be acquired from tonsillectomy, adenoidectomy, or palatal paresis. It commonly occurs in people who undergo cleft palate surgery and those with submucous cleft palates. Middle ear disease and hearing loss frequently accompany this disorder.

Pathophysiology

Velopharyngeal dysfunction (VPD) is a generic term, which describes a set of disorders resulting in the leakage of air into the nasal passages during

speech production. As a result, speech samples can demonstrate hypernasality, nasal emissions, and poor intelligibility. The finding of VPD can be secondary to several causes: anatomic, musculoneuronal, or behavioral/mislearning. To identify the etiology of VPD, patients must undergo a thorough velopharyngeal assessment comprised of perceptual speech evaluation and functional imaging, including video nasendoscopy and speech videofluoroscopy. These studies are then evaluated by a multidisciplinary team of specialists, who can decide on an optimal course for patient management. A treatment plan is developed and may include speech therapy, use of a prosthetic device, and/or surgical intervention. Different surgical options are discussed, including posterior pharyngeal flap, sphincter pharyngoplasty, Furlow palatoplasty, palatal re-repair, and posterior pharyngeal wall augmentation.

Complication

- ◆ Airway obstruction

Signs and Symptoms

Generally, this condition causes unintelligible speech, marked by hypernasality, nasal emission, poor consonant definition, and a weak voice. The patient experiences dysphagia and, if velopharyngeal insufficiency is severe, may regurgitate through the nose.

Diagnosis

Fiberoptic nasopharyngoscopy, which permits monitoring of velopharyngeal patency during speech, suggests this diagnosis. Ultrasound scanning, which shows air-tissue overlap, reflects the degree of velopharyngeal sphincter incompetence (an opening $>20\text{ mm}^2$ results in unintelligible speech). Videofluoroscopy simultaneously records the movement of the velopharyngeal sphincter and the patient's speech.

Treatment

Treatment consists of corrective surgery, usually at age 6 or 7. The preferred surgical method is the pharyngeal flap procedure, which diverts a tissue flap from the pharynx to the soft palate. Children with velopharyngeal

insufficiency shouldn't have adenoidectomy except in cases of life-threatening obstruction.

Other appropriate surgical procedures include:

- ◆ augmentation pharyngoplasty, which narrows the velopharyngeal opening by enlarging the pharyngeal wall with a retropharyngeal implant
- ◆ palatal push-back, which separates the hard and soft palates to allow insertion of an obturator, thus lengthening the soft palate
- ◆ pharyngoplasty, which rotates pharyngeal flaps to lengthen the soft palate and narrow the pharynx
- ◆ velopharyngeal sphincter reconstruction, which uses free muscle implantation to reconstruct the sphincter

Surgery eliminates hypernasality and nasal emission, but speech abnormalities persist and usually necessitate speech therapy. Immediate postoperative therapy includes antibiotics and a clear, liquid diet for the first 3 days, followed by a soft diet for 2 weeks.

Special Considerations

- ◆ After surgery for velopharyngeal insufficiency, maintain a patent airway (nasopharynx edema may obstruct the airway). Position the patient on their side, and suction the dependent side of their mouth, avoiding the pharynx.
- ◆ Control postoperative agitation, which may provoke pharyngeal bleeding, with sedation, as ordered.
- ◆ Administer high-humidity oxygen as ordered.
- ◆ Monitor vital signs frequently, and report any changes immediately. Observe for bleeding from the mouth or nose. Check intake and output, and watch for signs of dehydration.
- ◆ Advise the patient that preoperative and postoperative speech therapy require time and effort, but with persistence and practice, speech will improve. Before discharge, emphasize the importance of completing the prescribed antibiotic therapy.

Throat

PHARYNGITIS

The most common throat disorder, pharyngitis is an acute or chronic inflammation of the pharynx. It frequently accompanies the common cold.

Causes and Incidence

Pharyngitis is usually caused by a virus. The most common bacterial cause is group A beta-hemolytic streptococci. Other common causes include *Mycoplasma* and *Chlamydia*. In up to 30% of cases, no organism is identified.

Pharyngitis is widespread among adults who live or work in dusty or very dry environments, use their voices excessively, habitually use tobacco or alcohol, or suffer from chronic sinusitis, persistent coughs, or allergies.

Pathophysiology

Pharyngitis is an inflammatory illness of the mucous membranes and underlying structures of the throat (pharynx). Inflammation usually involves the nasopharynx, uvula, soft palate, and tonsils. The illness can be caused by bacteria, viruses, mycoplasmas, fungi, and parasites and by recognized diseases of uncertain causes. Infection by *Streptococcus* bacteria may be a complication arising from a common cold. The symptoms of streptococcal pharyngitis (commonly known as strep throat) are generally redness and swelling of the throat, a pustulant fluid on the tonsils or discharged from the mouth, extremely sore throat that is felt during swallowing, swelling of lymph nodes, and a slight fever; sometimes in children there are abdominal pain, nausea, headache, and irritability. Diagnosis is established by a detailed medical history and by physical examination; the cause of pharyngeal inflammation can be determined by throat culture. Usually only the symptoms can be treated—with throat lozenges to control sore throat and acetaminophen or aspirin to control fever. If a diagnosis of streptococcal infection is established by culture, appropriate antibiotic therapy, usually with penicillin, is instituted. Within approximately 3 days the fever leaves; the other symptoms may persist for another 2 to 3 days.

Complications

- ◆ Otitis media
- ◆ Sinusitis
- ◆ Mastoiditis
- ◆ Rheumatic fever

- ◆ Nephritis

Signs and Symptoms

Pharyngitis produces a sore throat and slight difficulty in swallowing. Swallowing saliva is usually more painful than swallowing food. Pharyngitis may also cause the sensation of a lump in the throat as well as a constant, aggravating urge to swallow. Associated features may include mild fever, headache, muscle and joint pain, coryza, and rhinorrhea. Uncomplicated pharyngitis usually subsides in 3 to 10 days.



PEDIATRIC TIP *More than 90% of cases of sore throat and fever in children are of viral origin. Associated symptoms usually include runny nose and nonproductive cough.*

Diagnosis

Physical examination of the pharynx reveals generalized redness and inflammation of the posterior wall, and red, edematous mucous membranes studded with white or yellow follicles. Exudate is usually confined to the lymphoid areas of the throat, sparing the tonsillar pillars. Bacterial pharyngitis usually produces a large amount of exudate.

A throat culture may be performed to identify bacterial organisms that may be the cause of the inflammation.

Treatment

Treatment for acute viral pharyngitis is usually symptomatic and consists mainly of rest, warm saline gargles, throat lozenges containing a mild anesthetic, plenty of fluids, and analgesics as needed. If the patient can't swallow fluids, I.V. hydration may be required.

Suspected bacterial pharyngitis requires rigorous treatment with penicillin or another broad-spectrum antibiotic because *Streptococcus* is the chief infecting organism. Antibiotic therapy should continue for 48 hours until culture results are back. If the culture (or a rapid strep test) is positive for group A beta-hemolytic streptococci, or if bacterial infection is suspected despite negative culture results, penicillin therapy should be continued for 10 days. This is to prevent the sequelae of acute rheumatic fever.

Chronic pharyngitis requires the same supportive measures as acute pharyngitis but with greater emphasis on eliminating the underlying cause, such as an allergen. Preventive measures include adequate humidification and avoiding excessive exposure to air-conditioning. In addition, the patient should be urged to stop smoking.

Special Considerations

- ◆ Administer analgesics and warm saline gargles, as ordered and as appropriate.
- ◆ Encourage the patient to drink plenty of fluids. Scrupulously monitor intake and output, and watch for signs of dehydration.
- ◆ Provide meticulous mouth care to prevent dry lips and oral pyoderma, and maintain a restful environment.
- ◆ Obtain throat cultures, and administer antibiotics as needed. If the patient has acute bacterial pharyngitis, emphasize the importance of completing the full course of antibiotic therapy.
- ◆ Teach the patient with chronic pharyngitis how to minimize sources of throat irritation in the environment, such as by using a bedside humidifier.
- ◆ Refer the patient to a self-help group to stop smoking if appropriate.
- ◆ Children attending school should receive at least 24 hours of therapy before being allowed to return to school.
- ◆ If the patient has exhibited three or more documented bacterial infections within 6 months, consider daily penicillin prophylaxis during the winter months. Also, consider treatment of carriers who live in closed or semiclosed communities.

TONSILLITIS

Tonsillitis—inflammation of the tonsils—can be acute or chronic. The uncomplicated acute form usually lasts 4 to 6 days. The presence of proven chronic tonsillitis justifies tonsillectomy, the only effective treatment. Tonsils tend to hypertrophy during childhood and atrophy after puberty.

Causes and Incidence

Tonsillitis generally results from infection with group A beta-hemolytic streptococci but can result from other bacteria or viruses or from oral

anaerobes. It commonly affects children between ages 5 and 10.

Pathophysiology

Tonsillitis is an inflammatory infection of the tonsils caused by invasion of the mucous membrane by microorganisms, usually hemolytic streptococci or viruses. The symptoms are sore throat, difficulty in swallowing, fever, malaise, and enlarged lymph nodes on both sides of the neck. The infection lasts about 5 days. The treatment includes bed rest until the fever has subsided, isolation to protect others from the infection, and warm throat irrigations or gargles with a mild antiseptic solution. Antibiotics or sulfonamides or both are prescribed in severe infections to prevent complications.

Complications

- ◆ Chronic upper airway obstruction
- ◆ Sleep apnea
- ◆ Cor pulmonale
- ◆ Failure to thrive
- ◆ Eating or swallowing disorders
- ◆ Febrile seizures
- ◆ Otitis media
- ◆ Cardiac valvular disease
- ◆ Peritonsillar abscesses
- ◆ Bacterial endocarditis
- ◆ Cervical lymph node abscesses

Signs and Symptoms

Acute tonsillitis commonly begins with a mild to severe sore throat. A very young child, unable to describe a sore throat, may stop eating. Tonsillitis may also produce dysphagia, fever, swelling and tenderness of the lymph glands in the submandibular area, muscle and joint pain, chills, malaise, headache, and pain (frequently referred to the ears). Excess secretions may elicit the complaint of a constant urge to swallow; the back of the throat may feel constricted. Such discomfort usually subsides after 72 hours.

Chronic tonsillitis produces a recurrent sore throat and purulent drainage in the tonsillar crypts. Frequent attacks of acute tonsillitis may also occur.

Complications include obstruction from tonsillar hypertrophy and peritonsillar abscess.

Diagnosis



CONFIRMING DIAGNOSIS *Diagnostic confirmation requires a thorough throat examination that reveals:*

- ◆ *generalized inflammation of the pharyngeal wall*
- ◆ *swollen tonsils that project from between the pillars of the fauces and exude white or yellow follicles*
- ◆ *purulent drainage when pressure is applied to the tonsillar pillars*
- ◆ *possible edematous and inflamed uvula*

Culture may determine the infecting organism and indicate appropriate antibiotic therapy. Leukocytosis is also usually present. Differential diagnosis rules out infectious mononucleosis and diphtheria.

Treatment

Treatment for acute tonsillitis requires rest, adequate fluid intake, administration of ibuprofen or acetaminophen, and, for bacterial infection, antibiotics. When the causative organism is group A beta-hemolytic streptococcus, penicillin is the drug of choice (another broad-spectrum antibiotic may be substituted). Most oral anaerobes also respond to penicillin. To prevent complications, antibiotic therapy should continue for 10 to 14 days.

Chronic tonsillitis or the development of complications (obstructions from tonsillar hypertrophy, peritonsillar abscess) may require a tonsillectomy, but only after the patient has been free from tonsillar or respiratory tract infections for 3 to 4 weeks.

Special Considerations

- ◆ Despite dysphagia, urge the patient to drink plenty of fluids, especially if the patient has a fever. Offer a child ice cream and flavored drinks and ices. Suggest gargling with warm salt water to soothe the throat, unless it exacerbates pain. Make sure the patient and parents understand the importance of completing the prescribed course of antibiotic therapy.

- ◆ Before tonsillectomy, explain to the adult patient that a local anesthetic prevents pain but allows a sensation of pressure during surgery. Warn the patient to expect considerable throat discomfort and some bleeding postoperatively. Watch for continuous swallowing, a sign of heavy bleeding.
- ◆ Postoperatively, maintain a patent airway. To prevent aspiration, place the patient on their side. Monitor vital signs frequently, and check for bleeding. Immediately report excessive bleeding, increased pulse rate, or dropping blood pressure. After the patient is fully alert and the gag reflex has returned, allow them to drink water. Later, urge them to drink plenty of nonirritating fluids, to ambulate, and to take frequent deep breaths to prevent pulmonary complications. Give pain medication as needed.
- ◆ Before discharge, provide the patient or their parents with written instructions on home care. Tell them to expect a white scab to form in the throat between 5 and 10 days postoperatively, and to report bleeding, ear discomfort, or a fever that lasts longer than 3 days.



PEDIATRIC TIP *For the pediatric patient, keep your explanation simple and nonthreatening. Show the patient the operating and recovery areas, and briefly explain the facility routine. Most facilities allow one parent to stay with the child.*

THROAT ABSCESES

Throat abscesses may be peritonsillar (quinsy) or retropharyngeal. Peritonsillar abscesses form in the connective tissue space between the tonsil capsule and the constrictor muscle of the pharynx. Retropharyngeal abscesses, or abscesses of the potential space, form between the posterior pharyngeal wall and the prevertebral fascia. With treatment, the prognosis for both types of abscesses is good.

Causes and Incidence

Peritonsillar abscess is a complication of acute tonsillitis, usually after streptococcal or staphylococcal infection. It occurs more commonly in adolescents and young adults than in children.

Acute retropharyngeal abscess results from infection in the retropharyngeal lymph glands, which may follow an upper respiratory tract

bacterial infection. Most common pathogens are beta-hemolytic *Streptococcus* and *S. aureus*. These lymph glands begin to atrophy after age 2. Acute retropharyngeal abscess most commonly affects infants and children younger than age 2.

Chronic retropharyngeal abscess may result from tuberculosis of the cervical spine (Pott disease) and may occur at any age.

Pathophysiology

Peritonsillar abscess, the most common deep infection of the head and neck that occurs in adults, is typically formed by a combination of aerobic and anaerobic bacteria. The presenting symptoms include fever, throat pain, and trismus. Ultrasonography and computed tomographic scanning are useful in confirming a diagnosis. Needle aspiration remains the gold standard for diagnosis and treatment of peritonsillar abscess. After performing aspiration, appropriate antibiotic therapy (including penicillin, clindamycin, cephalosporins, or metronidazole) must be initiated. In advanced cases, incision and drainage or immediate tonsillectomy may be required.

Complications

- ◆ Airway obstruction
- ◆ Cellulitis
- ◆ Endocarditis
- ◆ Pericarditis
- ◆ Pleural effusion
- ◆ Pneumonia

Signs and Symptoms

Key symptoms of peritonsillar abscess include severe throat pain, occasional ear pain on the same side as the abscess, and tenderness of the submandibular gland. Dysphagia causes drooling. Trismus may occur as a result of the spread of edema and infection from the peritonsillar space to the pterygoid muscles. Other effects include fever, chills, malaise, rancid breath, nausea, muffled speech, dehydration, cervical adenopathy, and localized or systemic sepsis.

Clinical features of retropharyngeal abscess include pain, dysphagia, fever, and, when the abscess is located in the upper pharynx, nasal obstruction; with a low-positioned abscess, dyspnea, progressive inspiratory

stridor (from laryngeal obstruction), neck hyperextension, and, in children, drooling and muffled crying occur. Other symptoms in children may include gurgling respirations, dyspnea and dysphagia, respiratory symptoms, and fever. A very large abscess may press on the larynx, causing edema, or may erode into major vessels, causing sudden death from asphyxia or aspiration.

Diagnosis

Diagnosis of peritonsillar abscess usually begins with a patient history of bacterial pharyngitis. Examination of the throat shows swelling of the soft palate on the abscessed side, with displacement of the uvula to the opposite side; red, edematous mucous membranes; and tonsil displacement toward the midline. Culture may reveal streptococcal or staphylococcal infection.

Diagnosis of retropharyngeal abscess is based on patient history of nasopharyngitis or pharyngitis and on physical examination revealing a soft, red bulging of the posterior pharyngeal wall. X-rays show the larynx pushed forward and a widened space between the posterior pharyngeal wall and vertebrae. If neck pain or stiffness occurs, look for extension to the epidural space or the cervical vertebrae. Culture and sensitivity tests isolate the causative organism and reveal the appropriate antibiotic.

Treatment

For early-stage peritonsillar abscess, large doses of penicillin or another broad-spectrum antibiotic is necessary. If the patient is immunocompromised or has been repeatedly hospitalized, antibiotic therapy should include coverage for staphylococci and gram-negative organisms. For late-stage abscess, with cellulitis of the tonsillar space, primary treatment is usually incision and drainage under a local anesthetic, followed by antibiotic therapy for 7 to 10 days. Tonsillectomy, scheduled no sooner than 1 month after healing, prevents recurrence but is recommended only after several episodes.

In acute retropharyngeal abscess, the primary treatment is incision and drainage through the pharyngeal wall. It's considered a surgical emergency. In chronic retropharyngeal abscess, drainage is performed through an external incision behind the sternomastoid muscle. During incision and drainage, strong, continuous mouth suction is necessary to prevent aspiration of pus, and the head should be kept down. Postoperative drug

therapy includes I.V. antibiotics (usually penicillin or clindamycin) and analgesics.

Special Considerations



ALERT *Be alert for signs of respiratory obstruction (inspiratory stridor, dyspnea, retractions and nasal flaring, increasing restlessness, and cyanosis). Keep emergency airway equipment nearby.*

- ◆ Explain the drainage procedure to the patient and their parents. Because the procedure is usually done under local anesthesia, the patient may be apprehensive.
- ◆ Assist with incision and drainage. To allow easy expectoration and suction of pus and blood, place the patient in a semirecumbent or sitting position.

After incision and drainage:

- ◆ Give antibiotics, analgesics, and antipyretics, as ordered. Stress the importance of completing the full course of prescribed antibiotic therapy.
- ◆ Monitor vital signs, and report significant changes or bleeding. Assess pain, and treat accordingly.
- ◆ If the patient is unable to swallow, ensure adequate hydration with I.V. therapy. Monitor fluid intake and output, and watch for dehydration.
- ◆ Provide meticulous mouth care. Apply petroleum jelly to the patient's lips. Promote healing with warm saline gargles or throat irrigations for 24 to 36 hours after incision and drainage. Encourage adequate rest.



PREVENTION *Encourage early treatment of tonsillitis.*

VOCAL CORD PARALYSIS

Vocal cord paralysis results from disease of, or injury to, the superior or, most commonly, the recurrent laryngeal nerve. It may also be congenital.

Causes and Incidence

Vocal cord paralysis commonly results from the accidental severing of the recurrent laryngeal nerve, or of one of its extralaryngeal branches, during thyroidectomy. Other causes include pressure from a thoracic aortic

aneurysm or from an enlarged atrium (in patients with mitral stenosis), bronchial or esophageal carcinoma, hypertrophy of the thyroid gland, trauma (such as neck injuries) and intubation, and neuritis due to infections or metallic poisoning. Vocal cord paralysis can also result from hysteria and, rarely, lesions of the central nervous system.

Pathophysiology

Unilateral vocal fold paralysis occurs from a dysfunction of the recurrent laryngeal or vagus nerve innervating the larynx. It causes a characteristic breathy voice often accompanied by swallowing disability, a weak cough, and the sensation of shortness of breath. This is a common cause of neurogenic hoarseness.

Complications

- ◆ Airway obstruction
- ◆ Respiratory failure

Signs and Symptoms

Unilateral paralysis, the most common form, may cause vocal weakness and hoarseness. Bilateral paralysis typically produces vocal weakness and incapacitating airway obstruction if the cords become paralyzed in the adducted position.



PEDIATRIC TIP Children may present with hoarseness, aspiration, and stridor. If the paralysis is unilateral, it typically involves the left recurrent laryngeal nerve. In unilateral paralysis, airway intervention involving intubation and tracheostomy is rarely indicated; it's usually required if the paralysis is bilateral.

Diagnosis

The patient history and characteristic features suggest vocal cord paralysis.



CONFIRMING DIAGNOSIS Visualization by indirect laryngoscopy shows one or both cords fixed in an adducted or partially abducted position and confirms the diagnosis.

X-ray or CT scan detect abnormalities in the mediastinum that may be responsible for the injury.

Treatment

Treatment for unilateral vocal cord paralysis consists of injection of Teflon into the paralyzed cord, under direct laryngoscopy. This procedure enlarges the cord and brings it closer to the other cord, which usually strengthens the voice and protects the airway from aspiration. Thyroplasty also serves to reposition the vocal cord, but in this procedure an implant is placed through a neck incision. The ansa cervicalis nerve transfer allows for reinnervation of the muscles of the vocal cord. Bilateral cord paralysis in an adducted position necessitates a tracheostomy.

Alternative treatments for adults include endoscopic arytenoidectomy to open the glottis, and lateral fixation of the arytenoid cartilage through an external neck incision. Excision or fixation of the arytenoid cartilage improves airway patency but produces residual voice impairment.

Treatment for hysterical aphonia may include psychotherapy and hypnosis.

Special Considerations

If the patient chooses direct laryngoscopy and Teflon injection, explain these procedures thoroughly. Tell the patient these measures will improve their voice but won't restore it to normal. Patients are sometimes placed on voice rest for 24 to 48 hours to reduce stress on the vocal cords, which would increase the edema and might lead to airway obstruction.

Many patients with bilateral cord paralysis prefer to keep a tracheostomy instead of having an arytenoidectomy; voice quality is generally better with a tracheostomy alone than after corrective surgery.

If the patient is scheduled to undergo a tracheostomy:

- ◆ Explain the procedure thoroughly, and offer reassurance. Because the procedure is performed under a local anesthetic, the patient may be apprehensive.
- ◆ Teach the patient how to suction, clean, and change the tracheostomy tube.
- ◆ Reassure the patient that they can still speak by covering the lumen of the tracheostomy tube with a finger or a tracheostomy plug.

If the patient elects to have an arytenoidectomy, explain the procedure thoroughly. Advise the patient that the tracheostomy will remain in place until the edema has subsided and the airway is patent.

VOCAL CORD NODULES AND POLYPS

Vocal cord nodules result from hypertrophy of fibrous tissue and form at the point where the cords come together forcibly. Vocal cord polyps are chronic, subepithelial, edematous masses. Both nodules and polyps have a good prognosis unless continued voice abuse causes recurrence, with subsequent scarring and permanent hoarseness.

Causes and Incidence

Vocal cord nodules and polyps usually result from voice abuse, especially in the presence of infection. Consequently, they're most common in teachers, singers, and sports fans, and in energetic children (ages 8 to 12) who continually shout while playing. Polyps are common in adults who smoke, live in dry climates, or have allergies.



PEDIATRIC TIP *In children, papillomas of the larynx (benign warty growths) are the most common laryngeal neoplasm. Suspected causes include human papillomavirus types 6, 11, and 16. The virus may be acquired during birth because many mothers have a history of condylomata acuminata at the time of delivery.*

Complication

- ◆ Permanent hoarseness

Signs and Symptoms

Nodules and polyps inhibit the approximation of vocal cords and produce painless hoarseness. The voice may also develop a breathy or husky quality.

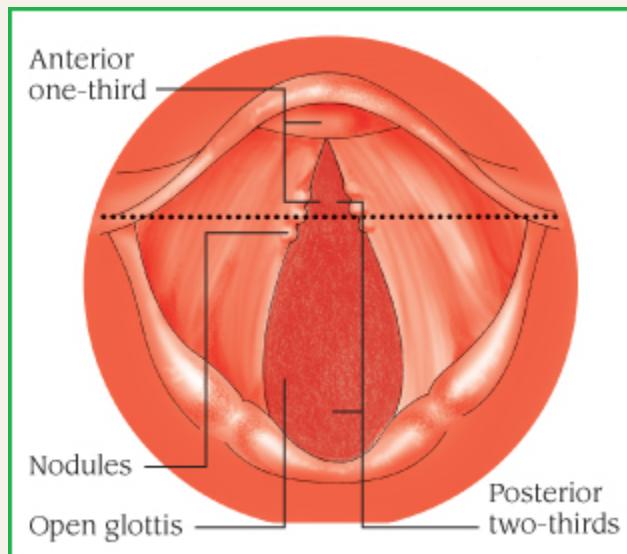
Diagnosis

Persistent hoarseness suggests vocal cord nodules and polyps; visualization by indirect laryngoscopy confirms it. In the patient with vocal cord nodules, laryngoscopy initially shows small red nodes and, later, white solid nodes

on one or both cords. (See *Vocal cord nodules*.) In the patient with polyps, laryngoscopy reveals unilateral or, occasionally, bilateral, sessile or pedunculated polyps of varying size, anywhere on the vocal cords.

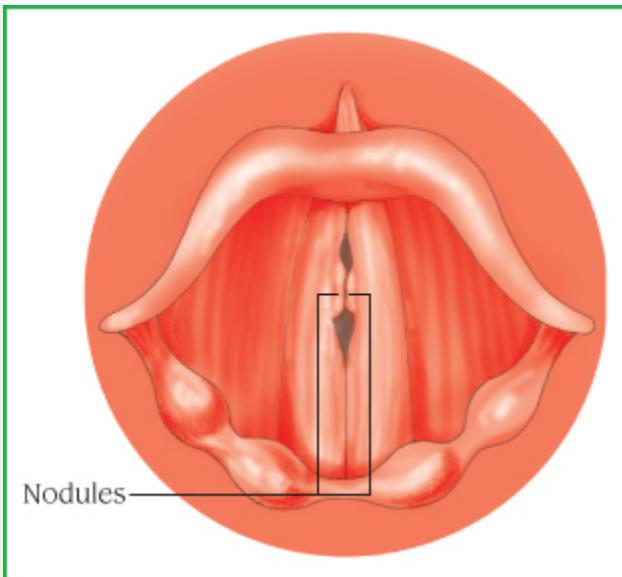
Vocal Cord Nodules

The most common site of vocal cord nodules is the point of maximal vibration and impact (junction of the anterior one third and the posterior two thirds of the vocal cord).



Vocal Cords Open

Vocal cord nodules affect the voice by inhibiting proper closure of the vocal cords during phonation.



Vocal Cords Closed

Treatment

Conservative management of small vocal cord nodules and polyps includes humidification, speech therapy (voice rest, training to reduce the intensity and duration of voice production), and treatment for any underlying allergies.

When conservative treatment fails to relieve hoarseness, nodules or polyps require removal under direct laryngoscopy. Microlaryngoscopy may be done for small lesions, to avoid injuring the vocal cord surface. If nodules or polyps are bilateral, excision may be performed in two stages: one cord is allowed to heal before excision of polyps on the other cord. Two-stage excision prevents laryngeal web, which occurs when epithelial tissue is removed from adjacent cord surfaces, and these surfaces grow together.



PEDIATRIC TIP *For children, treatment consists of speech therapy. If possible, surgery should be delayed until the child is old enough to benefit from voice training, or until the patient can understand the need to abstain from voice abuse.*

Special Considerations

- ◆ Postoperatively, stress the importance of resting the voice for 10 to 14 days while the vocal cords heal. Provide an alternative means of communication—Magic Slate, pad and pencil, or alphabet board. Place a sign over the bed to remind visitors that the patient shouldn't talk. Mark the intercom so other facility personnel are aware that the patient can't answer. Minimize the need to speak by trying to anticipate the patient's needs.
- ◆ If the patient is a smoker, encourage them to stop smoking entirely or, at the very least, to refrain from smoking during recovery from surgery.
- ◆ Use a vaporizer to increase humidity and decrease throat irritation.
- ◆ Make sure the patient receives speech therapy after healing if necessary, because continued voice abuse causes recurrence of growths.

LARYNGITIS

A common disorder, laryngitis is an acute or chronic inflammation of the vocal cords. Acute laryngitis may occur as an isolated infection or as part of a generalized bacterial or viral URTI. Repeated attacks of acute laryngitis produce inflammatory changes associated with chronic laryngitis.



ALERT *Several forms of laryngitis occur in children and can lead to significant or fatal respiratory obstruction, such as croup and epiglottitis.*

Causes and Incidence

Acute laryngitis usually results from infection (primarily viral) or excessive use of the voice, an occupational hazard in certain vocations (e.g., teaching, public speaking, or singing). It may also result from leisure activities (such as cheering at a sports event), inhalation of smoke or fumes, or aspiration of caustic chemicals. Chronic laryngitis may be caused by chronic upper respiratory tract disorders (sinusitis, bronchitis, nasal polyps, or allergy), mouth breathing, smoking, constant exposure to dust or other irritants, and alcohol abuse.

Pathophysiology

Acute laryngitis is an inflammation of the vocal fold mucosa and larynx that lasts less than 3 weeks. When the etiology of acute laryngitis is infectious, white blood cells remove microorganisms during the healing

process. The vocal folds then become more edematous, and vibration is adversely affected.

Complications

- ◆ Permanent hoarseness
- ◆ Airway obstruction in severe laryngitis

Signs and Symptoms

Acute laryngitis typically begins with hoarseness, ranging from mild to complete loss of voice. Associated clinical features include pain (especially when swallowing or speaking), a persistent dry cough, fever, laryngeal edema, and malaise. In chronic laryngitis, persistent hoarseness is usually the only symptom.

Diagnosis

Dx CONFIRMING DIAGNOSIS *Indirect laryngoscopy confirms the diagnosis by revealing red, inflamed, and, occasionally, hemorrhagic vocal cords, with rounded rather than sharp edges and exudate. Bilateral swelling may be present.*

In severe cases or if toxicity is a concern, a culture of the exudate is obtained. Consider 24-hour pH probe testing in chronic laryngitis and GERD. Also consider biopsy in chronic laryngitis in an adult with a history of smoking or alcohol abuse.

Treatment

Primary treatment consists of resting the voice. For viral infection, symptomatic care includes analgesics and throat lozenges for pain relief. Bacterial infection requires antibiotic therapy. Severe, acute laryngitis may necessitate hospitalization. When laryngeal edema results in airway obstruction, a tracheostomy may be necessary. In chronic laryngitis, effective treatment must eliminate the underlying cause. Antacids or histamine-2 blockers may be used if GERD is the cause. Steam inhalation may also prove beneficial as are smoking cessation, reducing alcohol intake, and job change or modification if warranted.

Special Considerations

- ◆ Explain to the patient why they shouldn't talk, and place a sign over the bed to remind others of this restriction. Provide a Magic Slate or a pad and pencil for communication. Mark the intercom panel so other facility personnel are aware that the patient can't answer. Minimize the need to talk by trying to anticipate the patient's needs.
- ◆ For the patient with a bacterial infection, stress the importance of completing the full course of antibiotic therapy.
- ◆ Suggest that the patient maintain adequate humidification by using a vaporizer or humidifier during the winter, by avoiding air-conditioning during the summer (because it dehumidifies), by using medicated throat lozenges, and by not smoking.
- ◆ Obtain a detailed patient history to help determine the cause of chronic laryngitis. Encourage the patient to modify predisposing habits, especially to stop smoking.
- ◆ Provide the patient with assistance for smoking cessation as well as for modification of other predisposing habits or occupational hazards.

JUVENILE ANGIOFIBROMA

An uncommon disorder, juvenile angiofibroma is a highly vascular, nasopharyngeal tumor made up of masses of fibrous tissue that contain many thin-walled blood vessels. The prognosis is good with treatment.

Causes and Incidence

A type of hemangioma, this tumor grows on one side of the posterior nares and may completely fill the nasopharynx, nose, paranasal sinuses, and, possibly, the orbit. More commonly sessile than polypoid, juvenile angiofibroma is nonencapsulated; it invades surrounding tissue.

Juvenile angiofibroma is typically found in adolescent males and is extremely rare in females. It's associated with nasal obstruction and epistaxis.

Pathophysiology

JNA is a rare benign tumor arising predominantly in the nasopharynx of adolescent males. It is an aggressive neoplasm and shows a propensity for destructive local spread often extending to the base of the skull and into the

cranium. Clinically, however, it is obscure with painless, progressive unilateral nasal obstruction being the common presenting symptom with or without epistaxis and rhinorrhea. Diagnosis of JNA is made by complete history, clinical examination, radiography, nasal endoscopy and by using specialized imaging techniques such as arteriography, CT, and magnetic resonance imaging. Early diagnosis, accurate staging, and adequate treatment are essential in the management of this lesion.

Complication

- ◆ Secondary anemia

Signs and Symptoms

Juvenile angiofibroma produces unilateral or bilateral nasal obstruction and severe recurrent epistaxis, usually between ages 7 and 21. Recurrent epistaxis eventually causes secondary anemia. Associated effects include purulent rhinorrhea, facial deformity, and nasal speech. Serous otitis media and hearing loss may result from eustachian tube obstruction.

Diagnosis

A nasopharyngeal mirror or nasal speculum permits visualization of the tumor. X-rays show a bowing of the posterior wall of the maxillary sinus. Three-plane magnetic resonance imaging and CT scans determine the extent of the tumors, which are seldom limited to the nasopharynx. Angiography determines the size and location of the tumor and shows the source of vascularization.



ALERT Tumor biopsy is contraindicated because of the risk of hemorrhage.

Treatment

Surgical procedures range from avulsion to cryosurgical techniques. Surgical excision is preferred after embolization with Teflon or an absorbable gelatin sponge to decrease vascularization. Whichever surgical method is used, this tumor must be removed in its entirety and not in pieces.

Preoperative hormonal therapy may decrease the tumor's size and vascularity. Blood transfusions may be necessary during avulsion. Radiation therapy produces only a temporary regression in an angiofibroma

but is the treatment of choice if the tumor has expanded into the cranium or orbit. Because the tumor is multilobular and locally invasive, it recurs in about 30% of patients during the first year after treatment, but rarely after 2 years.

Special Considerations

- ◆ Explain all diagnostic and surgical procedures. Provide emotional support; severe epistaxis frightens many people to the point of panic. Monitor hemoglobin levels and HCT for anemia.
- ◆ After surgery, immediately report excessive bleeding. Make sure an adequate supply of typed and crossmatched blood is available for transfusion.
- ◆ Monitor for any change in vital signs. Provide good oral hygiene, and use a bedside vaporizer to raise humidity.
- ◆ During blood transfusion, watch for transfusion reactions, such as fever, pruritus, chills, or a rash. If any of these reactions occur, discontinue the blood transfusion and notify the physician immediately.
- ◆ Teach the patient's family how to apply pressure over the affected area, and instruct them to seek immediate medical attention if bleeding occurs after discharge. Stress the importance of providing adequate humidification at home to keep the nasal mucosa moist.

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2

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

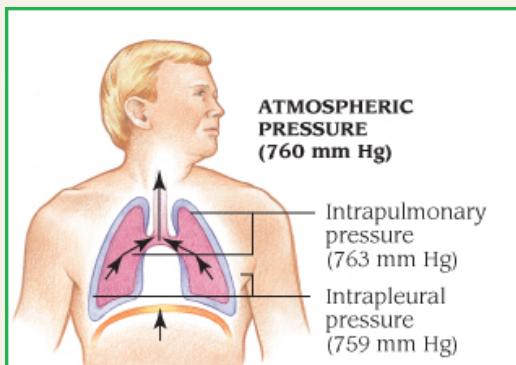
because of the vacuum created within the pleural space. As the thorax expands, negative pressure is created in the intrapleural space, causing the lungs to also expand and draw in the warmed, humidified air. The accessory muscles of inspiration, which include the scalene and sternocleidomastoid muscles, 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 *Mechanics of ventilation*, page 87.)

Mechanics of Ventilation

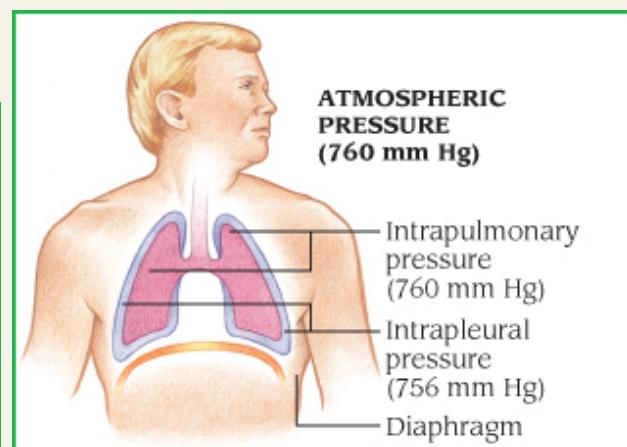
Breathing results from differences between atmospheric and intrapulmonary pressures, as described below.

Before inspiration, atmospheric pressure equals atmospheric pressure (~ 760 mm Hg). Intrapleural pressure is 756 mm Hg.



During inspiration, the diaphragm and external intercostal muscles contract,

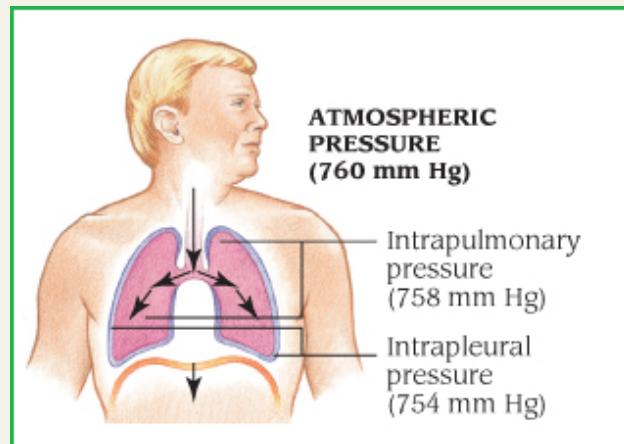
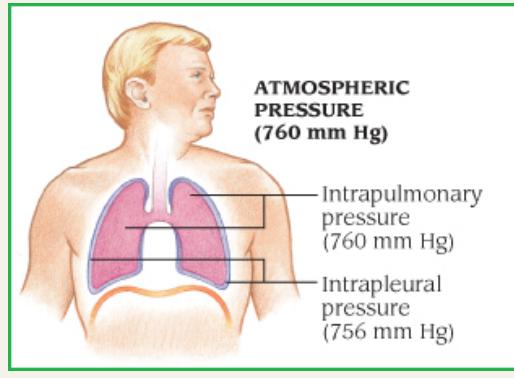
The intrapulmonary atmospheric pressure gradient pulls air into the lungs until the two pressures are equal.



During normal expiration, the diaphragm slowly relaxes and the lungs and thorax passively return to resting

enlarging the thorax vertically and horizontally. As the thorax expands, intrapleural pressure decreases and the lungs expand to fill the enlarging thoracic cavity.

size and position. During deep or forced expiration, contraction of internal intercostal and abdominal muscles reduces thoracic volume. Lung and thorax compression raises intrapulmonary pressure above atmospheric pressure.



An adult lung contains an estimated 300 million alveoli; each alveolus is supplied by many capillaries. To reach the capillary lumen, O₂ 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 O₂ tension of air entering the respiratory tract is approximately 150 mm Hg. In the alveoli, inspired air mixes with CO₂ and water vapor, lowering the O₂ pressure to approximately 100 mm Hg. Because alveolar partial pressure of O₂ is higher than that present in mixed venous blood entering the pulmonary capillaries (~40 mm Hg), O₂ diffuses across the alveolocapillary membrane into the blood.

O₂ AND CO₂ TRANSPORT AND INTERNAL RESPIRATION

Circulating blood delivers O₂ to the cells of the body for metabolism and transports metabolic wastes and CO₂ from the tissues back to the lungs. When oxygenated arterial blood reaches tissue capillaries, O₂ diffuses from the blood into the cells because of the O₂ tension gradient. The amount of O₂

available is determined by the concentration of hemoglobin (Hb; the principal carrier of O₂), the percentage of O₂ saturation of the Hb, regional blood flow, arterial O₂ content, and cardiac output.

Internal (cellular) respiration occurs as a part of cellular metabolism, which can take place with O₂ (aerobic) or without O₂ (anaerobic). The most efficient method for providing fuel (high-energy compounds such as adenosine triphosphate [ATP]) for cellular reactions is aerobic metabolism, which produces CO₂ 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 CO₂ as a metabolic by-product.

Because circulation is continuous, CO₂ doesn't normally accumulate in tissues. CO₂ produced during cellular respiration diffuses from tissues into regional capillaries and is transported by systemic venous circulation. When CO₂ reaches the alveolar capillaries, it diffuses into the alveoli, where the partial pressure of CO₂ is lower; CO₂ is removed from the alveoli during exhalation.

MECHANISMS OF CONTROL

The central nervous system's (CNS) control of respiration lies in the respiratory center, located in the lateral medulla oblongata of the brainstem. Impulses travel down the phrenic nerves to the diaphragm, and down 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 pneumotoxic centers in the pons influence the pattern of breathing. Stimulation of the lower pontine apneustic center (e.g., by trauma, tumor, or stroke) produces forceful inspiratory gasps alternating with weak expiration. The apneustic center continually excites the medullary inspiratory center and thus facilitates inspiration. Signals from the pneumotoxic 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 the vagus nerves are intact.

Partial pressure of arterial oxygen (Pao₂), pH, and pH of cerebrospinal fluid (CSF) influence output from the respiratory center. When CO₂ 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 overexpansion 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 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 PFTs) measure lung volume, flow rates, and compliance. Normal values, individualized by body stature, ethnicity, 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 (FEV₁), maximum volume of air that can be expired in 1 second from TLC; maximal voluntary ventilation, volume of air that can be expired in 1 minute with the patient's maximum voluntary effort; and forced vital capacity (FVC), 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.)

- ◆ Methacholine challenge is one method of assessing airway responsiveness and is used to determine a diagnosis of asthma.
- ◆ Exercise stress test evaluates the ability to transport O₂ and remove CO₂ 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 PaO₂ may indicate hypoventilation, ventilation–perfusion mismatch, or shunting of blood away from gas exchange sites. Increased partial pressure of arterial carbon dioxide (Paco₂) reflects marked ventilation–perfusion mismatch or hypoventilation; decreased Paco₂ 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 Paco₂ 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 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 or her respiratory problem. How long has he or she 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 or she smoked or smokes, and how long he or she 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 or she walk? How many flights of stairs can he or she climb? Has his or her exercise tolerance been decreasing? Can he or she 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 or she is frail or cachectic, he or she

may have a chronic disease that has impaired his or her appetite. If he or she is diaphoretic, restless, or irritable or protective of a painful body part, he or she 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 the patient 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 or her arms on the sides of a chair to increase expansion and lean 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.

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.*)

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	Hollow to loud	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.

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 re-expand 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:

- ◆ Monitor changes in suction pressure.
- ◆ 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.
- ◆ 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.

Additionally, if a water seal–wet suction system is in place:

- ◆ Check for fluctuation in the water-seal chamber as the patient breathes. Normal fluctuations of 2" to 4" (about 5 to 10 cm) reflect pressure changes in the pleural space during respiration.

- ◆ Watch for intermittent bubbling in the water-seal chamber. This bubbling occurs normally when the system is removing air from the pleural cavity. Absence of bubbling indicates that the pleural space has sealed.
- ◆ Check the water level in the suction-control chamber. If necessary, add sterile water to bring the level to the ordered level.
- ◆ Check for gentle bubbling in the suction-control chamber, which indicates that the proper suction level has been reached.

If a dry-suction system is in place, check that the rotary dry-suction control dial is turned to the ordered suction mark and verify that the appropriate indicator is present, indicating that the desired amount of suction is applied.

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. Positive-pressure ventilators cause inspiration while increasing tidal volume (V_T). The inspiratory cycles of these ventilators may vary in volume, pressure, time, or frequency. For example, a volume-cycled ventilator—the type most commonly used—delivers a preset volume of air each time, regardless of the amount of lung resistance. A pressure-cycled ventilator generates flow until the machine reaches a preset pressure regardless of the volume delivered or the time required to achieve the pressure. A time-cycled ventilator generates flow for a preset amount of time. A high-frequency ventilator uses high respiratory rates and low V_T to maintain alveolar ventilation. 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.

Implement strategies to prevent ventilator-associated pneumonia (VAP) and plan to remove the patient from ventilator support as soon as the cause of respiratory failure has resolved. (See *Preventing ventilator-associated pneumonia*, page 92.) Several weaning methods are used. 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.



PREVENTION PREVENTING PNEUMONIA

VENTILATOR-ASSOCIATED

Ventilator-associated pneumonia (VAP) is the leading cause of death among all hospital-acquired infections. VAP also prolongs time spent on the ventilator, length of critical care unit (CCU) stay, and length of hospital stay after discharge from the CCU. Research has shown that the mortality rate due to VAP can be reduced by early recognition of pneumonia and consistent application of evidence-based practices. The Ventilator Bundle is a group of interventions related to ventilator care that, when implemented together, achieve significantly better outcomes than when implemented individually. The key components of the Ventilator Bundle include:

- ◆ elevating the head of the bed 30 to 45 degrees
- ◆ interrupting sedation daily and assessing the readiness to extubate
- ◆ instituting peptic ulcer disease prophylaxis
- ◆ instituting deep vein thrombosis prophylaxis
- ◆ providing daily oral care with chlorhexidine

Various other best practices can be combined with the bundle to prevent VAP. They include:

- ◆ adhering to Centers for Disease Control and Prevention or World Health Organization hand hygiene guidelines to prevent the spread of infection
- ◆ using noninvasive ventilatory support, such as bilevel positive-airway ventilation instead of endotracheal (ET) intubation and mechanical ventilation, to eliminate the risk of VAP
- ◆ using the oral route instead of the nasal route for ET intubation to prevent sinusitis
- ◆ maintaining ET tube cuff pressure at 20 cm or more to prevent aspiration
- ◆ using a cuffed ET tube with in-line and subglottic suctioning to prevent secretion aspiration
- ◆ avoiding gastric distention to reduce the risk for aspiration

- ◆ avoiding unexplained extubation and reintubation to prevent secretion aspiration
 - ◆ minimizing equipment contamination (by removing condensate from ventilator circuits, keeping the circuit closed during removal, changing the ventilator circuit only when visibly soiled or malfunctioning, and disinfecting and storing respiratory equipment properly) to prevent airway contamination
 - ◆ teaching the patient and family about measures to prevent VAP and involving them in monitoring
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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 or her own. 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 the 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 $5\frac{1}{2}$ lb (2.5 kg). Aggressive management using mechanical ventilation can improve the prognosis, but some surviving neonates may develop some degree of bronchopulmonary dysplasia (BPD).

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 because of a lack of surfactant, a lipoprotein present in alveoli and respiratory bronchioles. The 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 g. 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.

Pathophysiology

The lack of surfactant coating the alveoli reduces the available pulmonary surface and decreases the area for gas exchange. Worsening hypercapnia and hypoxia cause metabolic and respiratory acidosis leading to pulmonary vasoconstriction and peripheral vasodilation. Damage to the endothelial and alveolar cells results from the ongoing hypoxia. The subsequent vascular disruption leads to plasma leakage into the alveolar spaces, layering of fibrin and necrotic cells creating hyaline membranes. These membranes impede the exchange of gases across the alveolar surface.

Complications

- ◆ Pneumothorax
- ◆ Pneumomediastinum
- ◆ Pneumopericardium
- ◆ 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, they usually develop 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 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

Dx 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 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 Pao_2 ; normal, decreased, or increased Paco_2 ; 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, ET intubation. Special ventilation techniques are now used on the patient's

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 ABGs 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 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, on the basis of findings.
- ◆ Mechanical ventilation in neonates is usually done in a pressure-limited mode rather than in 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 ROP.
- ◆ Teach the parents about their neonate's condition and, if possible, let them participate in their 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 or her 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 their back to sleep.

Causes and Incidence

SIDS is the third leading cause of death in infants between 1 month and 1 year old. 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 CO₂ 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.

Pathophysiology

Although the exact pathophysiology of SIDS is not known, there is a common theory. Abnormalities of the autoimmune nervous system and brainstem cause dysfunctions of breathing. Episodes of hypoxia contribute to delaying the arousal response when oxygen availability is decreased and potentially leading to death.

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, they 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 epiglottitis. 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, allergens, 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 3 months and 5 years old) that usually occurs during the winter. Up to 15% of patients have a strong family history of croup.

Pathophysiology

Infection of the laryngeal mucosa leads to edema and inflammation of the epiglottal area. This swelling leads to a narrowing of the airway and increasingly deep respirations. The ongoing effort to breath as the narrowing progresses becomes more difficult and the air flowing through the upper airway becomes turbulent. During inspiration, the flexible chest wall caves in slightly and causing paradoxical breathing.

Complications

- ◆ Respiratory distress
- ◆ Respiratory arrest
- ◆ Epiglottitis

- ◆ Bacterial tracheitis
- ◆ Atelectasis
- ◆ Dehydration

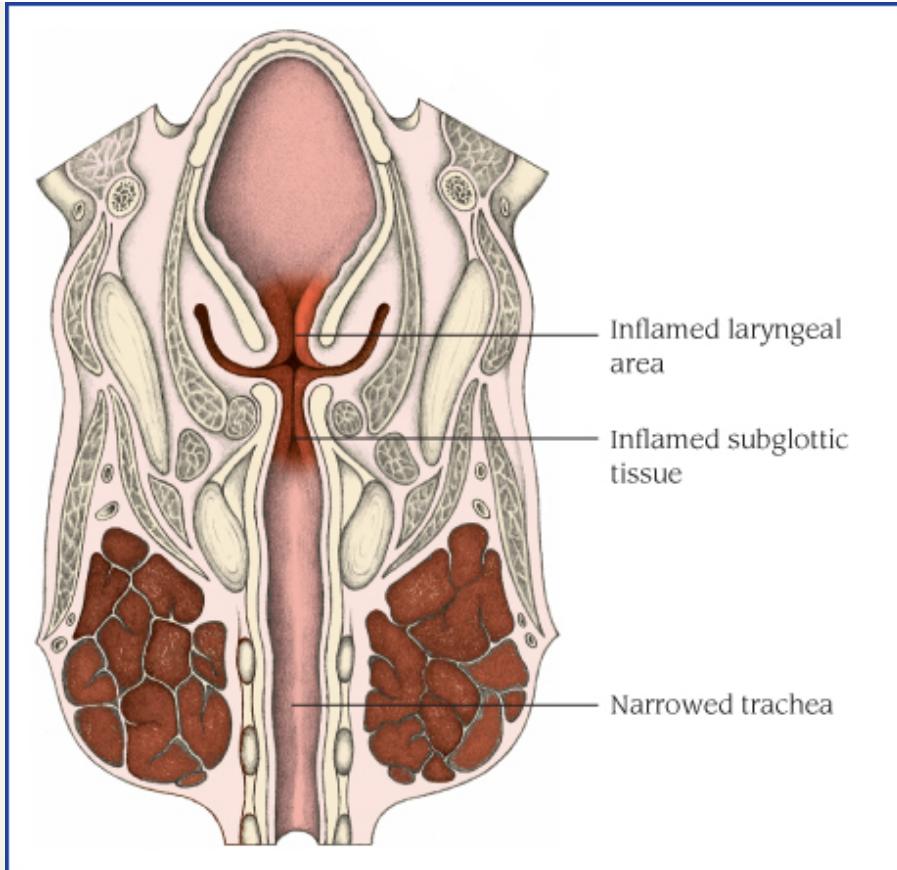
Signs and Symptoms

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*.)



PATHOPHYSIOLOGY 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.



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 1 and 3 years old, 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.

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 posterior–anterior 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 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 them in an infant seat or propped up with a pillow; place an older child in Fowler’s

position. If an older child requires a cool mist tent to help them 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 ice pops. 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 mist 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

- ◆ *Perform hand hygiene 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.*

EPIGLOTTITIS

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

Causes and Incidence

Epiglottitis usually results from infection with Hib and, occasionally, pneumococci and group A streptococci. It typically strikes children between 2 and 6 years old. (However, immunosuppression can predispose adults to epiglottitis.) Since the advent of the Hib vaccine, epiglottitis is becoming more rare.

Pathophysiology

The causative bacteria invade the mucosa and into the bloodstream causing bacteremia and infection of the epiglottis as well as surrounding tissues. Acute inflammation and edema begin in the epiglottic area and progressing to the epiglottic folds, arytenoids, and entire supraglottic larynx. The aggressive swelling and edema greatly reduces the available airway and quickly increasing the risk for a respiratory crisis.

Complications

- ◆ Respiratory failure
- ◆ Pneumonia
- ◆ Meningitis
- ◆ Death
- ◆ Pericarditis

Signs And Symptoms

Sometimes preceded by an upper respiratory infection, epiglottitis 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 epiglottitis may hyperextend his or her neck, sit up, and lean forward with his or her mouth open, tongue protruding, and nostrils flaring as he or she tries to breathe. The child may develop inspiratory retractions and rhonchi.

Diagnosis

In acute epiglottitis, 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 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 epiglottitis and airway obstruction requires emergency hospitalization; the child 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 epiglottitis 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 they won’t be able to cry or call out; provide emotional support. Reassure the patient and their 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 epiglottitis is contagious, and airborne or droplet precautions should be followed. Family members should be screened.



PREVENTION

- Perform hand hygiene frequently to prevent infections.
- Administer the Hib vaccine to children.

Acute Disorders

ACUTE RESPIRATORY DISTRESS SYNDROME

A form of noncardiogenic pulmonary edema that causes acute respiratory failure (ARF), 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 *Alveolar changes in ARDS*.)



PATHOPHYSIOLOGY ALVEOLAR CHANGES IN ARDS

The alveoli undergo major changes in each phase of ARDS.

Phase 1

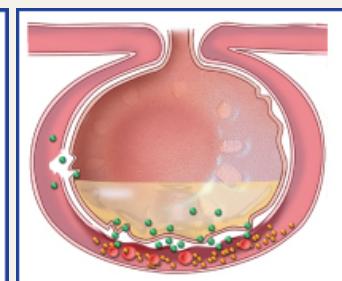
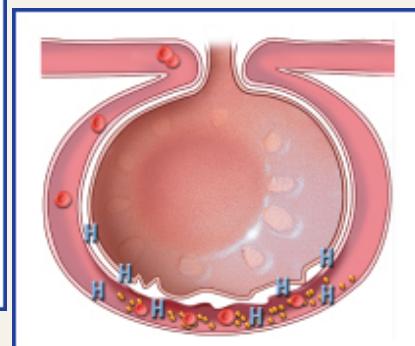
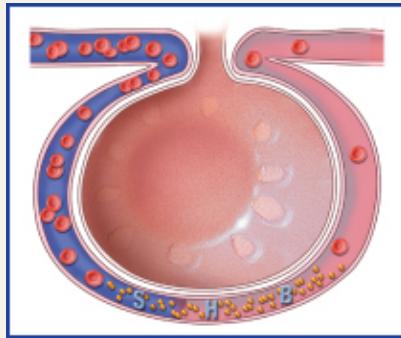
In phase 1, injury reduces normal blood flow to the lungs. Platelets aggregate and release histamine (H), serotonin (S), and bradykinin (B).

Phase 2

In phase 2, those substances—especially histamine—inflate and damage the alveolocapillary membrane, increasing capillary permeability. Fluids then shift into the interstitial

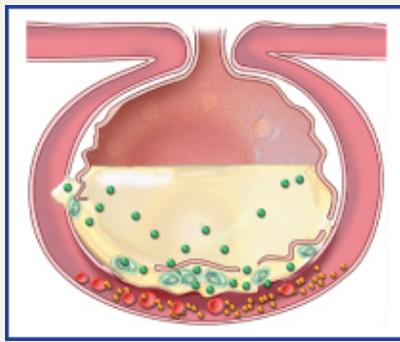
Phase 3

In phase 3, as capillary permeability increases, proteins and fluids leak out, increasing interstitial osmotic pressure and causing pulmonary edema.

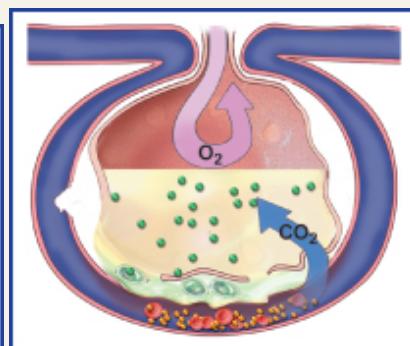


Phase 4

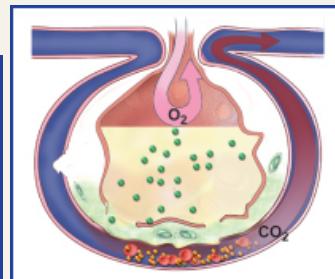
In phase 4, decreased blood flow and fluids in the alveoli damage surfactant and impair the cell's ability to produce more. As a result, alveoli collapse, impeding gas exchange and decreasing lung compliance.

**Phase 5**

In phase 5, sufficient oxygen can't cross the alveolocapillary membrane, but carbon dioxide (CO_2) can and is lost with every exhalation. Oxygen (O_2) and CO_2 levels decrease in the blood.

**Phase 6**

In phase 6, pulmonary edema worsens, inflammation leads to fibrosis, and gas exchange is further impeded.



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)
- ◆ blood transfusion
- ◆ smoke or chemical inhalation (nitrous oxide, chlorine, or ammonia)
- ◆ hydrocarbon and paraquat ingestion
- ◆ pancreatitis, uremia, or miliary tuberculosis (TB; 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.

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

Pathophysiology

An acute lung injury can begin the cascade of alveolar damage resulting in an altered permeability of the epithelial barrier and subsequent pulmonary edema. Diffuse alveolar damage progresses and granulation tissue forms in the alveolar spaces creating fibrosis. This fibrotic phase inhibits lung compliance and effective respiration.

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, ABG analysis initially shows decreased Pao₂ (less than 60 mm Hg) and Paco₂ (less than 35 mm Hg). The resulting pH usually reflects respiratory alkalosis. As ARDS becomes more severe, ABG analysis shows respiratory acidosis (increasing Paco₂ [more than 45 mm Hg]), metabolic acidosis (decreasing bicarbonate [less than 22 mEq/L]), and a decreasing Pao₂ 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 (PAWP); allows collection of pulmonary artery blood, which shows decreased oxygen saturation, reflecting tissue hypoxia; measures pulmonary artery pressure (PAP); 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 CPAP. Hypoxemia

that doesn't respond adequately to these measures requires ventilatory support with intubation, volume ventilation, and 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 CO₂ 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 family and the patient who's recovering from ARDS that recovery will take some time and that they 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).



PREVENTION Prevent VAP through use of the Ventilator Bundle and other best practices, such as continuous removal of subglottic secretions, change of ventilator circuit no more often than every 48 hours, and performance of hand hygiene before and after contact with each patient.

ACUTE RESPIRATORY FAILURE IN COPD

In patients with essentially normal lung tissue, ARF usually means Paco_2 above 50 mm Hg and Pao_2 below 50 mm Hg. These limits, however, don't apply to patients with COPD, who usually have a consistently high Paco_2 and low Pao_2 . In patients with COPD, only acute deterioration in 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:

- ◆ 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.

Pathophysiology

An acute and progressive exacerbation of COPD is triggered by the cessation of maintenance medications or some type of infection. Damage to the epithelium from ongoing exposure to noxious gases or particles impairs the mucociliary response causing the accumulation of mucus and bacteria and contributing to the obstruction of airways. The permeant enlargement of the airspaces by the terminal bronchioles leads to a decrease in the alveolar surface area for gas exchange and contributes to ineffective ventilation.

Complications

- ◆ Respiratory failure
- ◆ Pneumonia
- ◆ Hypoxemia
- ◆ Pneumothorax
- ◆ Heart failure

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 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 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; the patient 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, jugular vein distention, 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 O₂ therapy (using nasal prongs or Venturi mask) to raise the Pao₂. In patients with chronic hypercapnia, O₂ therapy can cause hypoventilation by increasing Paco₂ and decreasing the respiratory drive, necessitating mechanical ventilation. The minimum fraction of inspired air (FIO₂) required to maintain ventilation or O₂ saturation greater than 85% to 90% should be used. If significant uncompensated respiratory acidosis or unrefractory hypoxemia exists, mechanical ventilation (through an 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 O₂ at appropriate concentrations to maintain Pao₂ at a minimum of 50 to 60 mm Hg. Patients with COPD usually require only small amounts of supplemental O₂. 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 CO₂, encourage them to cough and to breathe deeply. Teach them to use pursed-lip and diaphragmatic breathing to control dyspnea. If the patient is alert, have them use an incentive spirometer; if they are intubated and lethargic, turn the patient 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 performing hand hygiene and 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 (NG) tube or if the patient 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.
- ◆ 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.



PREVENTION

- ◆ *To prevent VAP, implement Ventilator Bundle.*
- ◆ *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.*

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. (See *How pulmonary edema develops*, page 131.)

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 is 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 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

Pathophysiology

A hemodynamic disturbance or alteration in the permeability of the microvasculature allowing fluid to pass through into the interstitial space. This interstitial edema progresses when the capacity of the lymphatics is exceeded and unable to drain the fluid efficiently and subsequently decreasing lung compliance and shortness of breath.

Complications

- ◆ respiratory failure
- ◆ pleural effusion
- ◆ edema to lower extremities and abdomen
- ◆ death

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. ABG analysis usually shows hypoxia; the $Paco_2$ 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 PAWPs. This helps to rule out ARDS—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 Pao_2 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 end-diastolic 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 be anxious because of hypoxia and respiratory distress. Explain all procedures. Provide emotional support to the 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, 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)

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-aged to elderly men more often than women, but the 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.

Pathophysiology

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 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.

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 CNS 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

- ◆ PAP measurements show increased right ventricular and PAPs, 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 PAP 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.
- ◆ ABG analysis shows decreased 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.
- ◆ PFTs 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 their radial pulse before taking digoxin or any cardiac glycoside. They should be instructed to notify the physician if they detect 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 patient 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 healthcare 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 them 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 patient to return for periodic laboratory tests to monitor partial thromboplastin time (PTT), fibrinogen level, platelet count, HCT, Hb 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 air-conditioning 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-aged and elderly people
- ◆ immunocompromised patients (particularly those receiving corticosteroids, e.g., 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 COPD
- ◆ those with alcoholism
- ◆ cigarette smokers
- ◆ those on a ventilator for extended periods

Pathophysiology

When water droplets containing a sufficient amount of the *Legionella* bacterium enter the atmosphere they can be inhaled into the lungs. There they invade the epithelial cells of the lungs and begin to replicate intracellularly causing a Legionnaires' infection.

Complications

- ◆ Respiratory failure
- ◆ Septic shock
- ◆ Acute kidney failure

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, ARF, 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 Pao_2 and, initially, decreased Paco_2 . Bronchial washings and blood, pleural fluid, and sputum tests rule out other infections.



CONFIRMING DIAGNOSIS *Definitive tests include direct immunofluorescence 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. A 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 ABG 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 re-expansion of the collapsed lung.

Causes

Atelectasis commonly results from bronchial occlusion by mucus plugs. It's a problem in many patients with COPD, 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 RDS 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. CNS depression (as in drug overdose) eliminates periodic sighing and is a predisposing factor of progressive atelectasis.

Pathophysiology

Atelectasis results from some type of obstruction or compression of the lungs or bronchus. Retraction of the lung occurs when the blood circulating in the alveolar capillary bed absorbs the gas from the alveolar in the unventilated lung. The alveolar spaces fill with secretions and cells, preventing the complete collapse of the lung. The surrounding tissues distend and displace, shifting the heart and mediastinum toward the atelectatic area.

Complications

- ◆ Respiratory failure
- ◆ Pneumonia
- ◆ Hypoxemia

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. Implement the Ventilator Bundle to prevent VAP.
- ◆ Use an incentive spirometer to encourage deep inspiration through positive reinforcement. Teach the patient how to use the spirometer and encourage them 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 they are 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 them to appropriate support groups for help.
- ◆ Provide reassurance and emotional support; the patient may be anxious because of 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 (Paco_2 greater than 45 mm Hg), respiratory acidosis can be acute (because of 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.

- ◆ 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; COPD; asthma; severe acute respiratory distress syndrome (SARS); chronic bronchitis; large pneumothorax; extensive pneumonia; and pulmonary edema.

Hypoventilation compromises elimination of 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, the concentration of hydrogen ions in body fluids, which directly reflects acidity, increases.

Pathophysiology

Lung diseases or conditions that cause hypoventilation result in CO_2 being produced at a rapid rate. Lack of adequate ventilation quickly increases the partial pressure of arterial CO_2 . The increase in $Paco_2$ also results in the decrease in bicarbonate ration and decreasing the pH to an acidotic state.

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 exhibiting 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** 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, CT scan, and PFTs 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 correction of 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 CO₂ retention for signs of acidosis. Also, administer O₂ 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 Paco₂ 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 CO₂ by the lungs exceeds the production of CO₂ 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, CNS disease (inflammation or tumor), sepsis, hepatic failure, and pregnancy

Pathophysiology

An underlying condition or stimulus that causes hyperventilation, expels an increased amount of CO₂. CO₂ in the circulation is shifted causing hydrogen ions and bicarbonate to change into additional CO₂ via the enzyme carbonic anhydrase which, in turn, decreases the available hydrogen ions and increasing the pH.

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 (because of below-normal CO₂ levels that decrease cerebral blood flow), agitation, circumoral and peripheral paresthesias, carpopedal spasms, twitching (possibly progressing to tetany), and muscle weakness. Severe respiratory alkalosis may cause cardiac arrhythmias (that may fail to respond to conventional treatment), seizures, or both.

Diagnosis

 **CONFIRMING DIAGNOSIS** ABG analysis confirms respiratory alkalosis and rules out respiratory compensation for metabolic acidosis: Paco₂ less than 35 mm Hg; pH elevated in proportion to the fall in Paco₂ 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 PFTs 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 CO₂ 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. (See *Understanding tension pneumothorax*, page 112.)

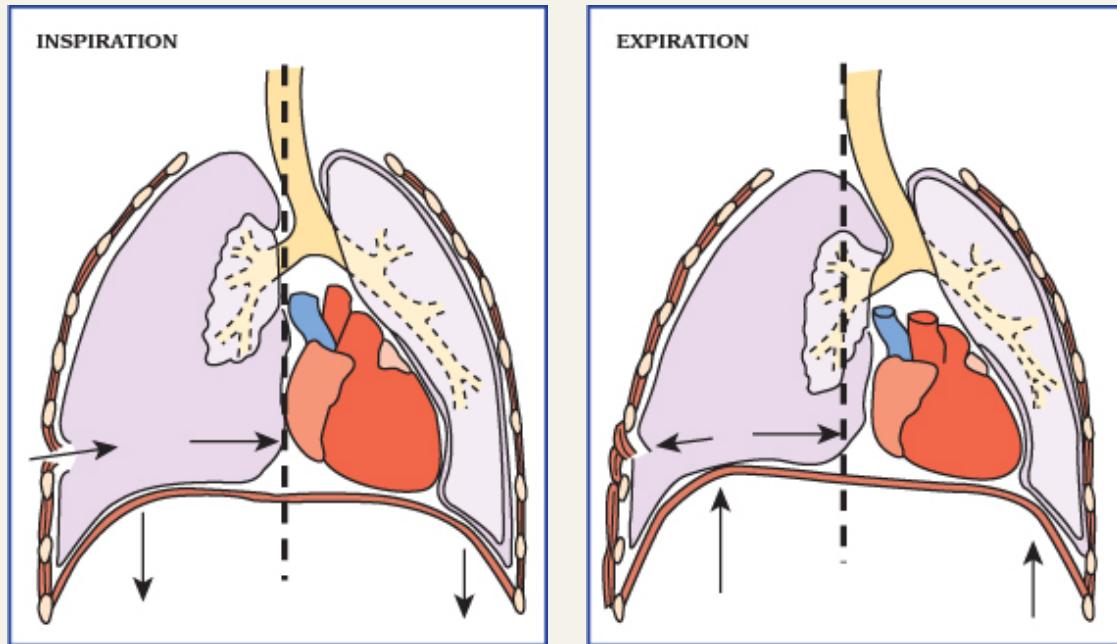


PATHOPHYSIOLOGY UNDERSTANDING TENSION PNEUMOTHORAX

In tension pneumothorax, air accumulates intrapleurally and can't escape. Intrapleural pressure rises, collapsing the ipsilateral lung.

On inspiration, the mediastinum shifts toward the unaffected lung, impairing ventilation.

On expiration, the mediastinal shift distorts the vena cava and reduces venous return.



Causes and Incidence

Spontaneous pneumothorax usually occurs in otherwise healthy adults 20 to 40 years old. 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 COPD, asthma, cystic fibrosis, TB, and whooping cough. Spontaneous pneumothorax may also occur in interstitial lung disease, such as eosinophilic granuloma or lymphangiomatosis.

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.

Pathophysiology

Pneumothorax can 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.

Complications

- ◆ 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 confirms 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, ABG findings include pH less than 7.35, Pao₂ less than 80 mm Hg, and Paco₂ 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 re-expand 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.

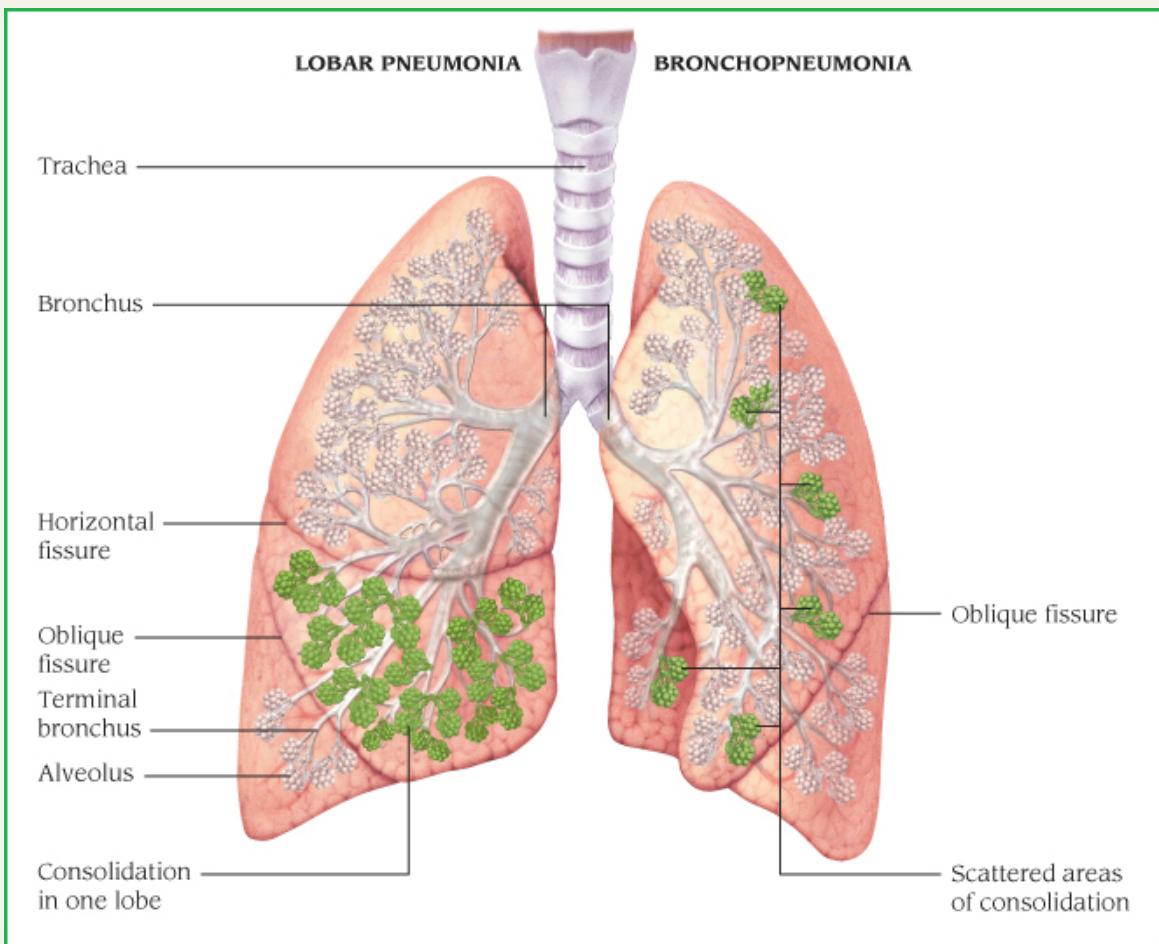
- ◆ Urge the patient to control coughing and gasping during thoracotomy. However, after the chest tube is in place, encourage them to cough and breathe deeply (at least once an hour) to facilitate lung expansion.
- ◆ If the patient is undergoing chest tube drainage, watch for continuing air leakage (bubbling), 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 them 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. (See *Looking at lobar pneumonia and bronchopneumonia.*)

Looking at Lobar Pneumonia and Bronchopneumonia

Pneumonia can involve the distal airways, alveoli, part of a lobe, or an entire lobe.



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 114 to 116.)

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	<ul style="list-style-type: none">◆ 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 occur if foreign body is present	<ul style="list-style-type: none">◆ Chest X-ray: locates areas of infiltrates, which suggest diagnosis	<ul style="list-style-type: none">◆ Antimicrobial therapy: penicillin G or clindamycin◆ Supportive: oxygen therapy, suctioning, coughing, deep breathing, and adequate hydration
Bacterial <i>Klebsiella</i>	<ul style="list-style-type: none">◆ 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	<ul style="list-style-type: none">◆ 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 <i>Klebsiella</i>	<ul style="list-style-type: none">◆ Antimicrobial therapy: an aminoglycoside and a cephalosporin
<i>Staphylococcus</i>	<ul style="list-style-type: none">◆ Temperature of 102° to 104° F (38.9° to 40° C), recurrent shaking chills, bloody sputum, dyspnea,	<ul style="list-style-type: none">◆ Chest X-ray: multiple abscesses and infiltrates; high incidence of empyema	<ul style="list-style-type: none">◆ Antimicrobial therapy: nafcillin or oxacillin for 14 days if staphylococci are

Type	Signs and symptoms	Diagnosis	Treatment
	<ul style="list-style-type: none"> ◆ tachypnea, and hypoxemia ◆ Should be suspected with viral illness, such as influenza or measles, and in patients with cystic fibrosis 	<ul style="list-style-type: none"> ◆ <i>WBC count</i>: elevated ◆ <i>Sputum culture and Gram stain</i>: may show gram-positive staphylococci 	<ul style="list-style-type: none"> ◆ <i>Supportive</i>: chest tube drainage of empyema
Streptococcus (<i>Streptococcus pneumoniae</i>)	<p>Sudden onset of severe, shaking chills and a sustained temperature of 102° to 104° F (38.9° to 40° C); commonly preceded by upper respiratory tract infection</p>	<ul style="list-style-type: none"> ◆ <i>Chest X-ray</i>: areas of consolidation, commonly lobar ◆ <i>WBC count</i>: elevated ◆ <i>Sputum culture</i>: may show gram-positive <i>S. pneumoniae</i>; this organism not always recovered 	<ul style="list-style-type: none"> ◆ <i>Antimicrobial therapy</i>: penicillin G (or erythromycin, if patient is allergic to penicillin) for 7 to 10 days beginning after obtaining 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 <i>Pneumocystis carinii</i> (jiroveci)	<ul style="list-style-type: none"> ◆ Occurs in immunocompromised persons ◆ Dyspnea and nonproductive cough ◆ Anorexia, weight loss, and fatigue ◆ Low-grade fever 	<ul style="list-style-type: none"> ◆ <i>Fiber-optic bronchoscopy</i>: obtains specimens for histologic studies ◆ <i>Chest X-ray</i>: nonspecific infiltrates, nodular lesions, or spontaneous pneumothorax 	<ul style="list-style-type: none"> ◆ <i>Antimicrobial therapy</i>: trimethoprim and sulfamethoxazole or pentamidine by I.V. administration or inhalation. (Prophylactic pentamidine may be used for high-risk patients.) ◆ <i>Supportive</i>: oxygen, improved nutrition, and mechanical ventilation
Viral Adenovirus (insidious onset;	<ul style="list-style-type: none"> ◆ Sore throat, fever, cough, chills, malaise, small 	<ul style="list-style-type: none"> ◆ <i>Chest X-ray</i>: patchy distribution of pneumonia, 	<ul style="list-style-type: none"> ◆ Treat symptoms only ◆ Mortality low; usually clears with no

Type	Signs and symptoms	Diagnosis	Treatment
generally affects young adults)	amounts of mucoid sputum, retrosternal chest pain, anorexia, rhinitis, adenopathy, scattered crackles, and rhonchi	more severe than indicated by physical examination ◆ <i>WBC count:</i> normal to slightly elevated	residual effects
Chicken pox (varicella) (uncommon in children, but present in 30% of adults with varicella)	◆ Cough, dyspnea, cyanosis, tachypnea, pleuritic chest pain, hemoptysis, and rhonchi 1 to 6 days after onset of rash	◆ <i>Chest X-ray:</i> shows more extensive pneumonia than indicated by physical examination and bilateral, patchy, diffuse, nodular infiltrates ◆ <i>Sputum analysis:</i> predominant mononuclear cells and characteristic intranuclear inclusion bodies, with characteristic skin rash, confirm diagnosis	◆ <i>Supportive:</i> adequate hydration, and oxygen therapy in critically ill patients ◆ Therapy with I.V. acyclovir
Viral 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	◆ <i>Chest X-ray:</i> in early stages, variable patchy infiltrates; later, bilateral, nodular, and more predominant in lower lobes ◆ <i>Percutaneous aspiration of lung tissue, transbronchial biopsy, or open lung biopsy:</i> 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 ◆ <i>Supportive:</i> adequate hydration and nutrition, oxygen therapy, and bed rest ◆ In immunosuppressed patients, disease is more severe and may be fatal; ganciclovir or foscarnet treatment is warranted

Type	Signs and symptoms	Diagnosis	Treatment
Influenza (prognosis poor even with treatment; 30% mortality)	<ul style="list-style-type: none"> ◆ Cough (initially 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 	<ul style="list-style-type: none"> ◆ <i>Chest X-ray:</i> diffuse bilateral bronchopneumonia radiating from hilus ◆ <i>WBC count:</i> normal to slightly elevated ◆ <i>Sputum smears:</i> no specific organisms 	<ul style="list-style-type: none"> ◆ <i>Supportive:</i> for respiratory failure, endotracheal intubation and ventilator assistance; for fever, hypothermia blanket or antipyretics; and for influenza A, amantadine or rimantadine
Measles (rubeola)	<ul style="list-style-type: none"> ◆ Fever, dyspnea, cough, small amounts of sputum, coryza, rash, and cervical adenopathy 	<ul style="list-style-type: none"> ◆ <i>Chest X-ray:</i> reticular infiltrates, sometimes with hilar lymph node enlargement ◆ <i>Lung tissue specimen:</i> characteristic giant cells 	<ul style="list-style-type: none"> ◆ <i>Supportive:</i> bed rest, adequate hydration, and antimicrobials; assisted ventilation if necessary
Respiratory syncytial virus (most prevalent in infants and children)	<ul style="list-style-type: none"> ◆ Listlessness, irritability, tachypnea with retraction of intercostal muscles, wheezing, slight sputum production, fine moist crackles, fever, severe malaise, and cough 	<ul style="list-style-type: none"> ◆ <i>Chest X-ray:</i> patchy bilateral consolidation ◆ <i>WBC count:</i> normal to slightly elevated 	<ul style="list-style-type: none"> ◆ <i>Supportive:</i> humidified air, oxygen, antimicrobials (commonly given until viral etiology confirmed), and aerosolized ribavirin ◆ Usually complete recovery

- ◆ 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.

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 (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, 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 2 to 3 years old. In school-age children, mycoplasma pneumonia is more common.

Pathophysiology

A pathogen or extrinsic agent enters the respiratory tract and invades or insults the alveoli. When the host defenses are overwhelmed by the inoculum size or virulence of the pathogen, it results in an infection of the lung parenchyma.

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 PEEP 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 ABG levels, especially in hypoxic patients. Administer supplemental oxygen if the Pao_2 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 them to do so often. In severe pneumonia that requires ET 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, sedation, 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 because 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 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 antibiotic-resistant bacteria. If the patient then develops pneumonia, the organisms producing the pneumonia may require treatment with more toxic antibiotics.
- ◆ Encourage pneumococcal vaccine (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

- *Perform hand hygiene 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 (LE); inflammatory diseases, such as Crohn 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 50 and 60 years old. Incidence is equally divided between men and women. A smoking history doesn't seem to increase the risk of developing BOOP.

Pathophysiology

BOOP is the result of an epithelial injury in the lung that progresses to the formation of fibrinoid inflammatory cell clusters and subsequently intra-

alveolar fibrosis. Upon biopsy the key characteristic is fibroblastic plugs present in the alveoli, alveolar ducts, and bronchioles.

Complications

- ◆ Respiratory failure
- ◆ Interstitial lung disease
- ◆ Death

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 pathophysiology 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 CT scans show areas of consolidation. Except for the migrating opacities, these findings are nonspecific and present in many other respiratory disorders.
- ◆ PFTs may be normal or show reduced capacities. The diffusing capacity for carbon monoxide is generally low.
- ◆ ABG 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, and an increased WBC count with a somewhat 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.

D **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 they exercise.

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

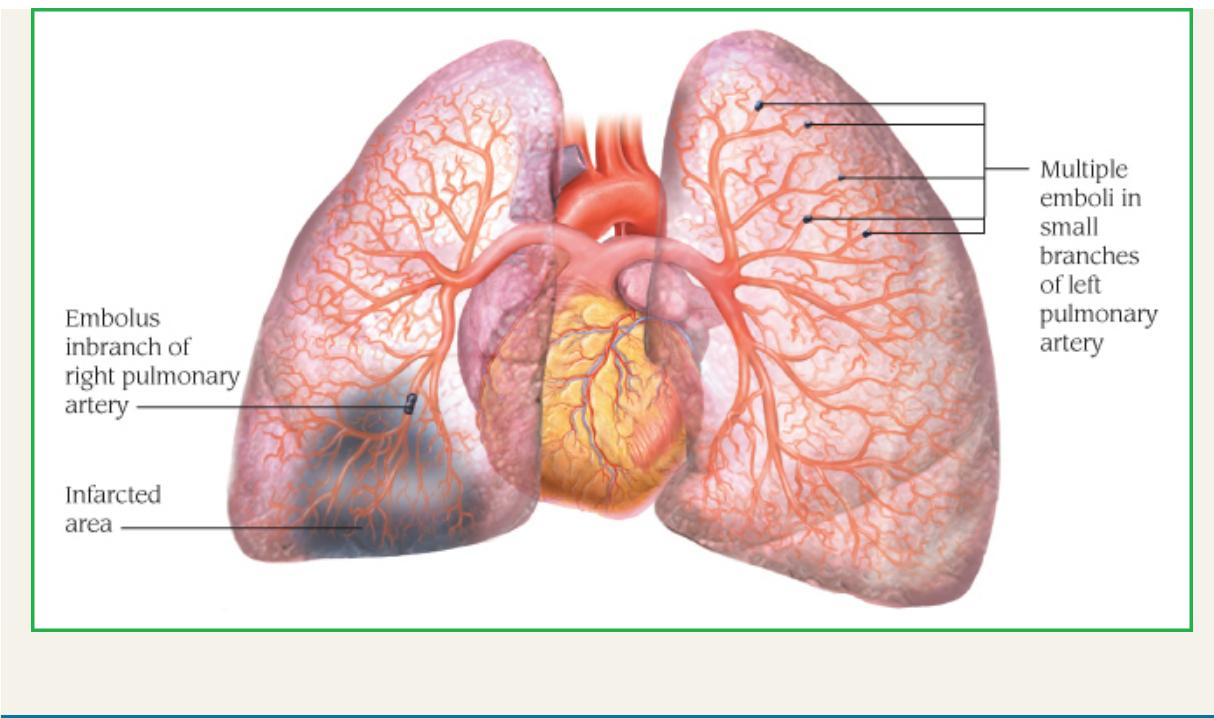
- ◆ Explain all diagnostic tests. The patient may experience anxiety and frustration because of the length of time and number of tests needed to establish the diagnosis.
- ◆ Explain the diagnosis to the patient and family. This uncommon diagnosis may cause confusion and anxiety.
- ◆ Monitor the patient 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 their 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. (See *Looking at pulmonary emboli*, page 120.)

Looking at Pulmonary Emboli

This illustration shows multiple emboli in pulmonary artery branches and a larger embolus that has resulted in an infarcted area in the lung.



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.

Pathophysiology

Once a thrombus dislodges, it travels through to the venous system, through the right side of the heart, and lodges in the pulmonary arteries. The thrombus can completely occlude the vessels or only partially block the flow of blood. Many physiologic factors participate in the potential outcomes including the ability of the body's thrombolytic system to break down clots, function of the right ventricle, over all condition of lungs, and number and size of emboli.

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, a prominent 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; ST-segment depression and T-wave inversions (indicative of right-sided heart strain); and supraventricular tachyarrhythmias in extensive pulmonary embolism. A pattern sometimes observed is S₁, Q₃, and T₃ (S wave in lead I, Q wave in lead III, and inverted T wave in lead III).
- ◆ Auscultation occasionally reveals a right ventricular S₃ gallop and increased intensity of the pulmonic component of S₂. Also, crackles and a pleural rub may be heard at the embolism site.
- ◆ ABG analysis showing a decreased PaO₂ and Paco₂ 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 (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 cava 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 ET 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 them 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 their legs; this promotes thrombus formation.
- ◆ To relieve anxiety, explain procedures and treatments. Encourage the patient's family to participate in their 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-molecular-weight 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 30 to 50 years old. In the United States, sarcoidosis occurs predominantly among blacks, affecting twice as many women as men.

Pathophysiology

In the development of sarcoidosis, T cells play an important role by initiating a significant immune reaction. In sites where there is disease activity, a concentration of CD4 cells exist along with a release of interleukin-2. This results in a disequilibrium of the CD4/CD8 ratio. The exaggerated immune

response creates the characteristic noncaseating granulomas, found primarily on the lungs and intrathoracic lymph nodes.

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
- ◆ CNS—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—noncaseating granulomas with negative cultures for mycobacteria and fungi
- ◆ Other laboratory data—rarely, increased serum calcium, mild anemia, leukocytosis, and hyperglobulinemia
- ◆ PFTs—decreased TLC and compliance, and decreased diffusing capacity
- ◆ 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 (e.g., anemia) 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

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.

Pathophysiology

SARS-CoV is replicated primarily in the lungs but is also found to have the same ability in the gastrointestinal (GI) tract. Once an infection has been established, the virus will cause lysis of cells in the lungs and GI tissues and initiating an immune response. Most tissue damage takes place in the pulmonary tissues and alveoli.

Complications

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

Signs and Symptoms

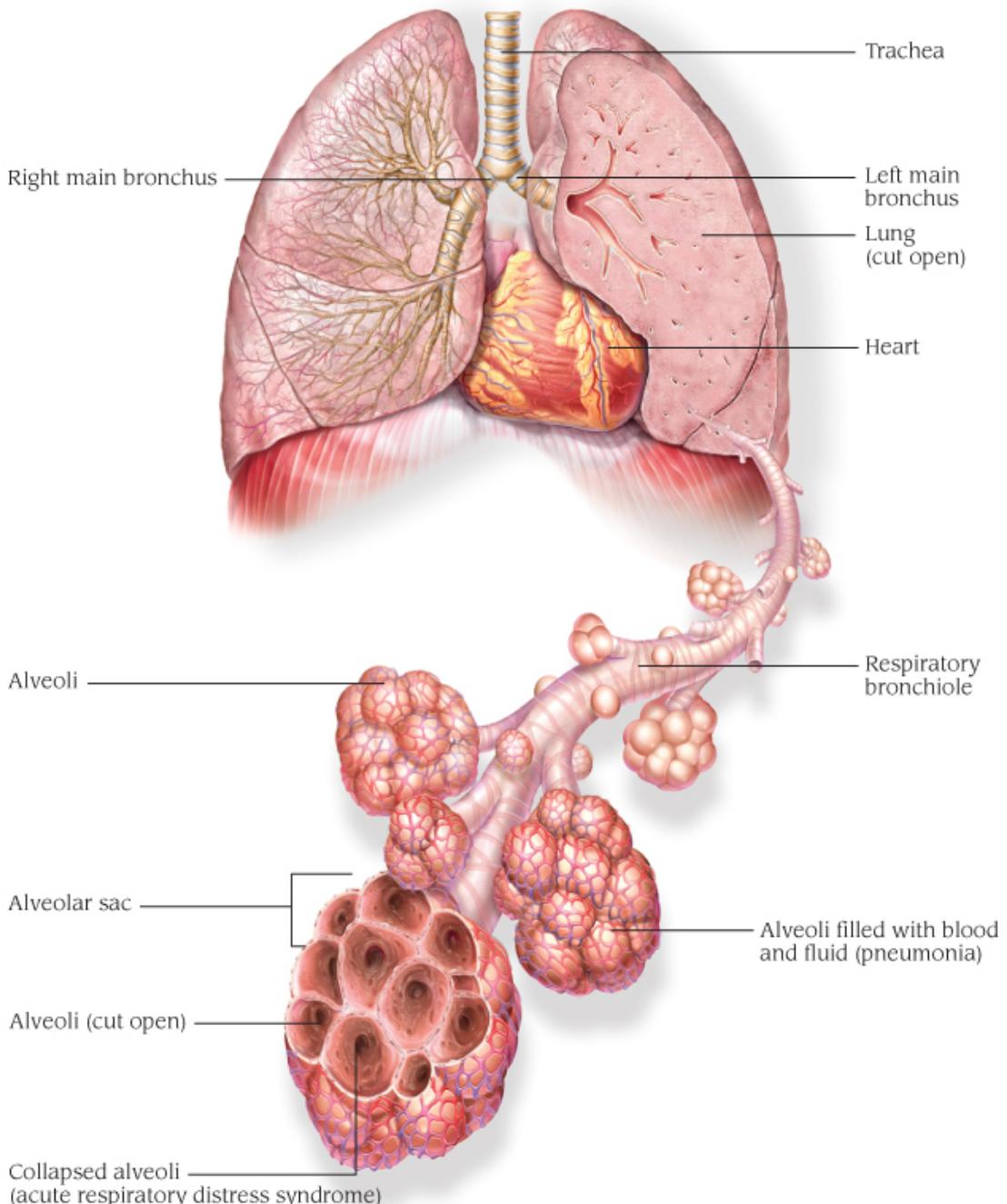
The incubation period for SARS is typically 3 to 5 days but may last as long as 14 days. Initial signs and symptoms include fever, shortness of breath and other minor respiratory symptoms, general discomfort, headache, rigors, chills, myalgia, sore throat, and dry cough. Some individuals may develop 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 ARDS. (See *Lungs and alveoli in SARS*, page 124.)

Lungs and Alveoli in SARS

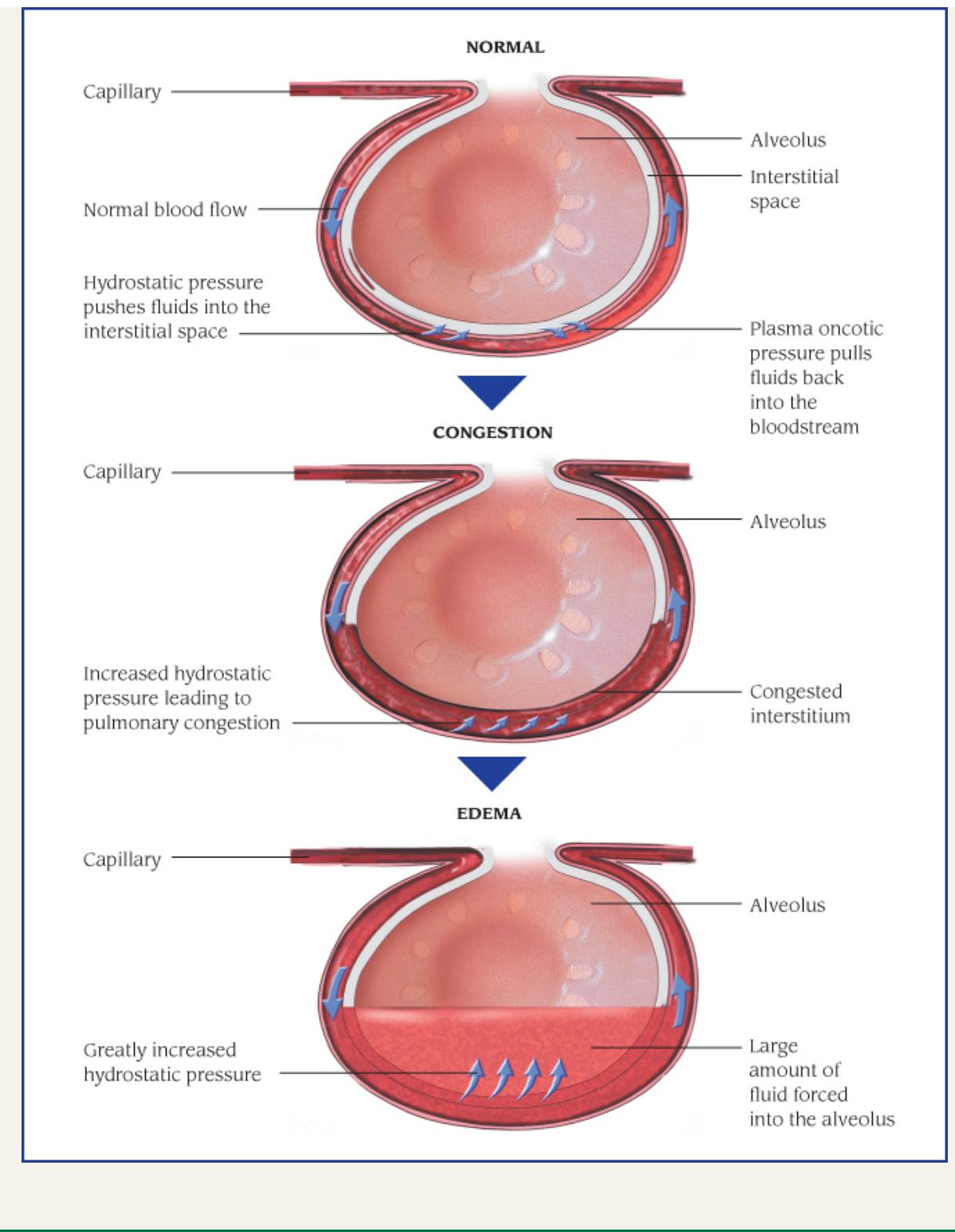
Severe acute respiratory syndrome (SARS) is a viral respiratory infection that can progress from minor respiratory symptoms to pneumonia and eventually death.



PATHOPHYSIOLOGY HOW PULMONARY EDEMA DEVELOPS

In pulmonary edema, diminished function of the left ventricle causes blood to pool there and in the left atrium. Eventually, blood backs up into the pulmonary veins and capillaries.

Increasing capillary hydrostatic pressure pushes fluid into the interstitial spaces and alveoli. The illustrations below show a normal alveolus and the effects of pulmonary edema.



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 N95 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 handwashing, covering the mouth and nose when coughing or sneezing, and avoiding close personal contact while infected or potentially infected. Instruct the patient and 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 healthcare 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.

Pathophysiology

The development of a lung abscess is a response to the introduction of a pathogen, likely from the mouth, into the lower airways. These anaerobes are not cleared away with the body's initial immune response and create a thick-walled cavity filled with purulent material in response to the infection. They can lead to tissue necrosis or possibly the destruction of the involved lung parenchyma.

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 (WBC count greater than 10,000/ μ L) 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 in such a way 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 as a complication of TB or anticoagulant therapy.

Pathophysiology

A hemothorax occurs when a disruption of the chest wall tissues or intrathoracic structures allows blood to enter the pleural space. Depending on the speed and volume of the circulating blood, there can be a significant hemodynamic shift including the early stages of shock.

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. ABG analysis may reveal respiratory failure; Hb 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 re-expand 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, they 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 their 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 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 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 COPD (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 venoocclusive 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 20 and 40 years old. Secondary pulmonary hypertension results from existing cardiac, pulmonary, thromboembolic, or collagen vascular diseases or from the use of certain drugs.

Pathophysiology

The primary pathogenic mechanism involved in pulmonary hypertension is vascular resistance. This resistance typically results from vasoconstriction, remodeling, or a formation of a microthrombus in the pulmonary arteries or arterioles.

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.
- ◆ ABG analysis indicates hypoxemia (decreased Pao₂).
- ◆ 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 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.
- ◆ PFTs may show decreased flow rates and increased residual volume in underlying obstructive disease and decreased TLC 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 they 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 their 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 they have 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 they will need special equipment, such as oxygen equipment, for home use. Make sure that they understand the prescribed medications and diet and the need to weigh themselves 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. Effusions frequently result from heart failure, hepatic disease with ascites, peritoneal dialysis, hypoalbuminemia, and disorders resulting in overexpanded intravascular volume.

Exudative pleural effusions occur with TB, subphrenic abscess, pancreatitis, bacterial or fungal pneumonitis or empyema, malignancy, pulmonary embolism with or without infarction, collagen disease (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.

Pathophysiology

Pleural effusions result from excessive hydrostatic pressure or decreased osmotic pressure causing 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. 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. An empyema results from an infection in the pleural space or accumulated fluid.

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 WBCs 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 (AFB), 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 the patient to expect a stinging sensation from the local anesthetic and a feeling of pressure when the needle is inserted. Instruct them to tell you immediately if they feel uncomfortable or has difficulty breathing during the procedure.
- ◆ Reassure the patient during thoracentesis. Remind them to breathe normally and avoid sudden movements, such as coughing or sighing. Monitor 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, TB, viruses, systemic LE, rheumatoid arthritis, uremia, Dressler 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.

Pathophysiology

There are pain fibers located in the two layers of the pleura; the visceral pleural covers the lung and the parietal pleural line the inner chest wall. Pleurisy or pleuritis is the result of the inflammation of these pleuritic layers, usually from an infectious source.

Complications

- ◆ Pneumonia
- ◆ Pleural effusion
- ◆ Lung collapse

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, TB 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 them to apply firm pressure at the site of the pain during coughing exercises to minimize pain.

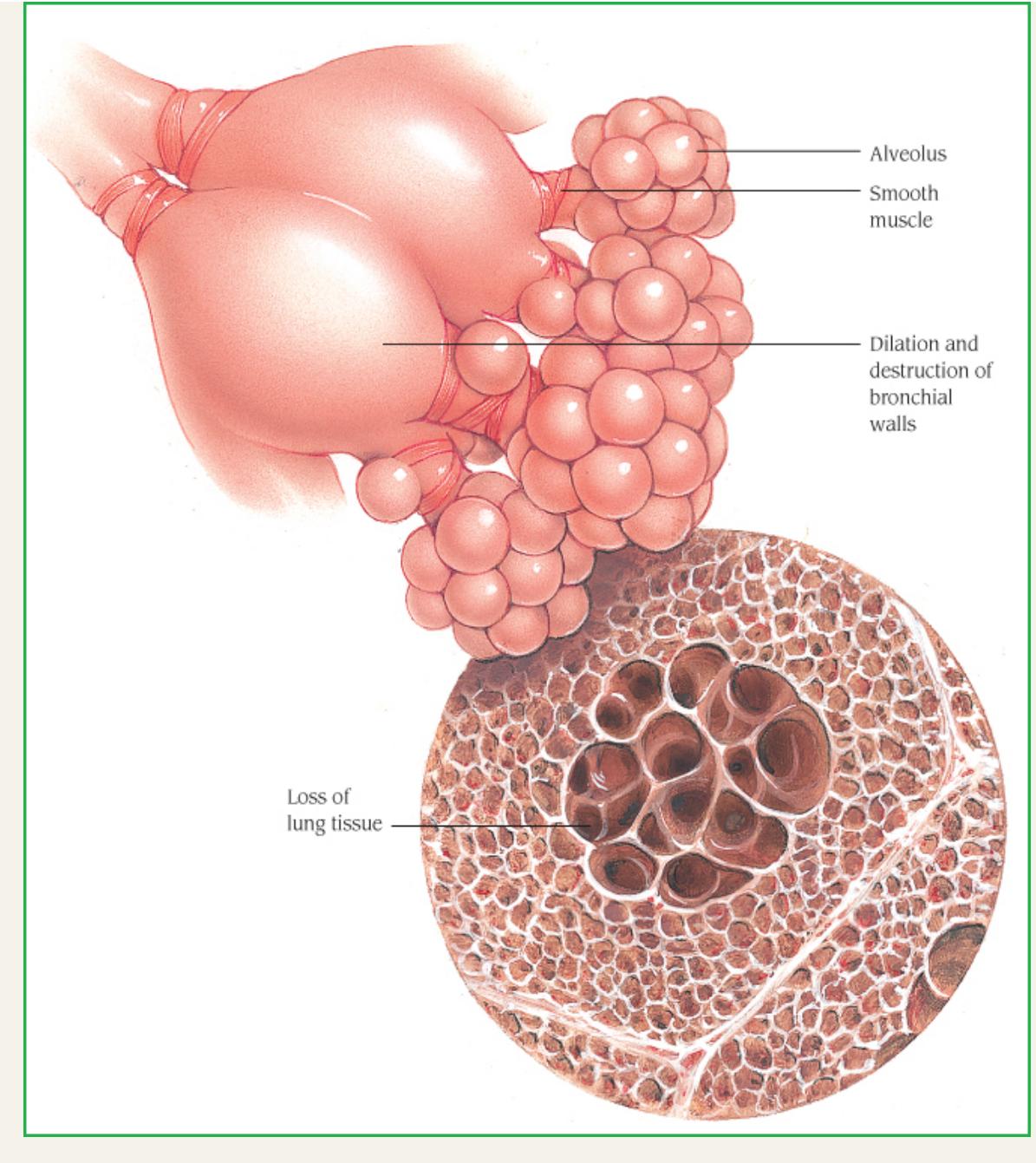
Chronic Disorders

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

COPD is chronic airway obstruction that results from emphysema, chronic bronchitis, asthma, or any combination of these disorders. (See *How pulmonary edema develops*, page 131. Also see *Lung changes in emphysema*, page 132.) 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.

Lung Changes in Emphysema

A form of chronic obstructive pulmonary disease, emphysema is the abnormal, permanent enlargement of the acini accompanied by the destruction of the alveolar walls. Obstruction results from tissue changes, rather than mucus production, as occurs in asthma and chronic bronchitis. The distinguishing characteristic of emphysema is airflow limitation caused by a lack of elastic recoil in the lungs.



Causes and Incidence

Predisposing factors include cigarette smoking, recurrent or chronic respiratory infections, air pollution, occupational exposure to chemicals, and allergies. 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.

Pathophysiology

Repeated exposure to carcinogens, such as smoking, is by far the most important causative action—it impairs ciliary action and macrophage function, inflames airways, increases mucus production, destroys alveolar septae, and causes peribronchiolar fibrosis.

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. The ability to exercise or do strenuous work gradually starts to decline, and the patient 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.

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 patient to enroll.

Urge the patient to stop smoking. Provide smoking cessation counseling or refer them 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 the home may be helpful.

The patient is usually treated with beta-agonist 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 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 or her 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 CO₂ narcosis. If the patient is to continue oxygen therapy at home, teach them 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 PaO_2 , but the patient whose ventilatory drive is largely based on hypoxemia commonly develops markedly increased Paco_2 . In these cases, chemoreceptors in the brain are relatively insensitive to the increase in CO_2 . Teach the patient and family that excessive oxygen therapy may eliminate the hypoxic respiratory drive, causing confusion and drowsiness, signs of CO_2 narcosis.

- ◆ Emphasize the importance of a balanced diet. Because the patient may tire easily when eating, suggest that they eat frequent, small meals and consider using oxygen, administered by nasal cannula, during meals.
- ◆ Help the patient and 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 the provider directs.
- ◆ As COPD progresses, encourage the patient to discuss their 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.

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

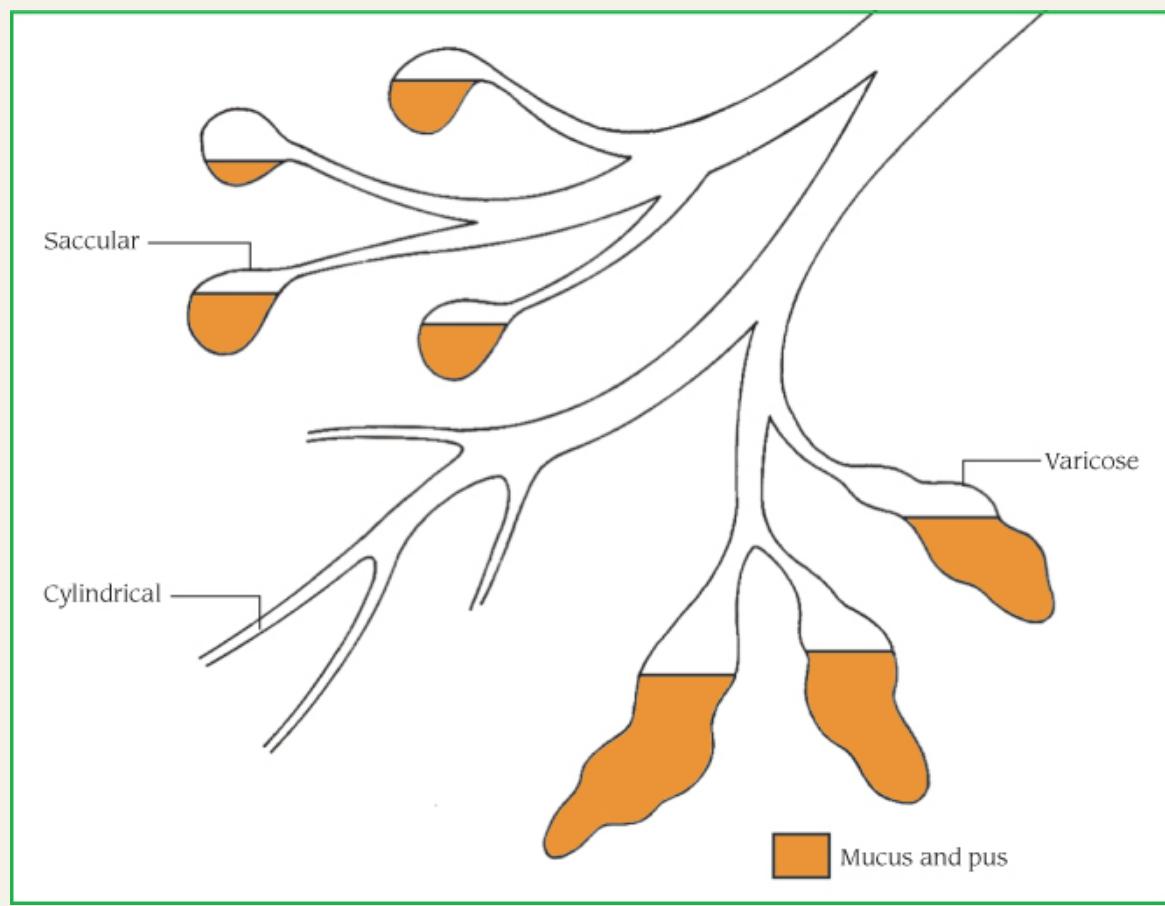
Because of the availability of antibiotics to treat acute respiratory tract infections, the incidence of bronchiectasis has dramatically decreased in the

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*, page 134.)

Forms of Bronchial Dilatation

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



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 (e.g., agammaglobulinemia)
- ◆ recurrent, inadequately treated bacterial respiratory tract infections, such as TB, 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 syndrome, a variant of immotile cilia syndrome characterized by situs inversus, bronchiectasis, and either nasal polyps or sinusitis

Pathophysiology

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 because of 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, CT 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
- ◆ PFTs 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 them 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 them 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])/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 them to seek prompt attention for respiratory infections.
- ◆ Tell the patient to avoid air pollutants and people with upper respiratory tract infections. Instruct them 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 50 to 70 years old.

Pathophysiology

IPF pathophysiology is thought to be initiated when alveolar epithelial cells signal an injury and then activates the excessive formation of fibroblast migration and differentiation. Scarring to the lung structures occurs when the fibroblasts and myofibroblasts secrete large amounts of extracellular matrix proteins, such as collagens, decreasing the overall lung volume.

Complications

- ◆ Respiratory failure
- ◆ Pneumonia
- ◆ Hypoxemia
- ◆ Pneumothorax
- ◆ Pulmonary hypertension

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. Expiratory crackles,

especially in the bases of the lungs, are usually heard early in the disease. Bronchial breath sounds appear later, when airway consolidation develops. Rapid, shallow breathing occurs, especially with exertion, and clubbing has been noted in more than 40% of patients. Late in the disease, cyanosis and evidence of pulmonary hypertension (augmented S₂ and S₃ gallop) commonly occur. As the disease progresses, profound hypoxemia and severe, debilitating dyspnea are the hallmark signs.

Diagnosis

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

Dx 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. Intra-alveolar 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 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 CT 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 TLC and impaired diffusing capacity for carbon monoxide. 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 pathophysiology 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 or she will 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 oxygen flow rate to the appropriate level for exercise.
- ◆ As IPF progresses, the patient's oxygen requirements will increase. They 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 them 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 their 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 their family as they deal with the patient's increasing disability, dyspnea, and probable death. Consult hospice as appropriate.

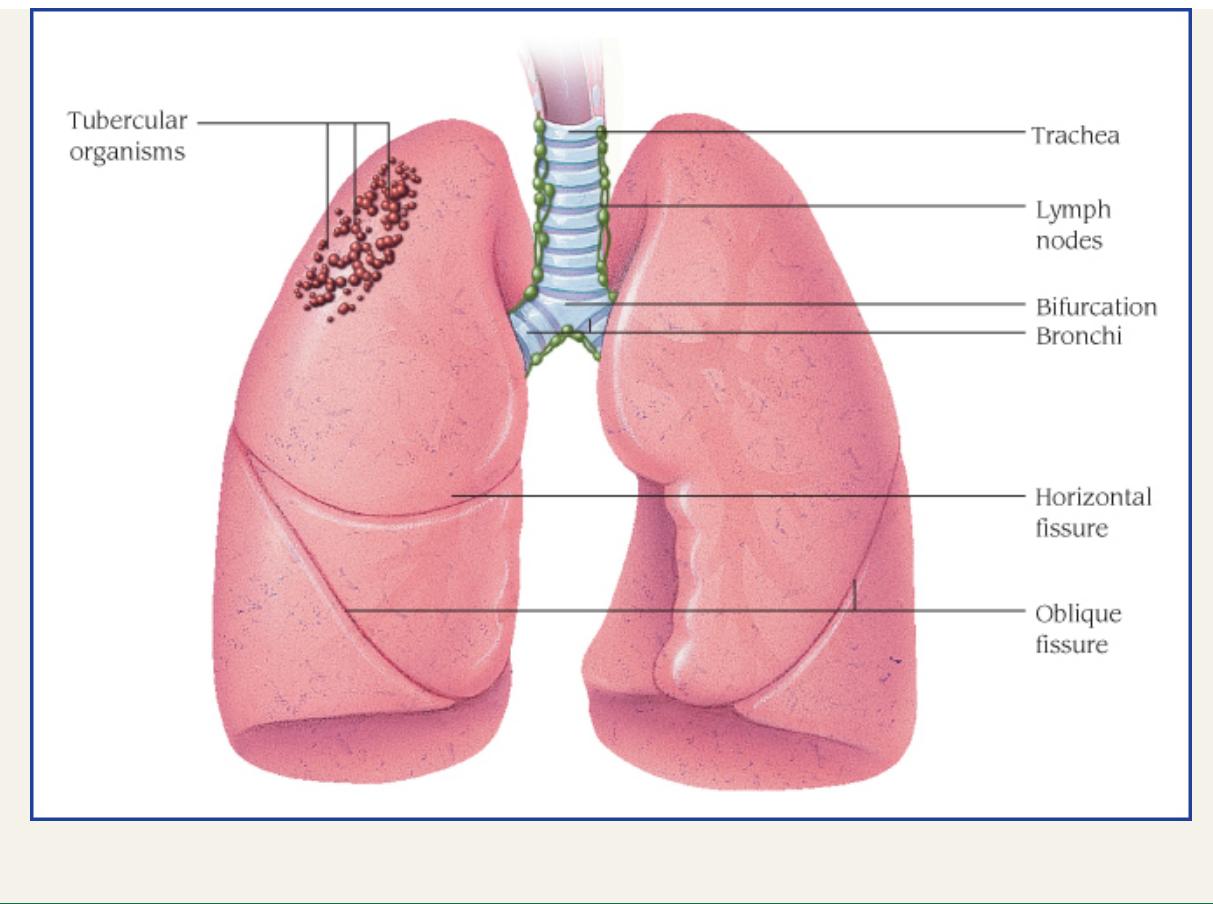
TUBERCULOSIS

An acute or chronic infection caused by *Mycobacterium tuberculosis*, TB is characterized by pulmonary infiltrates, formation of granulomas with caseation, fibrosis, and cavitation. (See *Understanding tuberculosis invasion*.) 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%.



PATHOPHYSIOLOGY UNDERSTANDING TUBERCULOSIS INVASION

After infected droplets are inhaled, they enter the lungs and are deposited either in the lower part of the upper lobe or in the upper part of the lower lobe. Leukocytes surround the droplets, which leads to inflammation. As part of the inflammatory response, some mycobacteria are carried off in the lymphatic circulation by the lymph nodes.



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 lymphoma, leukemia, silicosis, acquired immunodeficiency syndrome, treatment with corticosteroids or immunosuppressants, and advanced age.

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 cheese-like 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.

Pathophysiology

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.

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.

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 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 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 or her 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 or her 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 the patient 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 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. The patient may enroll in a supervised administration program to avoid the development of drug-resistant organisms. (See *Preventing tuberculosis*, page 139.)



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 high-efficiency particulate air 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 they receive annual TB testing. Annual testing is also recommended for healthcare 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 or she may be able to reduce the 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 or she has been taking the medications for 2 to 3 weeks. Encouraging the patient to stay indoors and home from school or work is recommended. If the patient must leave home, a mask is recommended during this initial treatment time to lessen the risk of transmission.

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 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 µm in diameter, but the disease-causing particles deposited in the alveolar space are usually 1 to 3 µm 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.

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.

Pathophysiology

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.

Complications

- ◆ Pulmonary fibrosis
- ◆ Cor pulmonale
- ◆ Ventricular or respiratory failure
- ◆ Pulmonary TB

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. CNS changes—confusion, lethargy, and a decrease in the rate and depth of respiration as the Paco_2 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 PFTs.

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.

PFTs show:

- ◆ FVC—reduced in complicated silicosis
- ◆ FEV₁—reduced in obstructive disease (emphysematous areas of silicosis); reduced in complicated silicosis, but ratio of FEV₁ to FVC is normal or high
- ◆ Maximal voluntary ventilation—reduced in restrictive and obstructive diseases
- ◆ CO₂ 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 or her daily activities to decrease the work of breathing. The patient should be instructed to pace himself or herself, rest often, and generally move slowly through his or her 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 µm or more in length and 0.5 µm 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, protein-like 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.

Pathophysiology

Asbestos fibers penetrate the pleura tissues and are phagocytosed permanently within the lungs. This sets up cycles of cellular events and the release of cytokines. The initial irritation occurs in the alveoli and initiates a surge of asbestos-activated macrophages. These specified macrophages then produce multiple varieties of growth factors that interact to produce fibroblast proliferation.

Complications

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

Signs and Symptoms

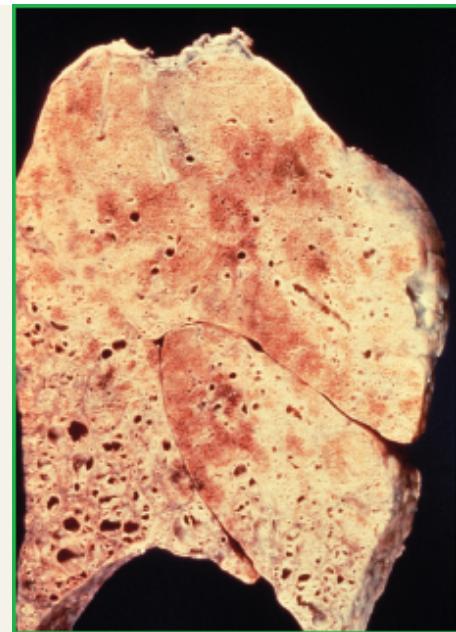
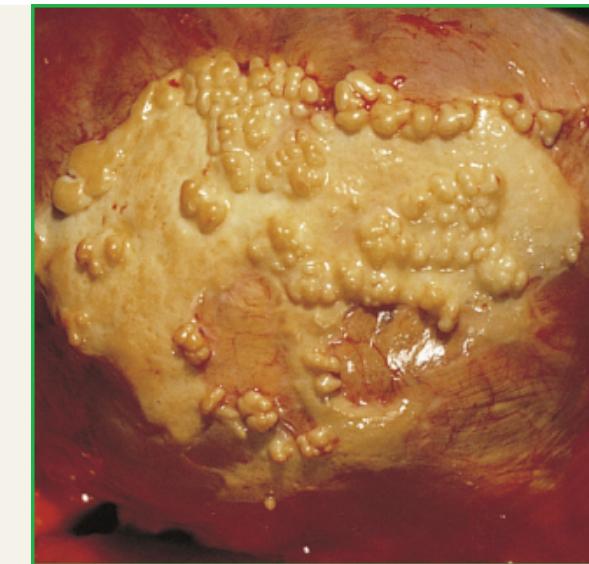
Clinical features may appear before chest X-ray changes. The first symptom is usually dyspnea on exertion, typically after 10 years' exposure. As fibrosis extends, dyspnea on exertion increases until, eventually, dyspnea occurs even at rest. Advanced disease also causes a dry cough (may be productive in smokers), chest pain (commonly pleuritic), recurrent respiratory infections, and tachypnea. (See *A close look at asbestosis*.)

A Close Look at Asbestosis

After years of exposure to asbestos, healthy lung tissue progresses from simple asbestosis to massive pulmonary fibrosis, as shown below.

Simple asbestosis

Progressive massive pulmonary fibrosis



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. CT scan of the lungs also aids in diagnosis.

PFTs show:

- ◆ Vital capacity, FVC, and TLC—decreased
- ◆ FEV₁—decreased or normal
- ◆ Carbon monoxide diffusing capacity—reduced when fibrosis destroys alveolar walls and thickens alveolocapillary membranes

ABG analysis reveals:

- ◆ PaO_2 —decreased
- ◆ PaCO_2 —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 on 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 μm 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.

Pathophysiology

When coal dust particles are inhaled into the lung bronchioles, the carbon is phagocytosed and transported by macrophages and microciliary into the mucus. An immune response is triggered as the coal-filled macrophages accumulate in the alveoli. Reticulin is secreted from the fibroblasts which then entrap the macrophages and become strangulated from the resultant interstitial fibrosis.

Complications

- ◆ Pulmonary hypertension
- ◆ Pulmonary TB
- ◆ 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, cor pulmonale, and pulmonary 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.

PFTs show:

- ◆ Vital capacity—normal in simple CWP but decreased with complicated CWP
- ◆ FEV₁—decreased in complicated disease
- ◆ Residual volume and TLC—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
- ◆ Paco₂—may be increased with concomitant COPD

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 Pao_2 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 pneumococcal vaccine polyvalent and annual influenza vaccines.
- ◆ Encourage the patient to stay active to avoid a deterioration in his or her physical condition but to pace activities and practice relaxation techniques.

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