:
Title: *Professional Guide to Diseases, 9th Edition*

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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*.)

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 *(canaliculi),* 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.

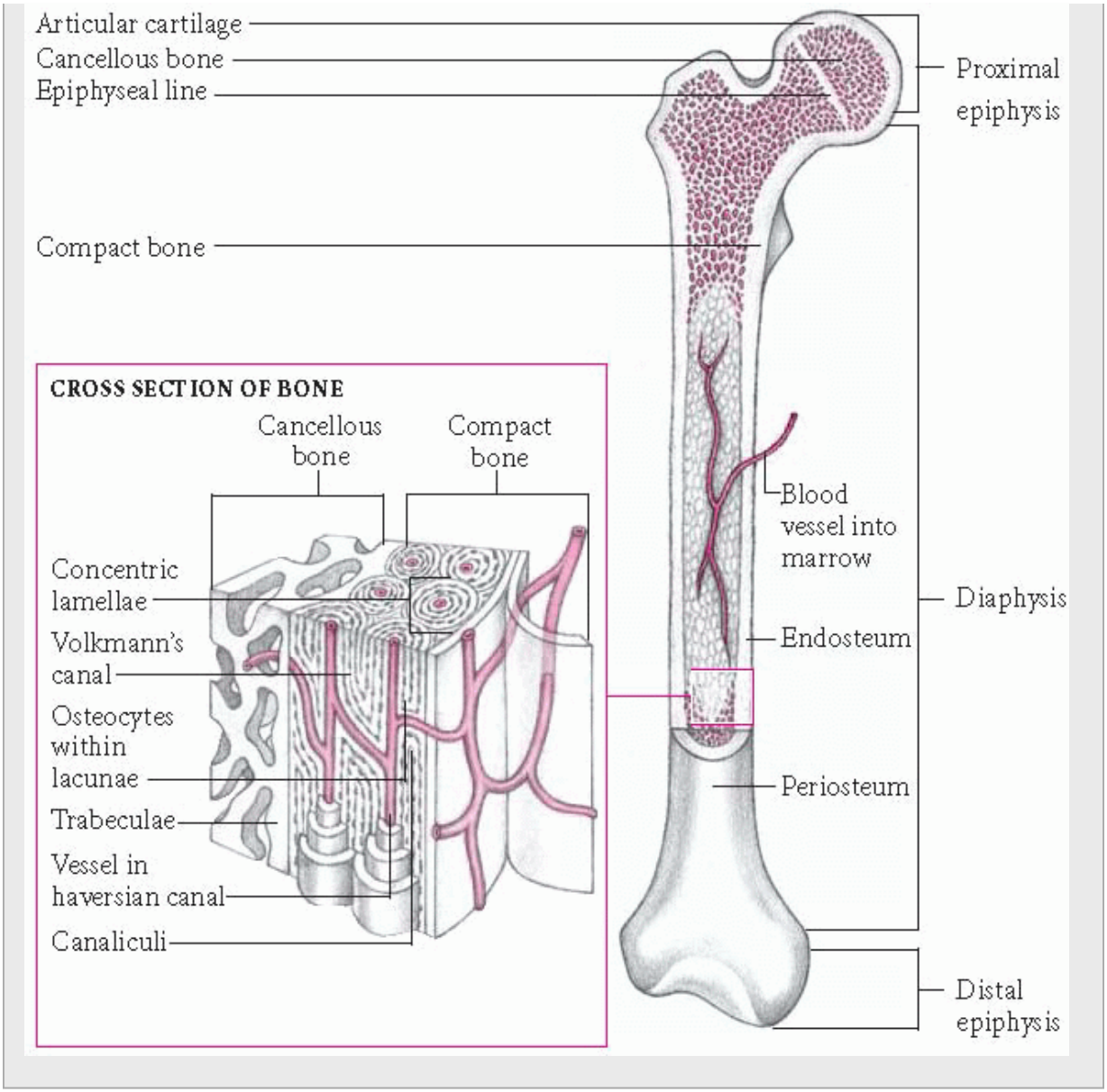
- Fibrous joints *(synarthroses)* have only minute motion and provide stability when tight union is necessary, as in the seams, called *sutures,* that join the cranial bones.

- 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, shoulder, 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.

LONG-BONE STRUCTURE

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



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 the 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 in order 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 (does he jog daily, or is he sedentary?), 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, use of safety devices, and try to assess how the problem will affect body image. Also, ask how he functions at home. Can he perform activities of daily living? Does he have difficulty getting around? Are there stairs where he lives? Where are the bathroom and bedroom? Does he use any prosthetic devices? Ask if other family members can help with his care.

Get an accurate account of the musculoskeletal problem. Ask the patient if it has caused him to change his everyday routine.

When did symptoms begin and how did they progress? Has the patient received treatment for this problem? If he's experienced trauma, find out how he was hurt.

Assess the level of pain. Is the patient in pain at the moment? 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 him about their effectiveness. Does he require more or less medication than before? Did he comply with the prescribed treatment?

The 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, 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 range of motion.

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 is useful in evaluating soft tissue injuries or ligament tears, such as rotator cuff tears or meniscal tears.
- Computed tomography 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 fiber-optic 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 his 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 range-of-motion 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 him about permissible amounts of activity and elevation of the head of the bed. Inform him of the anticipated duration of traction and whether or not the traction is removable. Teach active range-of-motion 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 with his fingers, 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, he'll feel 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 his 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 his 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 him of any activity limitations that must be observed. If the patient has a brace, check with his orthotist (orthopedic appliance specialist) about proper care. Encourage the patient to refer additional questions to his 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 regularly and, if possible, position him 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 range-of-motion (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 maneuver when using the upper arms and trunk to move, instruct the patient to exhale (instead of holding his breath) as he turns. 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 his 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 him finish difficult tasks by himself. 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

A combination of genetic and environmental factors in utero appears to cause clubfoot. 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.

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.

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.

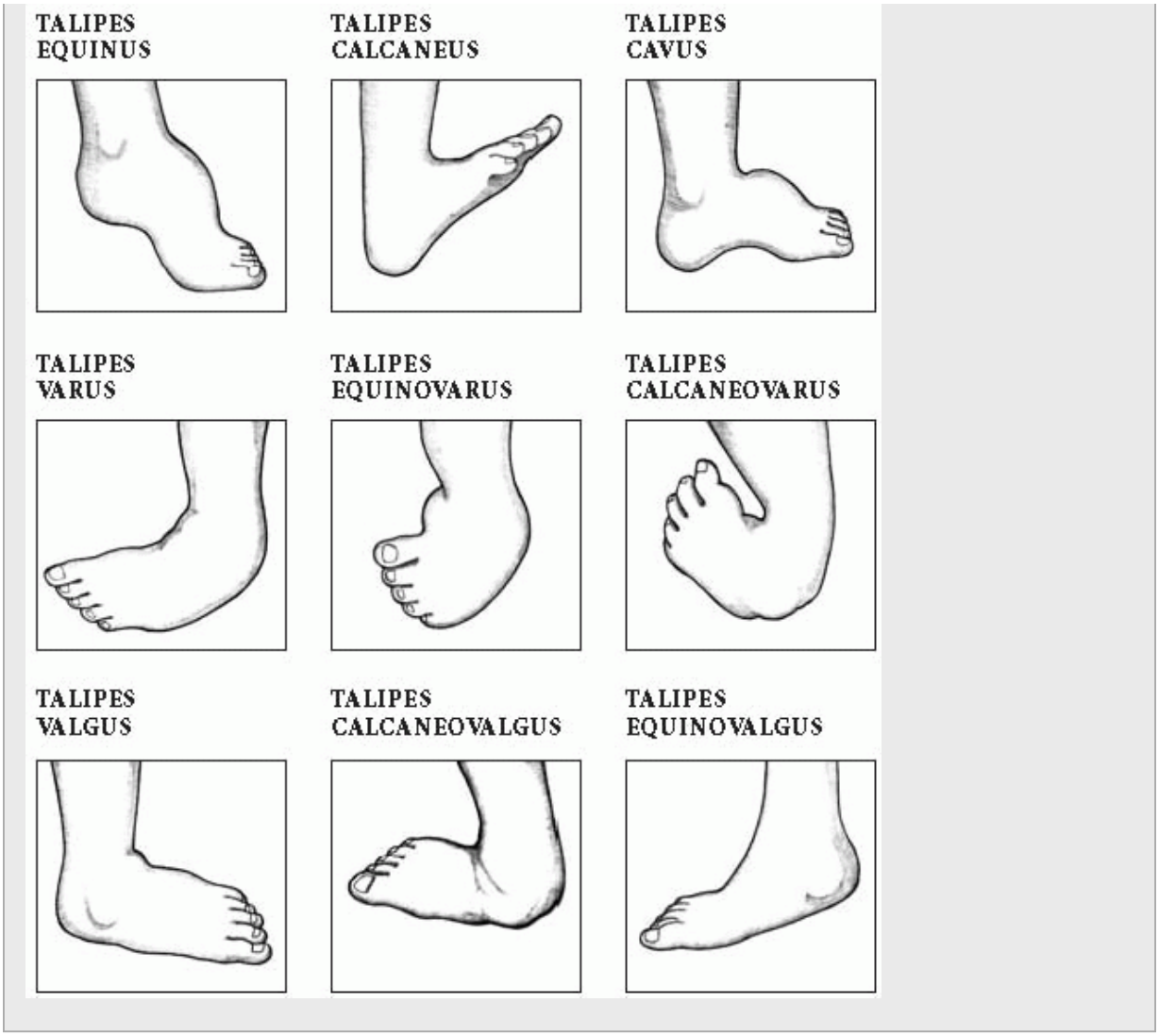
Diagnosis

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 results when a fetus maintains a position in utero that gives his feet a clubfoot appearance at birth. This can usually be corrected manually. Another form of 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

Clubfoot (*talipes*) may have various names, depending on the orientation of the deformity, as shown in the illustrations at right.



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 his 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.

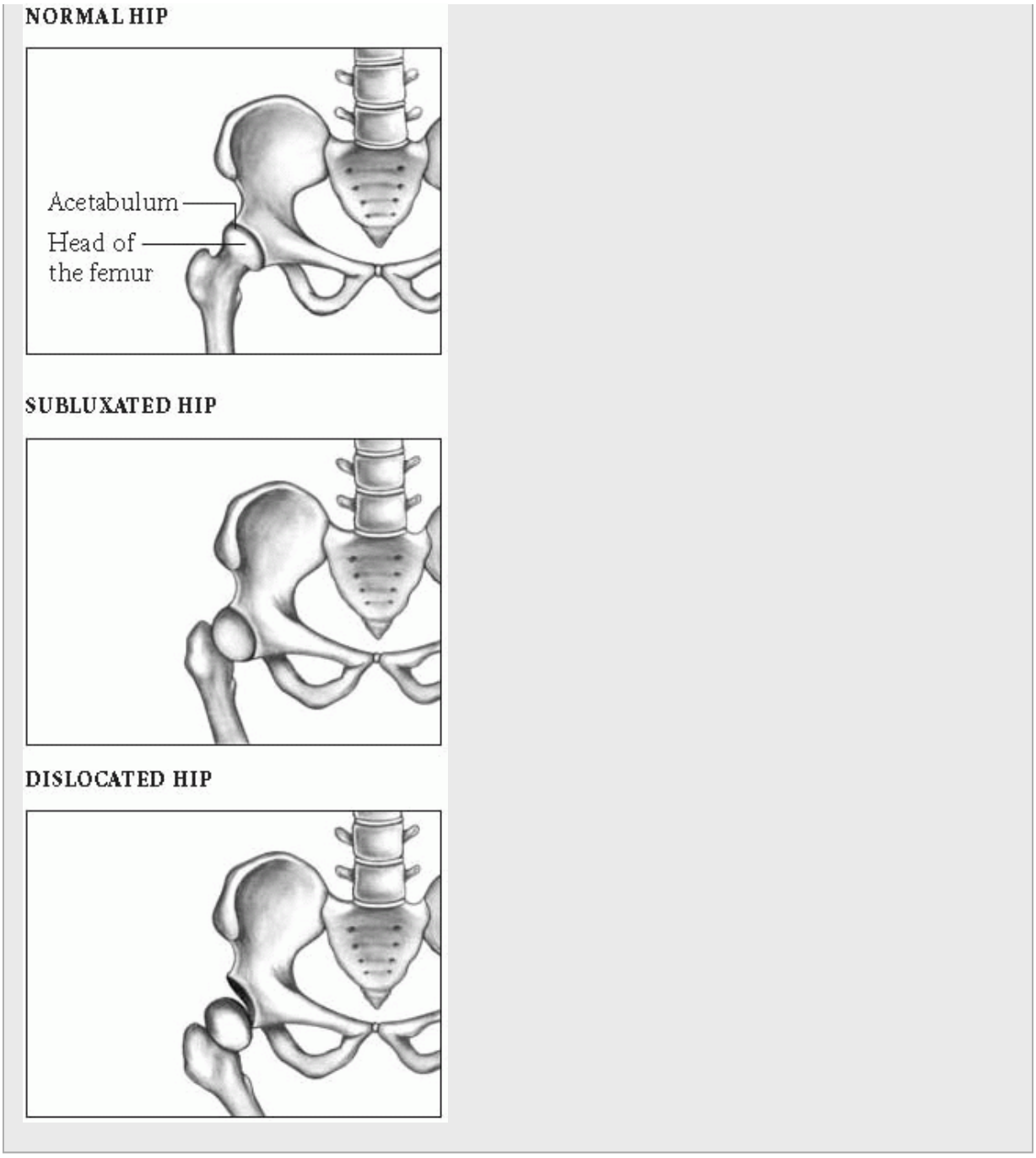
Developmental dysplasia of the hip

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 age 3. DDH can be unilateral or bilateral. 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. (See *Degrees of hip dysplasia.*)

DEGREES OF HIP DYSPLASIA

Normally, the head of the femur fits snugly into the acetabulum, allowing the hip to move properly. In developmental dysplasia of the hip, flattening of the acetabulum prevents the head of the femur from rotating adequately. The child's hip may be unstable, subluxated (partially dislocated), or completely dislocated, with the femoral head lying totally outside the acetabulum. The degree of dysplasia and the child's age are considered in determining the treatment choice.



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.

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 him 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 his back, and inspect the folds of skin over his 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.

CONFIRMING DIAGNOSIS

A positive Ortolani's or Trendelenburg's sign confirms DDH. To elicit Ortolani's sign, place the infant on his back, with his 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's sign, have the child rest his weight on the side of the dislocation and lift his other knee. His pelvis drops on the normal side because of weak abductor muscles in the affected hip. However, when the child stands with his 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. Magnetic resonance imaging may also be used to assess reduction.

Treatment

The earlier the infant receives treatment, the better his 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) in an attempt 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 age 2 to 5 years, 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 developmental hip dislocation, 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, he may be prone to irritability due to the unaccustomed restricted movement. Encourage his parents to stay with him as much as possible and to calm and reassure him.

- 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 his 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 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 his 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 him to interact 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 his parents to a child life specialist to ensure continued developmental progress.

Muscular dystrophy

Muscular dystrophy is actually 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's (pseudohypertrophic), Becker's (benign pseudohypertrophic), facioscapulohumeral (Landouzy-Dejerine), limb-girdle dystrophy, Emery-Dreifuss muscular dystrophy, and myotonia congenita.

The prognosis varies. Duchenne's muscular dystrophy generally strikes during early childhood and usually results in death by age 20. Patients with Becker's 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's and Becker's 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 muscular dystrophy is about 1 in 651,450 persons in the United States. Duchenne's and Becker's 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.

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's muscular dystrophy begins insidiously, between ages 3 and 5. 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 down 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's 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's muscular dystrophy resemble those of Duchenne's muscular dystrophy, but they progress more slowly. Although symptoms start around age 5, the patient can still walk well beyond age 15 — sometimes into his 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's dystrophy and diminished in Becker's dystrophy. Serum creatine kinase level is markedly elevated in Duchenne's, but only moderately elevated in Becker's and facioscapulohumeral dystrophies.

Immunologic and molecular biological assays available in specialized medical centers

facilitate accurate prenatal and postnatal diagnosis of Duchenne's and Becker's 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's.

Special considerations

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

- When respiratory involvement occurs in Duchenne's 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 range-of-motion 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, surgery to correct contractures, 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.
- 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; help him and his family plan a low-calorie, high-protein, high-fiber diet.
- Always allow the patient plenty of time to perform even simple physical tasks because he's likely to be 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's muscular dystrophy to maintain peer relationships and to realize his intellectual

potential by encouraging his parents to keep him in a regular school as long as 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 immunosuppressive 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 age 3. It's uncommon from age 3 until adolescence, at which time the incidence increases again.

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

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/mcl or more white cells, primarily neutrophils. Synovial fluid glucose is usually more than 40 mg/dl. (See *Other types of arthritis,* page 336.)

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 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.

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.

Schönlein-Henoch purpura

Schönlein-Henoch 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.

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 range-of-motion 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.

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.

- Warn the patient before the first aspiration that it will be *extremely* painful. Carefully evaluate the patient's condition after joint aspiration.

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.

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.

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.

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.

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.

Intercritical periods are the symptomfree 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 polyarthritis, 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 white blood cell

(WBC) count may be elevated, and WBC count shifts to the left.

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.

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*.)

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.

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 him 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's 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 age 40 to 60), 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.

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. Computed tomography 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, range of motion, 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.

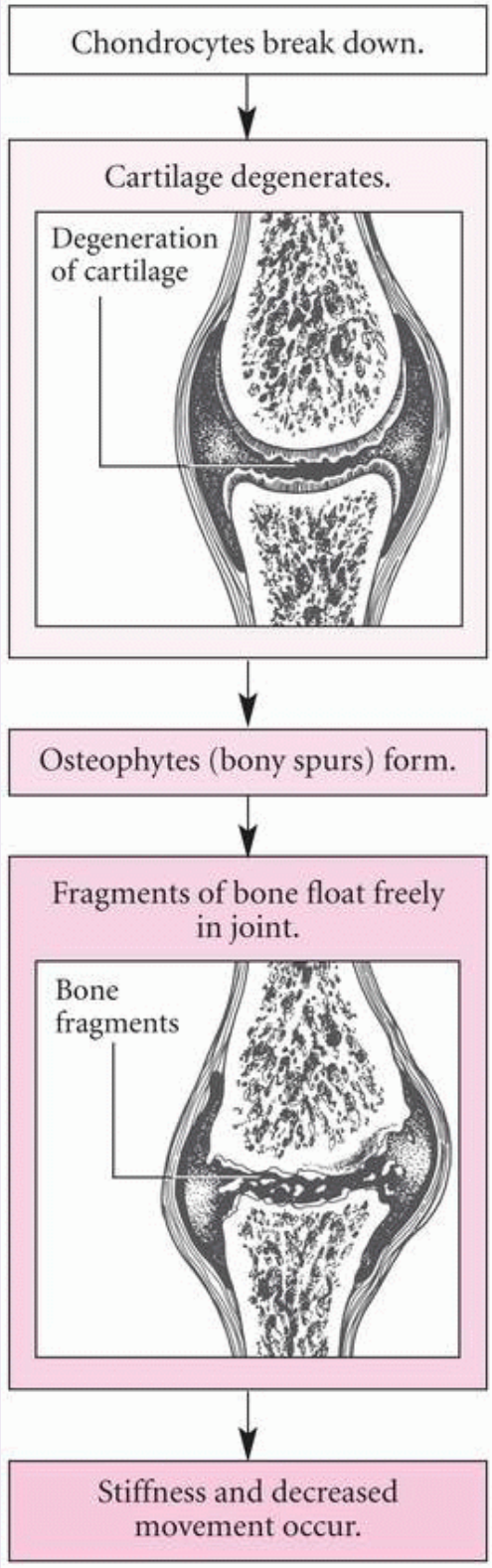
Complications

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

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.



- 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 exercise (relieved by rest), 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.*)

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 *Digital joint deformities.*)

MRI and CT 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 nonsteroidal anti-

inflammatory drugs, COX-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 spine (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:

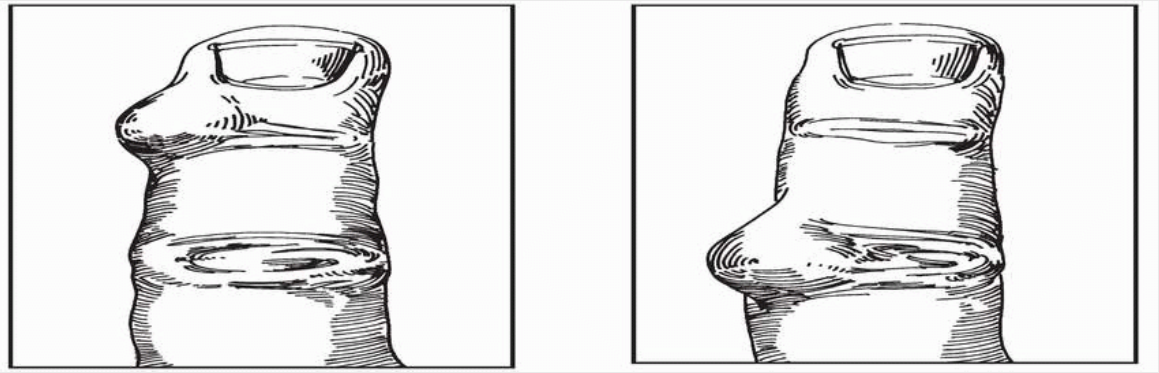
- Promote adequate rest, particularly after activity. Plan rest periods during the day, and provide for adequate sleep at night. Moderation is the key — teach the patient to pace daily activities.
- Assist with physical therapy, and encourage the patient to perform gentle, isometric range-of-motion exercises.
- 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.

DIGITAL JOINT DEFORMITIES

Osteoarthritis of the interphalangeal joints produces irreversible changes in the distal joints (Heberden's nodes, below left) and the proximal joints (Bouchard's nodes, below right). Initially painless, these nodes gradually progress to or suddenly flare up, resulting in redness, swelling, tenderness, and impaired sensation and dexterity.



- *Hip:* Use moist heat pads to relieve pain and administer antispasmodic drugs, as ordered. Assist with range-of-motion 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 range-of-motion 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 report adverse effects immediately.

- Advise the patient to avoid overexertion. He should take care to stand and walk correctly, to minimize weight-bearing activities, and to be especially careful when stooping or picking up objects.
- Instruct the patient to wear properfitting, 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.

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 as a result 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.

Complications

- Chronic osteomylelitis
- Poor joint function
- Amputation of limb

Signs and symptoms

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:

- White blood cell 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.
- Magnetic resonance imaging is best for detecting spinal infection.
- Computed tomography 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 plaster 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 tell him 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. 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.

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.*) 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's atrophy (localized to hands and feet, with recurring attacks), and endocrine disorders (hypopituitarism,

acromegaly, thyrotoxicosis, longstanding diabetes mellitus, hyperthyroidism).

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 age 50 accounting for 20% of cases. Another 30% of women have osteopenia, which can deteriorate into osteoporosis.

Complication

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

Signs and symptoms

Osteoporosis is usually discovered incidentally on roentgenograms; 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's 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 computed tomography scan shows demineralization. Quantitative computed tomography 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 plaster immobilization for 4 to 10 weeks.

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 age 65 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. As long as 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 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 range-of-motion 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 at all times. 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 her 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. Make sure she knows how to wear her back brace.

- Thoroughly explain osteoporosis to the patient and her family. If the patient and her 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-Calvé-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-Calvé-Perthes disease are unknown. Current etiological 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 ages 4 to 10 and tends to occur in families. Although typically unilateral, it occurs bilaterally in 20% of patients.

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 magnetic resonance imaging 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 range-of-motion 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 range of motion. 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 of 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 him to verbalize his 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 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.

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 area of 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's disease

Paget's 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's 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 age 40. 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's disease. Genetic factors are also suspected.

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's 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). Computed tomography scan or magnetic resonance imaging 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 etidronate, alendronate, pamidronate, tiludronate, and risedronate, 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 nonsteroidal anti-inflammatory drugs may be given to control pain.

Special considerations

Patients with Paget's 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 properly and rotate injection sites or how to perform nasal inhalation 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 him how to pace activities and, if necessary, how to use assistive devices. Encourage him 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 patient unaware of new fractures. To prevent falls at home, advise removal of throw rugs and other obstacles.
- Help the patient and his 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.

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*.)

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.

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.

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 a 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 having her dangle her 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 her feet whenever she feels pain or has edema, 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's 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's disease; and from prolonged steroid therapy. Adult kyphosis may also result from conditions such as arthritis, Paget's 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's 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.

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's nodes, irregular end plates, and possibly mild scoliosis of 10 to 20 degrees. Magnetic resonance imaging 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 and teach him 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. Warn him 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 himself.

- 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 him 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 usually occurring in adults (mostly men) less 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.

Complications

- Long-term back pain
- Rarely, spinal cord injuries, such as, loss of movement or sensation in legs and feet, a 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 hyperthesias, 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 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, computed tomography scans, and magnetic resonance imaging (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 nonsteroidal anti-inflammatory drugs, 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. Microdiskectomy 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 him 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. Assure him that he'll receive a sedative before the test, if needed, to keep him as calm and comfortable as possible. After the test, urge the patient to remain in bed with his 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 his 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, microdiskectomy, 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).

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 using 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.

- 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 his 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 his side (never on his 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. Warn him to avoid activities that require alertness until he has built up a tolerance to the drug's sedative effects.
- Provide emotional support. Try to cheer the patient up during periods of frustration and depression. Assure him of his progress, and offer encouragement. (See *Preventing a herniated disk*.)

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. Brain stem 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.

Complications

- Debilitating back pain
- Reduced pulmonary function
- Cor pulmonale

Signs and symptoms

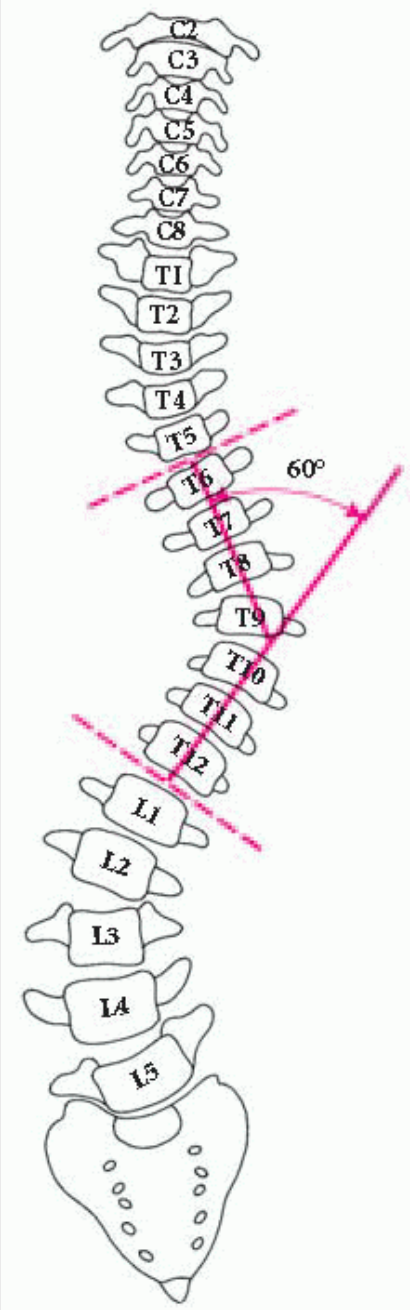
The most common curve in functional or structural scoliosis arises in the thoracic segment, with convexity to the right, and compensatory curves (S curves) in the cervical segment above and the lumbar segment below, both with convexity to the left. (See *Cobb method for measuring angle of curvature*.) 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; 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 less. 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°) 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 per day and to remove it only for bathing and exercise. While he's still adjusting to the brace, tell him 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. Tell him to keep 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 his whole body, instead of just his head, when looking to the side. To make reading easier, tell him to hold the book so he can look straight ahead at it instead of down. If he finds this difficult, help him to obtain prism glasses.

If the patient needs traction or a cast before surgery:

- Explain these procedures to the patient and her 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 range-of-motion 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, calfpumping, and active range-of-motion 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*.)
- Offer emotional support to help prevent depression that may result from altered body image and immobility. Encourage the patient to wear her own clothes, wash her 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.

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.

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.

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 his 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. Computed tomography scan and magnetic resonance imaging (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), nonsteroidal anti-inflammatory drugs (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 longer-term pain relief. Until the patient is free of pain and able to perform range-of-motion 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 range of motion. 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 range of motion to determine effectiveness of the treatment.
- Before injecting corticosteroids or local anesthetics, ask the patient about his 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 he's 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.

Complications

- Recurrence of injury
- Rupture of tendon

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 he grasps an object or twists his elbow. 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 range of motion.

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 involvement or by flexion and pronation with medial epicondyle involvement.

Treatment

Treatment aims to relieve pain, usually by nonsteroidal anti-inflammatory drugs or local injection of corticosteroids and an anesthetic. Supportive treatment includes an immobilizing splint from the distal forearm to the elbow, 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, range of motion, 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 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, he may stretch his arm and flex his wrist to the maximum, then press the back of his hand against a wall until he can feel a pull in his 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 his 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.

Complication

- Permanent weakness

Signs and symptoms

Sharp, spasmodic 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 his 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 having him dangle his foot over the side of the bed for short periods (5 to 15 minutes) before he gets out of bed, allowing for gradual increase of venous pressure. Assist the patient in walking, as ordered (usually within 24 hours of surgery), using crutches and a non-weight-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 him to elevate his 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.

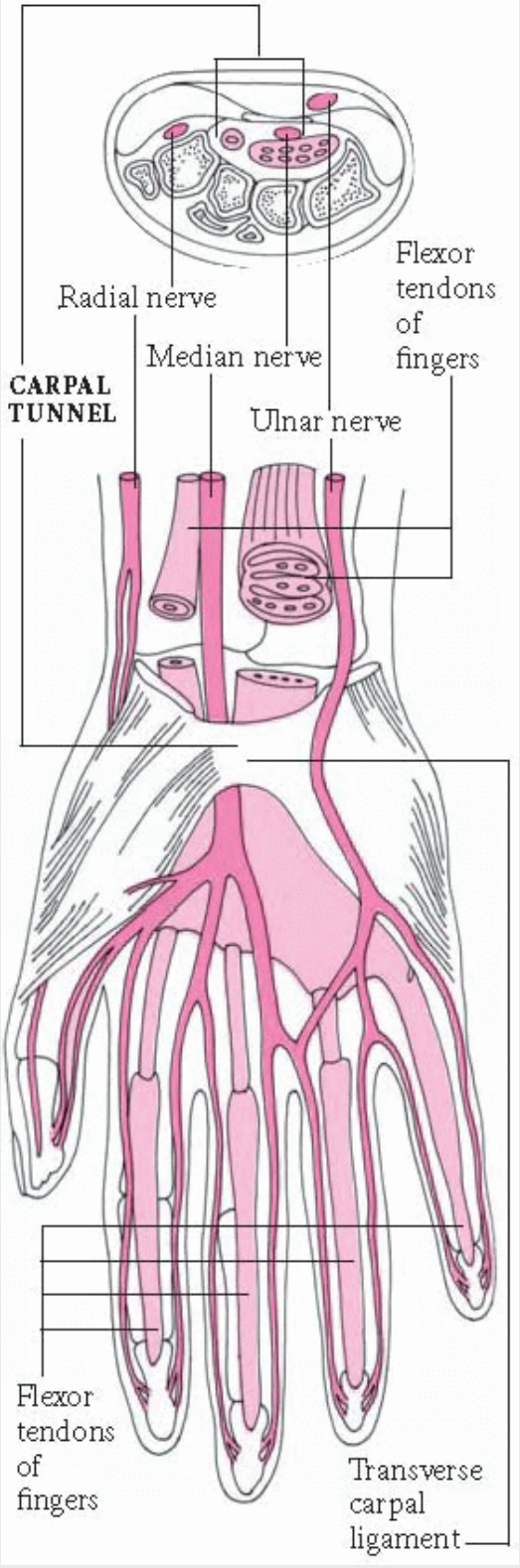
Causes and incidence

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. Such conditions include 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, tuberculosis, 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.


Radial nerve
Median nerve
Ulnar nerve
Flexor tendons of fingers
CARPAL TUNNEL
Flexor tendons of fingers
Transverse carpal ligament

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.

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*.)

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 his 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 his hands vigorously or dangling his arms at his side.

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 milliseconds. 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. Nonsteroidal anti-inflammatory drugs 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, he may have to seek other work. 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 his hands as much as possible. If his dominant hand has been impaired, you may have to help with eating and bathing.
- Teach the patient how to apply a splint. Tell him not to make it too tight. Show him how to remove the splint to perform gentle range-of-motion exercises, which should be done daily. Make sure the patient knows how to do these exercises before he's discharged.
- 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 his hands in warm water. If the arm is in a sling, tell him to remove the sling several times a day to do exercises for his elbow and shoulder.
- Suggest occupational counseling for the patient who has to change jobs because of repetitive stress injury.

Torticollis

Torticollis, sometimes called *wryneck,* is a neck deformity in which the sternocleidomastoid neck muscles are spastic or shortened, causing

bending of the head to the affected side and rotation of the chin to the opposite side.

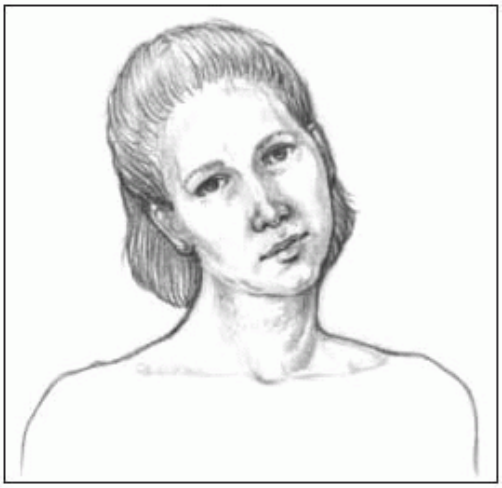
Causes and incidence

Torticollis may be congenital or acquired. The three types of acquired torticollis— acute, spasmodic, and hysterical—have differing causes. The acute form results from muscular damage caused by inflammatory diseases, such as myositis, lymphadenitis, or tuberculosis (TB); from cervical spinal injuries that produce scar tissue contracture; and, less commonly, from tumor or medication. The spasmodic 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. 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 sternocleidomastoid muscle, with hematoma and scar formation.

RECOGNIZING TORTICOLLIS

In torticollis, contraction of the sternocleidomastoid neck muscles produces a twisting of the neck and an unnatural position of the head.



Complication

- Permanent contracture

Signs and symptoms

PEDIATRIC TIP

The first sign of congenital torticollis is commonly a firm, nontender, palpable enlargement of the sternocleidomastoid 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 his head tilts to the shortened side. His 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. (See *Recognizing torticollis.*)

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). Computed tomography scan or magnetic resonance imaging 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 sternocleidomastoid 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 his degree of discomfort.
- Teach the parents of an affected child how to perform stretching exercises with him. 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 his head and stretch his 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 he's 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 his 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 his 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 legionnaire's 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 in children 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's muscular dystrophy, and malignant hyperthermia.

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 his family.
- To help prevent rhabdomyolysis from occurring, ensure adequate hydration, monitor the patient for adverse reactions to any of his prescribed drugs, and monitor blood transfusion administration carefully.

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