inflammation, which can, in turn, produce release of enzymes and trigger nociceptive stimulation.

SOURCES OF PAIN

Because cartilage is aneural, cartilage loss in a joint is not accompanied by pain. Thus, pain in OA likely arises from structures outside the cartilage. Innervated structures in the joint include the synovium, ligaments, joint capsule, muscles, and subchondral bone. Most of these are not visualized by the x-ray, and the severity of x-ray changes in OA correlates poorly with pain severity.

Based on MRI studies in osteoarthritic knees comparing those with and without pain and on studies mapping tenderness in unanesthetized joints, likely sources of pain include synovial inflammation, joint effusions, and bone marrow edema. Modest synovitis develops in many but not all osteoarthritic joints. Some diseased joints have no synovitis, whereas others have synovial inflammation that approaches the severity of joints with rheumatoid arthritis (Chap. 380). The presence of synovitis on MRI is correlated with the presence and severity of knee pain. Capsular stretching from fluid in the joint stimulates nociceptive fibers there, inducing pain. Increased focal loading as part of the disease not only damages cartilage but probably also injures the underlying bone. As a consequence, bone marrow edema appears on the MRI; histologically, this edema signals the presence of microcracks and scar, which are the consequences of trauma. These lesions may stimulate bone nociceptive fibers. Also, hemostatic pressure within bone rises in OA, and the increased pressure itself may stimulate nociceptive fibers, causing pain.

Pain may arise from outside the joint also, including bursae near the joints. Common sources of pain near the knee are anserine bursitis and iliotibial band syndrome.

Persons with chronic OA pain may develop nervous system alterations as a consequence of disease, changes which decrease inhibitory controls on nociception and its distribution. This may produce allodynia and hyperalgesia in some patients with OA.

CLINICAL FEATURES

Joint pain from OA is activity-related. Pain comes on either during or just after joint use and then gradually resolves. Examples include knee or hip pain with going up or down stairs, pain in weight-bearing joints when walking, and, for hand OA, pain when cooking. Early in disease, pain is episodic, triggered often by a day or two of overactive use of a diseased joint, such as a person with knee OA taking a long run and noticing a few days of pain thereafter. As disease progresses, the pain becomes continuous and even begins to be bothersome at night. Stiffness of the affected joint may be prominent, but morning stiffness is usually brief (<30 min).

In knees, buckling may occur, in part, due to weakness of muscles crossing the joint. Mechanical symptoms, such as buckling, catching, or locking, could also signify internal derangement, such as meniscal tears, and need to be evaluated. In the knee, pain with activities requiring knee flexion, such as stair climbing and arising from a chair, often emanates from the patellofemoral compartment of the knee, which does not actively articulate until the knee is bent ~35°.

OA is the most common cause of chronic knee pain in persons over age 45, but the differential diagnosis is long. Inflammatory arthritis is likely if there is prolonged morning stiffness and many other joints are affected. Bursitis occurs commonly around knees and hips. A physical examination should focus on whether tenderness is over the joint line (at the junction of the two bones around which the joint is articulating) or is outside of it. Anserine bursitis, medial and distal to the knee, is an extremely common cause of chronic knee pain that may respond to a glucocorticoid injection. Prominent nocturnal pain in the absence of end-stage OA merits a distinct workup. For hip pain, OA can be detected by loss of internal rotation on passive movement, and pain isolated to an area lateral to the hip joint usually reflects the presence of trochanteric bursitis.

No blood tests are routinely indicated for workup of patients with OA unless symptoms and signs suggest inflammatory arthritis.

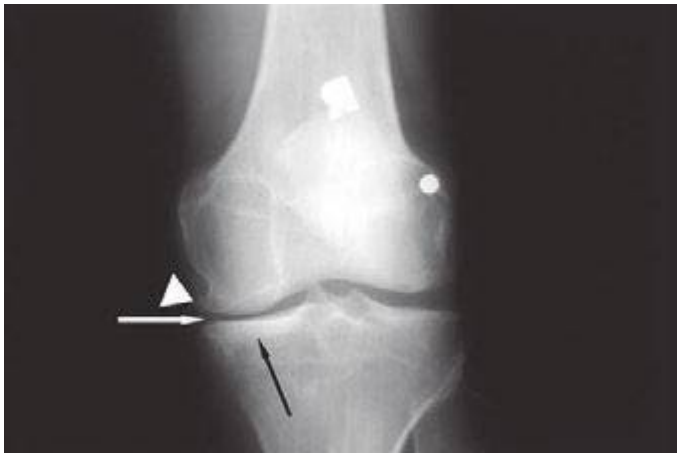

FIGURE 394-7 X-ray of knee with medial osteoarthritis. Note the narrowed joint space on medial side of the joint only (*white arrow*), the sclerosis of the bone in the medial compartment providing evidence of cortical thickening (*black arrow*), and the osteophytes in the medial femur (*white wedge*).

Examination of the synovial fluid is often more helpful diagnostically than an x-ray. If the synovial fluid white count is >1000/μL, inflammatory arthritis or gout or pseudogout is likely, the latter two being also identified by the presence of crystals.

X-rays are indicated to evaluate chronic hand pain and hip pain thought to be due to OA, as the diagnosis is often unclear without confirming radiographs. For knee pain, x-rays should be obtained if symptoms or signs are not typical of OA or if knee pain persists after inauguration of effective treatment. In OA, radiographic findings (Fig. 394-7) correlate poorly with the presence and severity of pain. Further, radiographs may be normal in early disease as they are insensitive to cartilage loss and other early findings.

Although MRI may reveal the extent of pathology in an osteoarthritic joint, it is not indicated as part of the diagnostic workup. Findings such as meniscal tears and cartilage and bone lesions occur in most patients with OA in the knee, but almost never warrant a change in therapy.

TREATMENT OSTEOARTHRITIS

The goals of the treatment of OA are to alleviate pain and minimize loss of physical function. To the extent that pain and loss of function are consequences of inflammation, of weakness across the joint, and of laxity and instability, the treatment of OA involves addressing each of these impairments. Comprehensive therapy consists of a multimodality approach including nonpharmacologic and pharmacologic elements.

Patients with mild and intermittent symptoms may need only reassurance or nonpharmacologic treatments. Patients with ongoing, disabling pain are likely to need both nonpharmacotherapy and pharmacotherapy.

Treatments for knee OA have been more completely evaluated than those for hip and hand OA or for disease in other joints. Thus, although the principles of treatment are identical for OA in all joints, we shall focus below on the treatment of knee OA, noting specific recommendations for disease in other joints, especially when they differ from those for the knee.

NONPHARMACOTHERAPY

Because OA is a mechanically driven disease, the mainstay of treatment involves altering loading across the painful joint and improving the function of joint protectors, so they can better distribute load across the joint. Ways of lessening focal load across the joint include: