Does Osteoarthritis of the Lumbar Spine Cause Chronic Low Back Pain?

David Borenstein, MD

Address
Arthritis and Rheumatism Associates, The George Washington University Medical Center, 2021 K Street, NW, Suite 300, Washington, DC 20006, USA.
E-mail: dborenstein715@aol.com

Current Pain and Headache Reports 2004, **8:**512–517
Current Science Inc. ISSN 1531-3433

The lumbar spine is a common location for osteoarthritis. The axial skeleton demonstrates the same classic alterations of cartilage loss, joint instability, and osteophytosis characteristic of symptomatic disease in the appendages. Despite these similarities, questions remain regarding the lumbar spine facet joints as a source of chronic back pain. The facet joints undergo a progression of degeneration that may result in pain. The facet joints have sensory input from two spinal levels that makes localization of pain difficult. Radiographic studies describe intervertebral disc abnormalities in asymptomatic individuals that are associated with, but not synonymous for, osteoarthritis. Patients who do not have osteoarthritis of the facet joints on magnetic resonance scan do not have back pain. Single photon emission computed tomography scans of the axial skeleton are able to identify painful facet joints with increased activity that may be helped by local anesthetic injections. Low back pain is responsive to therapies that are effective for osteoarthritis in other locations. Osteoarthritis of the lumbar spine does cause low back pain.

Introduction

Low back pain affects 80% of the world's population. The common cold is the only disorder that occurs more frequently than low back pain. Low back pain is a symptom of a wide variety of illnesses [1]. The illnesses that cause low back pain involve musculoskeletal structures as well as the surrounding organs (*eg*, aorta, colon, or kidney). The association of pain with disease in the lumbar spine region is not questioned. What is questioned is the source of discomfort that manifests as pain between the 12th rib and the crease of the buttocks. Anatomic abnormalities, including those of the intervertebral discs, that are common in the lumbar spine occur in asymptomatic individuals [2,3]. The conclusion of many authorities is that these disc abnormalities, including desiccation, protrusion, and outright herniation, and the diseases associated with them (osteoarthritis) are not associated with pain. Is this conclusion correct? Are the pathoanatomic abnormalities that cause radicular pain the same as those that cause somatic joint pain? Is there a time during the natural progression of disc degeneration to osteoarthritis of the lumbar spine and before neural compression associated with spinal stenosis when localized low back pain occurs? The question that needs to be answered is the true relationship between the clinical complaint of back pain and the anatomic and pathophysiologic abnormalities of osteoarthritis of the facet joints. Does osteoarthritis of the facet joints cause chronic low back pain?

Clinical Characteristics and Pathogenesis of Osteoarthritis

Osteoarthritis affects the appendicular and axial skeleton. The joints involved include knees, interphalangeal, hips, and facet joints of the lumbar and cervical spine [4,5]. Osteoarthritis of the lumbar spine causes pain as it does in the knee or hip. Osteoarthritis of the knee is a painful process in many, but not all, individuals who have radiographic evidence of the disease [6].

The process that causes osteoarthritis in the appendages or axial skeleton remains to be elucidated. Aging predisposes to osteoarthritis, but is not sufficient by itself to cause the disorder [7]. Is osteoarthritis a disease of cartilage or subchondral bone? Does joint instability result in the rapid progression of joint destruction? Trauma to the knee joint occurring as a young adult results in instability that is associated with the development of osteoarthritis of the knee [8]. Does weakness of periarticular muscles increase the risk of osteoarthritis? For example, quadricep muscle weakness occurs more frequently in individuals with knee osteoarthritis than in control individuals [9].

The presence of anatomic changes in osteoarthritis is not inadequate to determine those individuals who experience pain from the disorder. Some of the pain generators are radiolucent. Several articular and periarticular structures may be the source of pain in osteoarthritis regardless of the location of the affected joint. Structures similar in the knee and lumbar spine that are sources of pain include synovial cavity, synovium, subchondral bone, osteophytes, joint capsule, ligaments, and muscle. Osteoarthritis of the lumbar facet joint has pathologic and clinical characteristics similar to joints of the appendicular skeleton.

Lumbar spine pain related to osteoarthritis is somatic, not radicular, in nature. Radicular pain is characterized by radiation into the lower extremity. Radicular pain is most frequently caused by nerve compression by a herniated disc or spinal stenosis. Somatic pain related to lumbar spine osteoarthritis causes aching localized to the facet joint and areas lateral to the midline. Osteoarthritis of the lumbar spine may cause pain in the lower extremity related to referred neural pain. These two forms of pain should not be confused.

Facet Joint Anatomy, Nerve Supply, and Biomechanics

The facet joints are composed of articular processes arising from adjacent vertebrae. The articular processes project superiorly and inferiorly from the junction of the pedicles and the laminae. They form true synovial joints with synovial fluid (one superior process from below with one inferior process from above), and their purpose is to stabilize the motion between two vertebrae with respect to translation and torsion while allowing sagittal plane flexion and extension. Each joint is enclosed in a fibrous capsule. The facet joint capsule consists of two layers. The outer layer is a dense connective tissue composed of parallel bundles of collagenous fibers. The inner layer consists of elastic fibers similar to the ligamentum flavum. The fibers in the superior and middle portion of the joint align in the medial-to-lateral direction crossing the joint space. The inferior capsule consists of long diagonal thicker fibers covering the inferior articular recess [10]. The zygapophyseal or facet joints contain menisci that are rudimentary invaginations of the joint capsule that project into the joint space. The menisci function as fillers that provide stability and distribute loads over greater articular areas. The menisci are rarely entrapped between the articular cartilage [11].

The spinal nerve at each lumbar spinal level divides into an anterior and posterior primary ramus. The medial (posterior) branch descends posteriorly at the back of the transverse and superior articular processes to supply sensory fibers to two facet joint levels. The sensory fibers supply the inferior portion of a posterior joint facet and the superior part of the joint capsule at the next lower level. Therefore, each facet joint is supplied by sensory nerves from spinal nerves from two different segments. This organization of the facet joint sensory supply makes specific localization of spinal joint pain more difficult.

The function of the spine is divided between the anterior (static) and posterior (dynamic) portions of each functional unit. Each functional unit is composed of two vertebral bodies—an intervertebral disc anteriorly and facet joints posteriorly. The anterior portion is a weight-bearing, shock-absorbing, and flexible structure. The posterior portion protects the neural elements, acts as a fulcrum, and guides movement for the functional unit.

The posterior portion of the functional unit is composed of the two vertebral arches, two transverse processes, a spinous process, and paired superior and inferior facet joints. The posterior elements share some of the compressive loads and influence the pattern of spine motion. In cadaveric lumbar spines, Adams and Hutton [12] determined the mechanical function of the facet, or apophyseal, joints to be resistant to intervertebral shear forces and compression. Furthermore, the facet joints serve to prevent excessive motion from damaging the discs. In particular, the posterior annulus is protected in torsion by the lumbar facet surfaces and in flexion by the capsular ligaments of the facet joints.

In the lumbar spine, the facet joint planes of motion are vertical, permitting flexion and extension of the spine. In the neutral lordotic position, lateral or rotational movements are prevented because of the apposition of the joint surfaces. In a slightly forward flexed position (decreased lordosis), the facet surfaces separate, allowing some lateral and rotatory movement. With extension, the facet surfaces approximate, preventing any lateral or oblique movement. The extended posture decreases the volume of the lumbar spinal canal and neural foramina [13].

Mechanical injury and resultant inflammation of the apophyseal joint capsule and ligamentous structure of the spine may cause pain. Nerves that supply joints with sensory innervation also frequently supply surrounding muscle, bones, and skin. There is usually an overlap of nerve innervation, with several nerves supplying a single joint. Terminal branches of unmyelinated and myelinated fibers are distributed through the synovium and periosteum. The joint capsule is richly supplied with sensory innervation. Sensory innervation of the joints includes mechanoreceptors in the joint capsule and nociceptors surrounding blood vessels and near the surface of synovial cells. The most painful stimuli to a joint are twisting, tearing, and stretching of the joint capsule or surrounding ligaments. The unmyelinated C nerve fibers of the posterior primary rami of multiple segments supply these structures. Therefore, patients have difficulty identifying the exact location for sources of their pain. Percussion tenderness does not cause as much discomfort in the joints as percussion over muscles in spasm. However, because of the distribution of the sensory and motor nerves, patients with articular and ligamentous disease may develop reflex spasm on sides of the spine and cutaneous hyperesthesia over the same areas.

Mechanical stresses that stimulate nociceptors in the joint capsule may occur secondary to prolonged sitting or standing in inappropriate postures (hyperlordosis with high-heel shoes, soft chair, and sagging mattress), atrophy of paraspinous muscles, excess joint motion secondary to decreased disc or vertebral body height, or malformations. Structural changes affecting articular cartilage and synovium may not be associated with pain, because these tissues contain no free nerve endings. The clinical correlate of this is the lack of relationship between the extent of structural joint changes on radiographic evaluation of the spinal column and the severity of pain. What is visualized on radiographs is not the structure that will necessarily generate nociceptive signals.

Pathogenesis of Lumbar Facet Joint Osteoarthritis

In the first few decades of life, the intervertebral discs will maintain their full height, with a thickened, laminated annulus fibrosus and a tense nucleus pulposus. The vertebrae are completely ossified, except for their apophyseal rings, and are essentially square in shape. The facets are well defined, with smooth capsules and normal articular cartilage. The ligamenta flava are only a few millimeters thick, and the space available for the neural elements within the canal and the foramina is capacious.

Major changes occur in the lumbar spine between the third and fifth decades of life. The first manifestations of aging develop in the intervertebral discs. In the early years, the nucleus loses its vigor and the annulus fissures and degenerates. The first stage of lumbar osteoarthritis is the degeneration of the intervertebral disc. The progression of development of osteoarthritis of the facet joints is from disc space degeneration to joint space narrowing. Butler *et al.* [14] reviewed 68 sets of computed tomography (CT) and magnetic resonance (MR) scans, including 330 discs and 390 facet joints. They discovered 144 degenerated discs with 41 levels of facet osteoarthritis. Disc degeneration without facet arthritis was found at 108 levels, whereas all but one of 41 levels with facet osteoarthritis also had disc degeneration. Disc degeneration and facet osteoarthritis increased with advancing age. Men and women were affected equally. The lower levels had more disease than higher levels. Disc generation occurs before facet joint osteoarthritis that may be secondary to mechanical changes in the pressure on the facet joints.

The resulting biomechanical insufficiency inevitably results in a transfer of stresses posteriorly to the facet joints and ligaments, which are ill-suited to assume compressive, tensile, and shear loads. Capsular strains, hypermobility, and degenerative changes develop. These alterations are similar to those associated with instability of the knee. These changes are often manifested radiologically by traction spurs, which form anteriorly, 1 to 2 mm from the disc. The ligamentum flavum is compelled to assume unnatural tensile loads as the total spine length decreases with disc degeneration. The vertebrae themselves also tend to collapse and spread so as to further compromise the space available for the neural elements. Disc degeneration itself may not be a painful process. Patients with disc degeneration may be asymptomatic until alterations in facet joint alignment result in the onset of articular pain. This stage of illness may be characterized by pain localized to an area just lateral to the midline, over an apophyseal joint, and exacerbated by extension of the spine without radicular radiation of pain.

Fujiwara *et al.* [15,16] completed a series of studies correlating disc degeneration and effects on facet joints and motion of spinal segments. Female motion segments have greater motion in all planes than male segments. Motion diminished with increasing disc degeneration. The resulting facet joint cartilage loss allowed greater motion until severe narrowing occurred. Subchondral sclerosis decreased motion, whereas osteophytes had no significant effect on motion. Reconfiguration of the planes of the facet joints in a more sagittal orientation occurs secondary to osteoarthritis. Osteoarthritic facet joints are the cause of increased spinal instability, resulting in degenerative spondylolisthesis.

As the spine ages, one can also encounter postural alterations with reduction in lordosis. This is an attempt by the body to decompress the degenerated articular facets by maintaining a flexed rather than an extended posture; however, such postural alterations can lead to chronic muscle tension and become symptomatic. This flexed position also provides more room for the sensitive neural elements that are dynamically compressed in extension. Although most of the described changes in the motor segment units progress from decade to decade, there is a wide range in the rate of deterioration. These anatomic alterations do not necessarily dictate symptoms, define disability, or determine prognosis. As the spine ages, these phenomena appear to be tolerated to some degree by all individuals.

In some individuals, the final pathologic endstage of disc degeneration is a fibrous ankylosis between two adjacent vertebrae along with osteophyte formation and a marked narrowing of the disc space. If this is a stable phenomenon, the patient may be relatively free of symptoms or will be aware only of a sense of stiffness in the spine.

Facet Syndrome

Mooney and Robertson [17] reported on the results of the injection of noxious chemicals into facet joints. Distention of degenerated lumbar intervertebral discs or facet joints with injections of saline or contrast material can produce pain in the low back that radiates down the leg. The injections caused back and leg pain. This is not true radicular pain. This is referred pain that appears in mesenchymal structures of the same embryonic sclerotome in the injured tissue. When this type of pain is referred into the back and buttocks, it has a dull, aching quality unlike the sharp, lancinating pain of true sciatica. The symptoms were reversed by injection of anesthetics into the irritated joints. Eisenstein and Parry [18] described the benefits of injections of facet joints in a group of patients without roentgenographic alterations of osteoarthritis. These patients had symptoms relieved with flexion of the spine. These individuals underwent fusion of the lumbar spine. Histologic evaluation of the removed facet joints revealed focal cartilage necrosis without advanced alterations of osteoarthritis.

Jackson *et al.* [19] studied 454 patients with clinical symptoms of back and leg pain compatible with disorders of facet joints. The purpose of the study was to identify clinical characteristics of patients who responded to facet joint injections and document the alleviation of pain and increase in motion immediately after injection of corticosteroids and anesthetic. Injection localization was identified via fluoros-

copy and facet joint arthrography to ensure proper placement of intra-articular injection. Thirty patients (7.7%) had total pain relief after injection. No unique historical or physical examination characteristics identified these patients. Similarly, no specific findings designated 22 patients who had increased pain after injection. Mean pain relief for all patients was 29%. Characteristics of individuals who improved with an injection included older age, history of back pain, normal gait, maximum pain on extension, no leg pain, and muscle spasm.

Schwarzer *et al.* [20] described the pattern of low back pain associated with facet joint disease. Sixty-three patients with chronic low back pain underwent facet joint injection with radiographic imaging. Twenty-three of 57 patients (40%) who completed the study obtained relief of back pain from anesthetic injections. Saline control injections in paraspinous muscles did not alter back pain in these patients. Saline control injection was helpful in 20 (32%) of the remaining patients with chronic back pain. No specific historical or clinical test was useful for differentiating those who would respond to facet joint injection from those improving with saline injections. This study suggests that facet joint pain is a significant cause of low back pain, but cannot be accurately diagnosed without documentation of a response to a joint block. The assumption of this paper is that any response to the muscle injections must be solely placebo effect and excludes the benefit of the medication on the function of the facet joint. The result of this assumption by the authors decreases the frequency of joint disease to a minority of patients with low back pain who had a mean age of 59 years in this study. Joint disease may play a greater role in persistent back pain patients who are older.

Many questions remain regarding the efficacy of facet joint injections and the existence of facet syndrome. The Jackson *et al.* [19] study did not evaluate the long-term effects of facet joint injection. The absence of response cannot be used as the determination of the existence of the syndrome.

Risk Factors for Osteoarthritis of the Lumbar Spine

Osteophytes may not be a source of pain, but are a marker of the processes associated with the development of osteoarthritis. O'Neill *et al.* [21] measured the frequency and distribution of anterior vertebral osteophytes in the lumbar spine and their relationship with physical activity and obesity and self-reported back pain. In 681 women with a mean age of 63.3 years and 499 men with a mean age of 63.7 years, 74% of women and 84% of men had at least one vertebral level with a grade 1 or higher osteophyte. Osteophytes occurred most frequently at L3. Increasing body mass index was associated with more frequent osteophytes. Heavy physical activity, particularly in younger life, was linked with lumbar osteophytes in men. Self-reported back pain, ever or in the past year, was linked with lumbar osteophytes in men.

Radiographic Findings of Lumbar Spine Osteoarthritis

Wybier [22] described the alterations of the lumbar spine that occur with aging. These changes are similar to articular modifications that occur in the appendicular skeleton. Osteophytes and hyperostosis of the apophyseal processes are common radiographic findings. Osteophytes form slowly. In the anteroposterior radiograph, enlarged apophyseal processes project laterally to the lateral cortex of the vertebral body. On the lateral radiograph, the superior apophyseal process erodes at the apex and remodels with an anterior curve, whereas the inferior apophyseal process remodels in a horizontal orientation. Facet joint subchondral bone osteoarthritic modifications include subchondral bone cysts, bone erosions, and bone sclerosis. CT evaluation can document thickness of facet joint cartilage and the narrowing that occurs in osteoarthritic joints. Joint space narrowing is associated with erosive alterations and subluxations. Joint space widening is not necessarily an indication of a healthy joint. Widening in the setting of advanced degenerative changes is indicative of retrolisthesis of the joint. CT and MR axial images document more accurate alterations of the apophyseal processes. The apophyseal joints lose their normal curvilinear alignment. Joints become flattened, allowing for ventral and dorsal movement.

Jarvik and Deyo [23] reported on CT and MR imaging of the lumbar spine in asymptomatic patients with intervertebral disk degeneration. The authors describe a variety of disc alterations that may be unassociated with back pain. The apophyseal joints are not described. This suggests that the facet joints may be a source of pain overlooked in radiographic studies.

Weishaupt *et al.* [24••] completed an important MR study of the lumbar spine that investigated the prevalence of lumbar spine abnormalities in asymptomatic individuals. Sagittal MR images were obtained in 60 asymptomatic volunteers aged 20 to 50 years. The MR images were studied for intervertebral disc abnormalities, endplate abnormalities, and osteoarthritis of the facet joints. Disc bulging or protrusion was found in 42 and 48 of the intervertebral spaces in 37 and 40 subjects, respectively. Disc extrusions were less common with 11 lesions in 11 intervertebral spaces in 11 subjects. Endplate abnormalities were rare. Severe osteoarthritis of the facet joints was absent in the MR images of 60 asymptomatic individuals. The authors suggest that severe osteoarthritis of the facet joints may play an active role in low back pain, because they are not present in asymptomatic individuals.

Correlative findings supporting the role of osteoarthritis of the facet joints as a cause of back pain were reported by Dolan *et al.* [25]. Single photon emission computed tomography (SPECT) bone scanning offers more accurate localization of painful skeletal lesions. Activity can be localized to specific areas of a vertebra, including the vertebral body, pedicle, spinous process, lamina, or facet joint. Facet joints that have increased activity on SPECT scan are more likely sources of pain. Dolan *et al.* [25] studied 58 patients with back pain and clinical findings comparable with facet joint

disease with SPECT scans. Of the 58 patients, 22 had positive scans and 36 were negative. All patients received injections with 40 mg of methylprednisolone and 1 mL 1% lignocaine under radiograph control. Ninety-five percent of the positive-scan patients had pain relief at 1 month, and 73% had pain relief at 3 months. The control patients had no improvement with injections. Joints with increased activity were more responsive to local anesthetic injections, but were not the joints with the greatest degree of osteoarthritis. SPECT scan may identify facet joints with an earlier stage of osteoarthritis that is more painful.

Therapy for Osteoarthritis of the Lumbar Spine

Treatment of osteoarthritis includes physical interventions, drugs, and injections. Dieppe and Brandt [26••] reviewed the benefits of these therapies for osteoarthritis. The benefits of these therapies for appendicular osteoarthritis are modest. These authors believe that nondrug therapies are important in the care of these patients. Many of the therapies for osteoarthritis have generalized effects throughout the body. Nonsteroidal anti-inflammatory drugs decrease pain secondary to osteoarthritis in the knee, hip, hand, or lumbar spine. The cyclooxygenase-2 inhibitors have demonstrated pain relief compared with placebo in patients with low back pain [27]. Some of these patients have osteoarthritis of the facet joints.

More specific therapy for the treatment of osteoarthritis of facet joints is periarticular or intra-articular injections. The confusion that exists regarding effective therapy for facet joint disease is the appropriate location of the injection. Intra-articular injections may decrease local inflammation, but may not numb the medial branch supplying the joint. Conversely, anesthetization of the medial branch may not affect the intra-articular components of osteoarthritis. Therefore, the partial response of facet joint osteoarthritis patients to injections is expected.

Kaplan *et al.* [28] demonstrated the benefits of medial branch blocks for facet joint distention, a manifestation of osteoarthritis. Eighteen asymptomatic individuals had L4-L5 or L5-S1 facet joints injected with contrast to cause pain secondary to capsular swelling. One week later, 15 blinded individuals had randomized saline or lidocaine injection of the medial branch nerve to the corresponding injected joint. The injections were performed so that inadvertent venous uptake was prevented. Thirty minutes later, another injection with anesthesia was given. The control patients developed pain with joint distention. Eight of nine patients who received 2% lidocaine had pain relief with repeat injection. Medial branch injections inhibit pain related to facet joint distention.

Percutaneous radiofrequency neurotomy is another procedure that prolongs the effects of lumbar medial branch anesthetic injections. Dreyfuss *et al.* [29] completed diagnostic lumbar medial branch blocks in 15 patients with chronic low back pain. The patients underwent neurotomy at the corresponding facet joint and were observed over the next 12 months. Sixty percent of patients obtained at least 90% pain relief at 12 months, with 87% receiving at least 60% pain relief. This form of therapy can offer prolonged analgesia for patients with chronic low back pain.

Conclusions

Does osteoarthritis of the lumbar spine cause low back pain? The answer is sometimes. The questions that remain regarding this clinical entity are the same that exist for osteoarthritis in other portions of the skeleton. Clinicians' ability to identify those individuals who suffer pain from osteoarthritis of the lumbar spine results from an inability to identify their discomfort on physical examination because of the absence of differentiating physical findings. Individuals who state that radiographic techniques identify abnormalities in asymptomatic people overlook the majority with other radiographic findings that are significant. Although a radiographic technique may not be necessary early in the evaluation of patients with back pain, it does not mean that the abnormal findings are inconsequential in all individuals.

Patients with low back pain that is increased with standing, worsened with extension of the spine, with facet joint arthritis on plain roentgenograms, and increased uptake on SPECT scan have symptomatic osteoarthritis of the lumbar spine. These individuals should learn the benefits of flexion exercises and the use of non-narcotic analgesics, nonsteroidal anti-inflammatory drugs, or cyclooxygenase-2 inhibitors. They should consider local injections into the joint capsule or blocking the corresponding lumbar medial branch nerve. Neurotomy should be reserved for the rare patients who are not responsive to these noninvasive therapies.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- • Of importance
- •• Of major importance

1. Borenstein D, Wiesel S, Boden S: *Low Back and Neck Pain: Comprehensive Diagnosis and Management.* Philadelphia: Elsevier; 2004.
2. Boden SD, Davis DO, Dina TS, *et al.*: **Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects: a prospective investigation.** *J Bone Joint Surg* 1990, **72:**403–408.
3. Jensen MC, Brant-Zawadzki MN, Obuchowski N, *et al.*: **Magnetic resonance imaging of the lumbar spine in people without back pain.** *N Engl J Med* 1994, **331:**69–73.
4. Hochberg MC: **Osteoarthritis B: clinical features.** In *Primer on the Rheumatic Diseases.* Edited by Klippel JH. Atlanta: Arthritis Foundation; 2001:289–293.
5. Altman RD, Lozada C: **Clinical features.** In *Rheumatology,* edn 3. Edited by Hochberg MC, Silman AJ, Smolen JS, et al. Edinburgh: Mosby; 2003:1793–1800.
6. Loeser RF, Shakoor N: **Aging or osteoarthritis: which is the problem?** *Rheum Dis Clin North Am* 2003, **29:**653–673.

7. Miller ME, Rejeski WJ, Messier SP, Loeser RF: **Modifiers of change in physical functioning in older adults with knee pain: the Observational Arthritis Study in Seniors (OASIS).** *Arthritis Rheum* 2001, **45:**331–339.
8. Gelber AC, Hochberg MC, Mead LA, *et al.*: **Joint injury in young adults and risk for subsequent knee and hip osteoarthritis.** *Ann Intern Med* 2000, **133:**321–328.
9. Fisher NM, Pendergast DR: **Reduced muscle function in patients with osteoarthritis.** *Scand J Rehabil Med* 1997, **29:**213–221.
10. Yamashita T, Minaki Y, Ozaktay AC, *et al.*: **A morphological study of the fibrous capsule of the human lumbar facet joint.** *Spine* 1996, **21:**538–543.
11. Bogduk N, Engel R: **The menisci of the lumbar zygapophyseal joints: a review of their anatomy and clinical significance.** *Spine* 1984, **9:**454–460.
12. Adams MA, Hutton WC: **The mechanical function of the lumbar apophyseal joints.** *Spine* 1983, **8:**327–330.
13. Inufusa A, An HS, Lim TH, *et al.*: **Anatomic changes of the spinal canal and intervertebral foramen associated with flexion-extension movement.** *Spine* 1996, **21:**2412–2420.
14. Butler D, Trafimow JH, Andersson GB, *et al.*: **Discs degenerate before facets.** *Spine* 1990, **15:**111–113.
15. Fujiwara A, Lim T, An HS, *et al.*: **The effect of disc degeneration and facet joint osteoarthritis on the segmental flexibility of the lumbar spin.** *Spine* 2000, **25:**3036–3044.
16. Fujiwara A, Tamai K, An HS, *et al.*: **Orientation and osteoarthritis of the lumbar facet joint.** *Clin Orthop* 2001, **385:**86–94.
17. Mooney V, Robertson J: **The facet syndrome.** *Clin Orthop* 1976, **115:**149–156.
18. Eisenstein SM, Parry CR: **The lumbar facet arthrosis syndrome: clinical presentation and articular surface changes.** *J Bone Joint Surg* 1987, **69:**3–7.
19. Jackson RP, Jacobs RR, Montesano PX: **1988 Volvo Award in clinical sciences. Facet joint injection in low-back pain: a prospective statistical study.** *Spine* 1988, **13:**966–971.
20. Schwarzer AC, Wang SC, Bogduk N, *et al.*: **Prevalence and clinical features of lumbar zygapophyseal joint pain: a study in an Australian population with chronic low back pain.** *Ann Rheum Dis* 1995, **54:**100–106.
21. O'Neill TW, McCloskey EV, Kanis JA, *et al.*: **The distribution, determinants, and clinical correlates of vertebral osteophytosis: a population based survey.** *J Rheumatol* 1999, **26:**842–848.
22. Wybier M: **Imaging of lumbar degenerative changes involving structures other than disk space.** *Radiol Clin North Am* 2001, **39:**101–114.
23. Jarvik JG, Deyo RA: **Imaging of lumbar intervertebral disk degeneration and aging, excluding disk herniations.** *Radiol Clin North AM* 2000, **38:**1255–1266.
24. •• Weishaupt D, Zanetti M, Hodler J, Boos N: **MR imaging of the lumbar spine: prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities, and osteoarthritis of the facet joints in asymptomatic volunteers.** *Radiology* 1998, **209:**661–666.

This paper describes abnormalities that occur in individuals who are asymptomatic in regard to low back pain. Severe osteoarthritis of facet joints was absent in asymptomatic individuals.

25. Dolan AL, Ryan PJ, Arden NK, *et al.*: **The value of SPECT scans in identifying back pain likely to benefit from facet joint injection.** *Br J Rheumatol* 1996, **35:**1269–1273.
26. •• Dieppe P, Brandt KD: **What is important in treating osteoarthritis? Whom should we treat and how should we treat them?** *Rheum Dis Clin North Am* 2003, **29:**687–716.

The therapy of osteoarthritis is multifactorial including physical therapy, drug, and injection modalities. Therapies that are effective for osteoarthritis in the appendicular skeleton are also effective for axial skeletal disease.

27. Katz N, Ju WD, Krupa DA, *et al.*: **Efficacy and safety of rofecoxib in patients with chronic low back pain: results from two 4-week, randomized, placebo-controlled, parallel-group, double-blind trial.** *Spine* 2003, **28:**851–859.
28. Kaplan M, Dreyfuss P, Halbrook B, Bogduk N: **The ability of lumbar medial branch blocks to anesthetize the zygapophyseal joint: a physiologic challenge.** *Spine* 1998, **23:**1847–1852.
29. Dreyfuss P, Halbrook B, Pauza K, *et al.*: **Efficacy and validity of radiofrequency neurotomy for chronic lumbar zygapophyseal joint pain.** *Spine* 2000, **25:**1270–1277.