Anatomy and Pathophysiology of the Sacroiliac Joint

Octavio Calvillo, MD, PhD, Ioannis Skaribas, MD, and Joseph Turnipseed, MD

Address
Center for Pain Medicine, Department of Anesthesiology, Baylor College of Medicine, 6560 Fannin, Suite 1900, Houston, TX 77030, USA.
E-mail: ocalvillo@mysurgeon.com

Current Review of Pain 2000, **4**:356–361
Current Science Inc. ISSN 1069–5850

The sacroiliac joint as a source of chronic pain has been a subject of debate for a long period of time. This controversy stems from the inherent anatomic location of the sacroiliac joint. Adjacent spinal structures may cause pain to be referred to the sacroiliac joint, thus making a precise diagnosis difficult. The most reliable method to establish the diagnosis of sacroiliac arthralgia is fluoroscopic-guided intra-articular injection of a local anesthetic preceded by a sacroiliac arthrogram.

Although there are many therapeutic options for sacroiliac joint syndrome, the ideal treatment has not yet been discovered. There is evidence that intra-articular viscosupplementation of the sacroiliac joint with hylan can consistently and reliably induce a prolonged analgesic response in sacroiliac joint syndrome. Viscosupplementation restores joint homeostasis, allows increased joint motion, and induces analgesia. Hylan is highly viscoelastic hyaluronan (hyaluronic acid), and is capable of increasing the viscoelastic properties of synovial fluid.

The notion that the sacroiliac joint (SIJ) is a source of pain is an old hypothesis. However, this issue continues to create controversy. Some authors consider it a significant source of pain [1,2], whereas others treat the concept with some degree of skepticism [3]. The controversy surrounding this topic stems from the inherent anatomic location of the SIJ. Many of the provocative tests also stress the lumbar spine and the hip joints.

There are other pain-sensitive structures in the lumbar spine that may refer pain to the SIJ region, *ie*, facet joints, intervertebral disks, or nerve roots. This article reviews pertinent anatomic and pathophysiologic data that may help our understanding of SIJ pain and its possible treatment.

Anatomy

The adult SIJ is auricular and C-shaped. The convexity is anterior and somewhat inferior. The long arm of the joint is oriented posterolaterally and caudally, whereas the short arm is directed posteromedially and caudally. Anatomically, the size and shape of the SIJ varies among individuals. The surface of the SIJ at birth is 1.5 cm^2, at puberty it is 7 cm^2, and in the adult it reaches 17.5 cm^2 [4]. The SIJ has been considered to be an amphiarthrosis, *ie*, two hyaline cartilage surfaces joined by fibrocartilage. It has also been classified as a synarthrosis, *ie*, the articular surfaces are joined by fibrous tissue. There is general agreement that the SIJ meets the criteria for a synovial joint, at least in its anterior and inferior portions [4]. The characteristics of a synovial joint are 1) the presence of a joint cavity containing synovial fluid; 2) adjacent bones are united by ligaments; 3) a fibrous capsule surrounds the joint, with an inner synovial lining; and 4) surfaces allow motion. The anterior articular surface is covered with a thin layer of cartilage and is thicker on the sacrum than on the ilium.

Age-related changes

Prior to puberty, the sacral side of the joint appears smooth and cartilaginous. In the second decade of life, a crescent-shaped ridge develops along the iliac surface of the joint, with a corresponding depression on the sacral side [5]. In the third decade, this anatomic change is well developed, thus limiting joint motion in the x-axis.

In men, moderate degenerative changes occur on the iliac side as early as the third decade. These changes are manifested by increased joint irregularity, fibrillation, and crevice formation. Degenerative changes do not affect the sacral side until the fourth decade. Accelerated changes occur in men after the age of fifty. Fibrous ankylosis may develop at this age, giving the SIJ characteristics of an amphiarthrosis. It is possible that these changes predispose to sacroiliac joint syndrome (SIJS); however, this has not been demonstrated [4].

Ligaments

The anterior sacroiliac ligament is a thickening of the anterior joint capsule. It consists of many bands of tissue connecting the anterior surfaces of the sacrum and ilium. The joint capsule is absent posteriorly, and the interosseous ligament forms the posterior border of the joint space. The accessory ligaments are the iliolumbar, sacrotuberous, and sacrospinous [6]. The sacroiliac ligament complex immobilizes the sacrum between the two ilia, thus preventing x-axis rotation secondary to gravi-

tational forces. In men, the ligaments remain well developed. In women, the ligaments are weaker, allowing the mobility that is required during pregnancy and delivery.

Innervation

The synovial capsule of the SIJ and the adjacent ligaments are supplied with small diameter, unmyelinated nerves. Their role is to convey thermal and nociceptive information to the spinal cord. Large diameter fibers also innervate the SIJ, and these probably mediate propioceptive and mechanical information [7].

The nerve supply to the SIJ varies between individuals, and this may account for the variable patterns of referred pain from the SIJ. Posteriorly, the innervation arises from the lateral branches of the posterior primary rami from L4 to S3. The anterior aspect of the SIJ receives innervation from L2 to S2 [7].

Biomechanics

From a physiologic point of view, the SIJ is designed to dissipate the loading of upper body structures to the lower extremities. There are direct connections between the sacrotuberous ligament and the long head of the biceps femoris, the piriformis, and the gluteus maximus muscles. It is probable that these muscles could dynamically influence SIJ mobility, thus leading to SIJ dysfunction [8]. Lumbar spine fusion seems to be a predisposing factor for SIJ dysfunction. Increased uptake within the SIJ during scintigraphy has been demonstrated after spinal fusion and laminectomy [9]. SIJ dysfunction after lumbar fusion is probably due to altered spinal mechanics. The SIJ may be subjected to greater stress after lumbar fusion, thus leading to accelerated degeneration.

Studies on SIJ mobility have been conducted in vivo using a variety of methods. This research on SIJ motion has concentrated mainly on the degree and the axes of movement. There is consensus of opinion that motion must exist in order to create dysfunction, and this may validate certain treatments like manual therapy of the SIJ. Various motions of the SIJ have been proposed. These include gliding, tilting, rotation, and translation. Even though the precise nature of these motions is unclear, SIJ function may be affected by dynamic changes of the lumbar spine, hips, and symphysis pubis [10].

Clinical Presentation

Sacroiliac joint syndrome is considered to be a common source of low back pain. It may be present alone or in association with conditions such as herniated nucleus pulposus, lateral recess stenosis, or a facet syndrome. These conditions share a similar pain referral pattern as SIJS, and it can be difficult to establish a precise diagnosis. The true incidence of SIJS in the general population is unknown. It has been reported to be the primary source of pain in 22.5% of 1293 patients [10]. The physical findings include tenderness over the SIJ sulcus and the posterior SIJ area in general. The muscles adjacent to the SIJ are usually tender as well.

Lumbosacral flexion and extension may elicit pain, whereas lateral motion only rarely evokes pain in SIJS. Neurologic findings are usually absent. The specificity of provocative maneuvers to assess SIJ dysfunction is a questionable issue [11•]. Stressing the SIJ may evoke pain at the hip and the lumbosacral spine, thus making the interpretation of findings difficult.

It has been proposed that the most reliable mechanism to diagnose SIJ pain is fluoroscopic-guided local anesthetic injection [12]. The same authors recommend that the procedure be repeated at least twice using local anesthetics with different duration of action. This process might help eliminate false-positive responses.

The SIJ may be afflicted by a number of conditions, and pain is a common expression of these pathologic processes. Conditions affecting the SIJ include SIJS, inflammatory, infectious, traumatic, degenerative, metabolic, neoplasms, iatrogenic, and referred pain from other sources.

Radiologic Examination

The diagnosis of SIJS is predominantly clinical, and imaging evaluation only rarely adds any useful information. The presence of degenerative changes bears no relation with clinical findings. It has been reported that 24.5% of patients older than 50 years of age have abnormalities on plain radiography [13]. Radionuclide scanning of the SIJ is indicated for demonstrating infection [14], neoplasm [15], inflammation [16], and stress fractures [17].

Referred Pain to the Sacroiliac Joint

Referred pain to the SIJ can be due to a variety of conditions, *ie*, facet syndrome, herniated nucleus pulposus, lateral recess stenosis, internal disk disruption, hip joint disease, and some myofascial syndromes. Lumbar facet disease at L5-S1 can cause referred pain at the SIJ. Nucleus pulposus herniation often causes pain at the SIJ; a detailed history and a deliberate physical examination can help establish a differential diagnosis. Spinal pathology such as internal disc disruption or lateral recess stenosis can be responsible for pain referred to the SIJ. Hip dysfunction and piriformis muscle syndrome can produce a clinical picture resembling SIJS.

Treatment

Treatment of SIJ dysfunction should be directed at restoring joint homeostasis; unfortunately, the ideal treatment has not been discovered. Exercise, joint manipulation, and joint injection provide the possibility of re-establishing

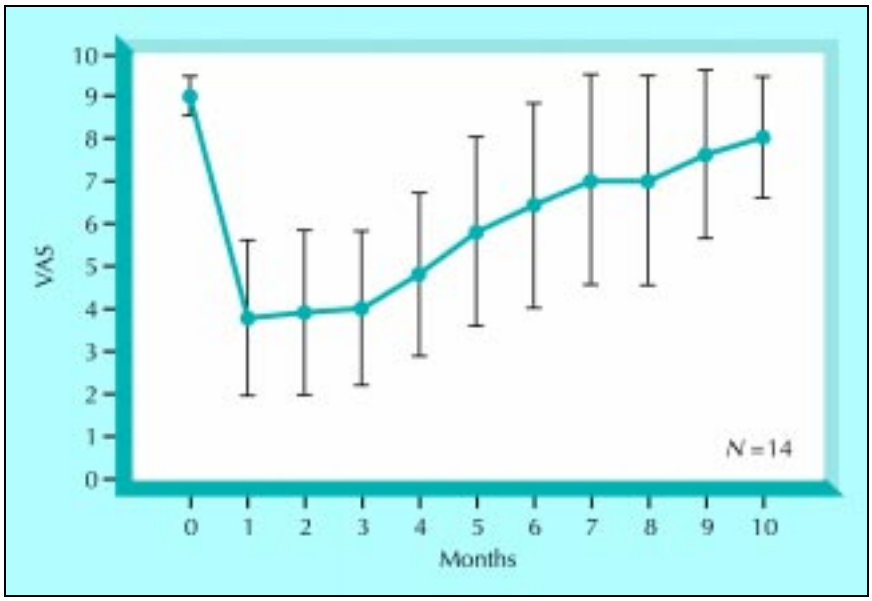

Figure 1. Effects of intra-articular hylan injection in 14 patients suffering from sacroiliac joint syndrome. Hylan was injected three times in each patient, with an interval between injections of 1 week. Pain relief became evident within 60 minutes, and the maximum analgesic effect was reached in about 4 to 5 days. The analgesic effect was better after each injection as well as on completion of the injection series. The pain returned to almost pre-injection levels in about 8 months. The injection series has been repeated in some patients with similar results. VAS—visual analogue scale.

joint mechanics. Calvillo *et al.* [18] have demonstrated that neuroaugmentation of the third sacral root can benefit some patients with resistant SIJ pain. With the exception of joint manipulation [19], there are no controlled, prospective studies on the efficacy of any treatment modality.

It has been suggested that SIJ dysfunction is the result of subluxation of the articular surfaces [19]. Even though this concept is not supported by roentgen and stereophotogrammetric quantitative studies [20], manipulation of the SIJ appears to benefit a certain number of patients. Therapeutic options for SIJS include physical medicine, manipulation under joint anesthesia, intra-articular steroid injection, nonsteroidal anti-inflammatories, analgesics, neuroaugmentation, and viscosupplementation.

Pharmacotherapy

Nonsteroidal anti-inflammatory medication may benefit and sometimes complement other treatments. The advent of highly selective cyclo-oxygenase-2 (COX-2) inhibitors should reduce the incidence of adverse events associated with nonselective COX inhibitors [21].

Sacroiliac joint injection

Intra-articular or periarticular injection of local anesthetic and a steroid can be an effective treatment for SIJS. The duration of the analgesic response is unpredictable, although usually short-lived. Repeated intra-articular steroid injection is not recommended as a long-term treatment for SIJS.

Viscosupplementation

Srejic *et al.* [22••] have reported that viscosupplementation of the SIJ with hylan can afford significant and prolonged pain relief in some patients diagnosed with SIJS. The onset of pain relief is about 45 to 60 minutes, peaking at about 4 to 5 days, and the mean duration can be 6 to 8 months (Fig. 1). Hylan did not resolve the pain permanently; however, the effect was significant and longer lasting than the steroid response. In a number of patients, the injection series has been repeated with similar results. No adverse effects have been noted in any patients.

Pathophysiology of osteoarthritis

Arthropathy of the SIJ can be an isolated entity; however, it is commonly seen as a component of more complex pain syndromes. SIJ pain can be associated with degenerative disk disease, spinal stenosis, facet syndrome, and postlaminectomy syndrome [4].

Lumbar spine arthrodesis seems to be a significant predisposing factor for SIJ dysfunction. It is possible that spinal surgery increases impact loading on the SIJ, leading to mechanical overload and sacroiliitis.

In osteoarthritis, there is progressive erosion of the articular cartilage, synovial inflammation, and changes in the rheological properties of the synovial fluid [23]. Properties of synovial fluid are: 1) it influences intercellular matrices of joint soft tissues; 2) its physiologic role is due to its viscoelasticity; 3) it has a high hyaluronan content; 4) hyaluronan is responsible for the elastoviscous properties of synovial fluid; 5) elastoviscosity is critical for normal joint function; and 6) elastoviscosity is reduced in osteoarthritis. The cartilage is progressively eroded due to a breakdown of type II collagen fibers [24] and degradation of proteoglycan macromolecules [25]. This breakdown is due to increased production of collagenase, stromelysin, and metalloproteases [24]. It is believed that chondrocytes release these enzymes following trauma and inflammation.

Another finding in osteoarthritis is the loss of the viscoelastic properties of the synovial fluid during the course of the disease [26]. The diminished viscoelasticity of synovial fluid increases the susceptibility of cartilage to mechanical wear and tear. The loss of viscoelasticity of the

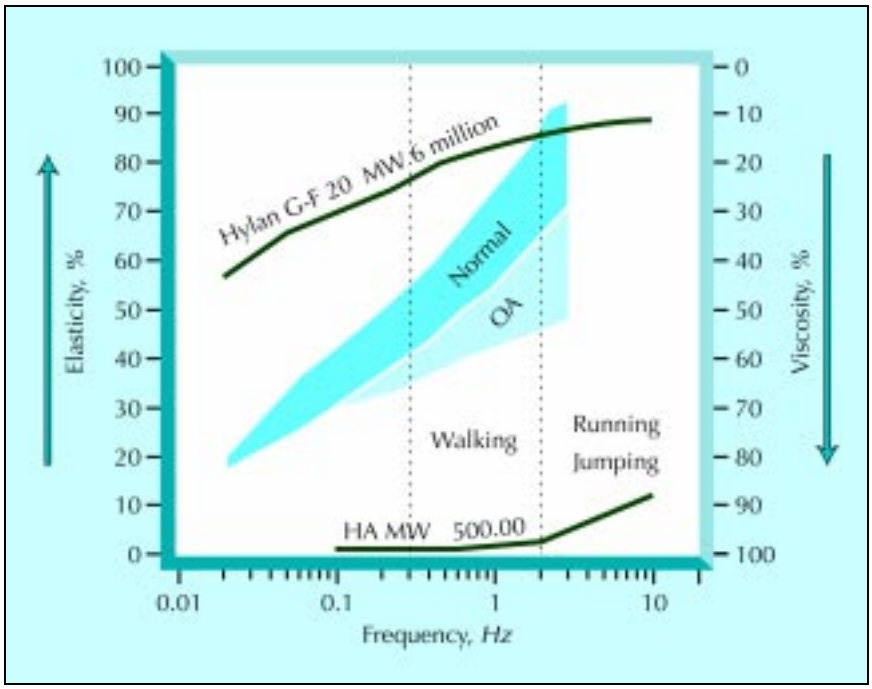

Figure 2. The fundamental principle in viscosupplementation can be appreciated in the graph. At low frequency of the deforming force (shear rate), the viscous behavior of synovial fluid and hylan predominates over the elastic properties. At high deformation frequency, the elastic properties of synovial fluid and hylan become apparent. During normal activities, the behavior of synovial fluid crosses over from predominantly viscous to predominantly elastic. In osteoarthritis (OA), the synovial fluid is less elastic than normal and the crossover from viscous to elastic may not occur, or it may occur much later. HA—hyaluronic acid; MW—molecular weight.

synovial fluid in osteoarthritis results partly from a decrease of hyaluronan. Hyaluronan is one of the major components of synovial fluid.

The loss of the protective effect of hyaluronan with osteoarthritis is due to two main factors. One factor is a decrease in the local amount of hyaluronan present on the cartilage surface; this can be aggravated in the presence of a joint effusion. Another factor may be a decrease in the molecular weight of the synovial fluid, eventually leading to matrix breakdown.

Viscosupplementation is a process that attempts to restore or augment the rheological properties of the synovial fluid. This involves the intra-articular injection of a highly viscous solution. Viscosupplementation replaces pathologic synovial fluid; supplements elasticity and viscosity of synovial fluid; has analgesic properties of hylan that help reduce pain; and improves joint. Hylan has been used extensively in the treatment of osteoarthritis of the knee [27]. It presumably slows the progression of osteoarthritis by inhibiting the diffusion of chondrocyte enzymes into the cartilage. Hylan also protects the proteoglycan macromolecules, probably due to properties related to its high molecular weight. Hylans are comprised of cross-linked hyaluronan; have a high-molecular weight; form a continuous molecular network; have higher elastoviscosity than purified hyaluronan; and have long tissue residence.

Hylan

The elastoviscosity of synovial fluid is entirely due to its hyaluronan content. Hyaluronan is a very long polysaccharide chain and is made up of repeating disaccharide units of *N*-acetylglucosamine and glucuronic acid. Combining 12,500 disaccharide units produces a molecule of hyaluronan with a molecular weight of about 5 million [28].

Hyaluronan is a hydrophilic polysaccharide belonging to the group of glycosaminoglycans. It has a larger molecular volume when fully hydrated, thus occupying a large spheroidal domain. The molecular network of hyaluronan is permeable to molecules that are smaller than the network elements. This network behaves like a sieve for larger molecules [29].

A hyaluronan solution exhibits both elastic and viscous properties [30]. This elastoviscosity is dependent on exposure to shear forces. When mechanical forces induce slower motion and lower shear forces, the solution behaves like a viscous fluid. With more rapid motion and higher shear forces, the behavior resembles that of an elastic body. The elastic properties of the solution mean that the mechanical energy is stored in the molecular network; the viscous behavior means that the mechanical energy is dissipated as heat through the movement of the network (Fig. 2). Consequently, diluted solutions of hyaluronan of sufficiently high molecular weight can function as effective lubricants when movements are slow and as shock absorbers when movements are fast.

Synovial fluid permeates the superficial layer of the articular cartilage as well as the intercellular matrix of the synovial tissue and capsule. This effectively fills the collagen matrix of the intercellular space with viscoelastic hyaluronan [28]. The movement of the joint generates a flow of synovial fluid, maintaining a continuous exchange of hyaluronan between the synovial fluid and the intercellular fluid of the joint tissue.

In the normal joint, the molecular mass of hyaluronan is about 4 to 5 million. The estimated total hyaluronan in

the human knee joint is between 4 to 8 mg. The rheological properties of arthritic synovial fluid are less than that of normal fluid; therefore, a substance intended for viscosupplementation must have considerably greater elastoviscosity than the synovial fluid present in an arthritic joint. This was achieved by the development of a highly elastoviscous solution, composed of two cross-linked hyaluronans. Hylan is an easily deformable gel, with fluid-like properties.

Mechanisms of analgesia of hylan

The exact mechanism of the analgesia with hylan is not known; however, it is reasonable to assume that by restoring joint homeostasis, pain might decrease. The most significant force that drives fluid out of a joint is the pressure exerted during articular motion. Fluid is taken up by lymph vessels in the joint, thus maintaining fluid homeostasis. When fluid accumulates in a joint, the concentration of hyaluronic acid decreases, thus perpetuating a vicious circle. Joint motion decreases in the presence of arthralgia as an antalgic mechanism. Hylan restores the rheological properties of synovial fluid and may improve fluid mechanics in the joint. This probably allows improved joint motion, along with a reduction in pain.

Hylan has been demonstrated to have an effect on joint nociceptors. Pozo *et al.* [31] have provided evidence that intra-articular injection of hylan can decrease nociceptive responses of acutely inflamed joints in rats. The time course of this effect was about 60 minutes; this is remarkably concordant with the time course for analgesia observed by Srejic *et al.* [22••]. Evidently, hylan has analgesic properties that may be due to an anti-bradykinin effect. This mechanism remains to be demonstrated. Therefore, it is possible that the analgesic effect of hylan might be due to its ability to restore joint homeostasis. It is also conceivable that it might have a direct analgesic effect on joint nociceptors.

Conclusions

The true incidence of SIJS is unknown; however, there is general agreement that it is common in the practice of pain medicine [12]. The ideal treatment of SIJS has not yet been defined. Some patients may respond to physical medicine and joint manipulation in conjunction with intra-articular steroid injections. Other patients can be managed with nonsteroidal anti-inflammatories as the sole agent.

Srejic *et al.* [22••] have introduced the concept of SIJ viscosupplementation with hylan (a highly viscous hyaluronic acid derivative). Hylan may induce profound and prolonged pain relief in patients suffering from SIJS. The mechanism of action is probably related to improved rheological properties of synovial fluid. This may restore joint homeostasis, thus reducing pain. Hylan also has a direct effect on articular nociceptors; this may de due to the inhibition of intra-articular bradykinin. SIJ viscosupplementation with hylan might be an option in some patients with SIJS and severe pain.

References and Recommended Reading

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

- • Of importance
- •• Of major importance

1. Greenman PE, Tait B: **Structural diagnosis in chronic low back pain.** *J Manual Med* 1988, **3:**114–117.
2. Beal MC: **The sacroiliac problem: review of anatomy, mechanics and diagnosis.** *J Am Osteopath Assoc* 1982, **81:**667–679.
3. Nachemson AL: **Newest knowledge of low back pain. A critical look.** *Clin Orthop* 1992, **279:**8–20.
4. Bernard Jr TN, Cassidy JD: **The sacroiliac joint syndrome, pathophysiology, diagnosis and treatment.** In *The Adult Spine: Principles and Practice.* Edited by Frymoyer JW. New York: Raven Press; 1991:2107–2130.
5. Weisl H: **The articular surfaces of the sacroiliac joint and their relation to the movements of the sacrum.** *Acta Anat (Basel)* 1954, **22:**1–14.
6. Weisl H: **The ligaments of the sacroiliac joint examined with particular reference to their function.** *Acta Anat (Basel)* 1954, **20:**201–213.
7. Grob KR, Neuhuber WL, Kissling RO: **Innervation of the sacroiliac joint of the human.** *Z Rheumatol* 1995, **54:**117–122.
8. Vleeming A, Stoekart, Snidjers CJ: **The sacrotuberous ligament: a conceptual approach to its dynamic role in stabilizing the sacroiliac joint.** *Clin Biomech* 1989, **4:**201–203.
9. Onsel C, Collier BD, Meting K, *et al.*: **Increased sacroiliac joint uptake after lumbar fusion and or laminectomy.** *Clin Nucl Med* 1992, **17:**283–287.
10. Bernard TN, Kirkaldy-Willis WH: **Recognizing specific characteristics of nonspecific low back pain.** *Clin Orthop* 1987, **217:**266–280.
11. • Bogduk N: **Pain provocation tests for the assessment of sacroiliac joint dysfunction.** *J Spinal Disord* 1999, **12:**357–358.

A fundamental article in the concept of SIJ dysfunction. In particular, it deals with the physical diagnosis of SIJ dysfunction.

12. Schwarzer AC, April CN, Bogduk N: **The sacroiliac joint in chronic low back pain.** *Spine* 1995, **20:**31–37 .
13. Jajic I, Jajic Z: **The prevalence of osteoarthritis of the sacroiliac joint in the urban population.** *Clin Rheumatol* 1987, **6:**39–41.
14. Kerr R: **Pyogenic sacroilitis.** *Orthopedics* 1985, **11:**187–190.
15. Narvaez JA, Narvaez J, Clavaguera MT, *et al.*: **Bone and skeletal muscle metastasis from gastric adenocarcinoma: unusual radiographic and scintigraphic features.** *Eur Radiol* 1998, **8:**1366–1369.
16. Mirzaei S, Wagner E, Knoll P, *et al.*: **Decisive diagnosis of seronegative polyarthritis by 3-phase bone scintigraphy.** *Nuklearmedizin* 1998, **37:**254–256.
17. Marymont JV, Lynch MA, Henning CE: **Exercise related stress reaction of the sacroiliac joint. An unusual cause of low back pain in athletes.** *Am J Sports Med* 1986, **14:**320–323.
18. Calvillo O, Esses S, Ponder C, *et al.*: **Neuroaugmentation in the management of sacroiliac joint pain.** *Spine* 1998, **23:**1069–1072.
19. Dreyfuss P, Michaelsen M, Horne M: **MUJA: manipulation under joint anesthesia/analgesia; a treatment for recalcitrant low back pain of synovial joint origin.** *J Manipulative Physiol Ther* 1995, **18:**537–546.
20. Tullberg T, Blomberg S, Branth B, Johnson R: **Manipulation does not alter the position of the sacroiliac joint. A roentgen stereophotogrammetric analysis.** *Spine* 1998, **23:**1124–1128.
21. Portenoy RK: **Current pharmacotherapy of pain.** *J Pain Symptom Manage* 2000, **19(suppl 1):**S16–20.

22.•• Srejic U, Calvillo O, Kabakibou K: **Viscosupplementation: a new concept in the treatment of sacroiliac joint syndrome: a preliminary report of four cases.** *Reg Anesth Pain Med* 1999, **24:**84–88.

This article demonstrates the application of hylan to treat SIJS. It discusses the mechanism of action of hylan.

23. Leardini G, Mattara I, Franceschini M, Parbellini A: **Intra-articular treatment of knee osteoarthritis: a comparative study between hyaluronic acid and 6-methyl prednisolone acetate.** *Clin Exp Rheumatol* 1991, **9:**375–381.
24. Pelletier JP, Martel-Pelletier J, Howell DS, *et al.*: **Collagenase and collagenolytic activity in human osteoarthritic cartilage.** *Arthritis Rheum* 1983, **26:**63–68.
25. Martel-Pelletier J, Pelletier JP: **Neural proteases and age related changes in human cartilage.** *Ann Rheum Dis* 1987, **46:**363–369.
26. Peyron JG: **Intraarticular hyaluronan injections in the treatment of osteoarthritis: state of the art review.** *J Rheumatol* 1993, **20 (suppl 39):**10–15.
27. Cohen MD: **Hyaluronic acid treatment (viscosupplementation) for OA of the knee.** *Bull Rheum Dis* 1998, **47:**4–7.
28. Balasz EA, Denlinger JL: **Viscosupplementation: a new concept in the treatment of osteoarthritis.** *J Rheumatol* 1993, **20(suppl 39):**3–9.
29. Laurent TC: **The interaction between polysaccharides and other macromolecules. The exclusion of molecules from hyaluronic acid gels and solutions.** *Biochem J* 1964, **63:**106–112.
30. Gibbs DA Merrill EW, Smith KA ,Balasz EA: **The rheology of hyaluronic acid.** *Biopolymers* 1968, **6:**777–791.
31. Pozo MA, Balasz EA, Belmonte C: **Reduction of sensory responses to passive movements of inflamed knee joints by hylan, a hyaluronan derivative.** *Exp Brain Res* 1997, **116:**3–9.