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# Anterior Dysfunction of the Sacroiliac Joint as a Major Factor in the Etiology of Idiopathic Low Back Pain Syndrome

**Richard L. DonTigny**

*The purpose of this article is to describe the author's theories as to how anterior dysfunction of the sacroiliac joints (SIJs) is a major factor in the etiology of idiopathic low back pain syndrome (ILBPS). Most research and treatment have been directed toward the intervertebral disk; however, it is unlikely that disk dysfunction is always the primary etiology. A review of the literature is used to outline and describe the characteristics of ILBPS and to make a case that these characteristics are consistent with those of a specific dysfunction of the SIJs. Functions of the intervertebral disks and the SIJs are described and related to SIJ dysfunction and to some of its common consequences. Treatment is discussed as it relates to the pathomechanics and their correction. [DonTigny RL: Anterior dysfunction of the sacroiliac joint as a major factor in the etiology of idiopathic low back pain syndrome. Phys Ther 70:250–265, 1990]*

**Key Words** *Backache; Kinesiology/biomechanics, trunk; Pain; Sacroiliac joint.*

The ubiquity and complexity of idiopathic low back pain syndrome (ILBPS) is probably unequaled by any other musculoskeletal lesion. Because a multiplicity of tissues have been implicated as etiological sources of musculoskeletal pain, Troup has suggested a multifactorial etiology.[1] Mooney[2] believes that the intervertebral disk is the source of most low back pain, although Nachemson—even after thorough examination—could find no objective cause for the pain in 80% of his patients with low back pain.[3] Because nonspecific back pain often precedes disk herniation, the disk is a likely source of the preceding idiopathic back pain.[4(p319)] Schultz reminds us that idiopathic low back disorders are often ascribed to disk disease, but this etiology has never been proven.[5] Intervertebral disks frequently degenerate without producing any symptoms of low back disorders.[5] White suggests

> It may well be that idiopathic backache will be found to be caused by some condition that is a subtle variation from normal. Otherwise, we probably would have found the cause already. If back pain were caused by a highly unusual condition, then fewer people would suffer from this disorder.[6]

If a primary, nondiskogenic etiology of ILBPS exists, it is probably a subtle, commonly overlooked musculoskeletal condition that may affect many types of tissues and that may mimic or cause disk dysfunction. This condition may not be measurable with currently applied methodology.

The purpose of this article is to present an argument that a dysfunction of the sacroiliac joint (SIJ) is a major factor in the etiology of ILBPS. Characteristics of ILBPS will be delineated and compared with similar characteristics of this subtle SIJD.

## Idiopathic Low Back Pain Syndroma

Certain features appear to be common to ILBPS, although these factors are not readily obvious. These features may be used to describe the essence of ILBPS within which the etiology must lie.

### *Common Characteristics*

Although the severity of the onset of ILBPS may vary considerably among

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different patients, certain aspects of that onset may be similar. The onset may be sudden and specific, such as in the industrial setting where it usually occurs while the patient is leaning forward and lifting, lowering, pushing, or pulling.[7] The onset may also be nontraumatic and insidious. Rowe reported that 65% of workers with low back pain who visited an industrial medical clinic could think of no unusual circumstance associated with the onset of symptoms.[8] The characteristic common to both the sudden and the insidious onset of ILBPS may be as simple as an anterior shift of the line of gravity over the acetabula while leaning forward to perform some task.[9]

Back pain is commonly localized by the patient to the region of one or both SIJs.[4(p315)] When palpating the region of the lumbosacral promontory using an anterior approach, O'Brien found tenderness in more than 75% of patients with low back pain.[10] Of a control group of 50 asymptomatic individuals, only 2 individuals exhibited tenderness and both experienced low back pain during the previous three months. Pain is frequently increased in various positions (eg, sitting, leaning forward when standing) and during coughing or straining, which is usually attributed to the increase in intradiskal pressure that occurs with these activities.[11] Pain may be altered and assessed with passive straight leg raising (PSLR)[9,12] and other movement tests.

In an epidemiological study to determine the magnitude and nature of ILBPS in a population of 6,584 men and women over age 20 years, Valkenburg and Haanen reported large discrepancies among the focus of pain, the radiological findings, and the clinical findings.[13] A large number of people who have severe radiological abnormalities often have no history of low back pain.[13] Radiological findings of computed tomography scans are not always consistent with clinical findings and frequently reveal no obvious abnormalities.[14,15] Macnab noted that a patient may report severe low back pain, and roentgenograms may show evidence of disk degeneration with segmental instability and posterior joint subluxation. After a period of conservative therapy, the pain disappears and the patient returns to heavy work. Follow-up roentgenograms show identical changes even though the patient is symptom-free.[16] Almost every pathological change and lumbosacral anomaly to which back pain has been attributed has subsequently been demonstrated in the symptom-free population.[17,18] In many cases, although roentgenograms are necessary to rule out serious pathology, conventional radiography has not identified any particular lesion common to a predominance of patients with ILBPS. Two things must be considered: 1) A painful musculoskeletal lesion, not readily identifiable by radiography, may exist simultaneously with and independent of these other lesions, and 2) that lesion may exist without other evidence of serious pathology.

A proposed etiology for ILBPS apparently affects, or is affected by, leg length. Giles and Taylor have described a relationship between leg-length inequality, low back pain, pelvic torsion, and pelvic obliquity.[19] Stoddard found that more than twice the number of patients with backache had a leg-length discrepancy, compared with a control group without backache.[20] Greenman recommended that shoe lifts be used only to make the sacral base plane more level and not to equalize leg length or to influence lumbar scoliosis.[21] Grundy and Roberts found that shortening of the lower limb, whatever the anatomical cause, is not a significant causal factor for chronic low back pain.[22] There is a strong association between leg-length inequality and low back pain of at least three months' duration.[19,23] The pain is most frequently found on the side of the long leg,[23] which Chamberlain identified as being caused by a downward rotation of the innominate bone on the sacrum.[24] Unfortunately, these studies considered only that a leg-length discrepancy might cause low back pain and did not consider that the pathomechanics that caused the pain in the low back might also cause a discrepancy in leg length. An observed leg-length discrepancy is frequently reversible and may be changed with mobilization of the SIJs.[9,25–31] This finding suggests a possible biomechanical factor in the etiology of ILBPS.

Idiopathic low back pain may also be capable of spontaneous remission and of stubborn resistance to treatment. It may respond favorably to treatment by manipulative techniques,[9,25–32] and it may or may not be responsive to orthotic supports[32] or traction.[33] I believe that because 90% of all cases of ILBPS resolve within six weeks,[34] the abnormality is probably not attributable to a ligamentous injury. Ligament injuries take between six weeks and six months to fully heal[35]; therefore, most episodes of low back pain do not appear to involve ligamentous injuries.[36]

The frequent inability of investigators to demonstrate a precise pathology with consistent findings has unfortunately led many to suggest that the patient's problem may be psychological in nature. In a study of 141 patients with chronic pain, however, Merskey and Boyd found the emotional disturbance associated with chronic pain to be a secondary effect.[37] Lesions that cause chronic pain can tend to produce psychiatric disturbances.[37] Beals and Hickman[38] and Thompson[39] reported that backache associated with hysteria or malingering was rare.

Nachemson stated

> Whether it is suspected that back pain is caused by an injury to the disc, to the cartilage of the facet joints, to the spongious trabeculae of the vertebral body, or to a muscle, all scientific evidence currently indicates the beneficial effect of motion on symptoms, as well as on healing.[3(p81)]

He recommended early, gradual, biomechanically controlled return to activity and work for the 80% of patients with back pain in whom no


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objective cause for pain can be found after thorough examination.[3] This observation of a beneficial response to early movement is supported by Deyo et al.[40]

Despite a lack of evidence, a huge body of research implicates many other spinal structures as potential sources of low back pain. It is unlikely that movement could so frequently be beneficial to this wide variety of lesioned spinal structures unless a motion-responsive lesion is stressing a multiplicity of pain-sensitive structures. If each affected structure is examined individually without consideration of the basic lesion, it may give the appearance of a multifactorial etiology. In considering the type of lesion, these various structures should respond positively to a precise movement or series of movements.

It appears that not only can the incidence of idiopathic low back pain be as high as 80%, it may be even higher. The pain in patients with identifiable lesions may not be coming from those lesions.[16–18]

Therefore, based on the literature, I believe that ILBPS can be considered as a nonligamentous, reversible, painful lesion that results from joint dysfunction or soft tissue disruption and that is a subtle variation from normal. An anterior shift of the line of gravity also probably occurs. In my opinion, the biomechanics of the pelvis are altered so as to cause an apparent change in leg length, usually an elongation. The principal focus of the pain is in the SIJs posteriorly and near the lumbosacral promontory anteriorly. This pain can be exacerbated with postural changes such as sitting or leaning forward, with an increase of intra-abdominal pressure such as coughing or straining, or with PSLR. The etiology of ILBPS appears to be a subtle, seldom-considered, and commonly overlooked nondiskogenic condition.

### *Consideration of the Disk*

Because the intervertebral disk is often believed to be a common source of low back pain, a common goal of treatment is the relief of intradiskal pressure. Similarly, programs for the prevention of low back pain attempt to minimize compressive forces on the disk. I believe that a review of disk mechanics demonstrates that the effect of the disk on low back pain may not be as great as has been surmised and suggests that disk degeneration may be an effect of the pathomechanics of ILBPS rather than a causative factor.

In symmetric axial loading of the spine, the nucleus pulposus is pressed against the annulus fibrosus, thus providing stability to the disk. This stiffening of the annulus serves as a damper on the axial loading of the spine.[41] In radiographic studies of fresh postmortem vertebra, Vogel[42] and Stahl[43] measured the movements of small metal pins within the nucleus pulposus during asymmetric loading and found that the central part of the disk containing the nucleus pulposus migrated toward the area of least load. The greatest migration took place in the first three minutes at a rate of 0.6 mm/min. This relatively slow migration of the nucleus pulposus prevents rapid and acute flexion of the annulus during asymmetric loading, and stiffening the annular wall on the off-loaded side serves to protect the disk from shear forces. With offset compressive loading, the side of the annulus under compression always bulges and the side under tension stiffens (Fig. 1).[44–50]

With maximal bending, compression, or torsion of the spine, a vertebral fracture arises much sooner than an injury to the intact intervertebral disk.[42(p22),47,48] The increases in intradiskal pressure during normal movements described by Nachemson and Morris reflect normal pressures in the normal disks of subjects without low back pain.[51] The disks are affected only slightly by these pressures. Increased intradiskal pressure is a normal, necessary phenomenon

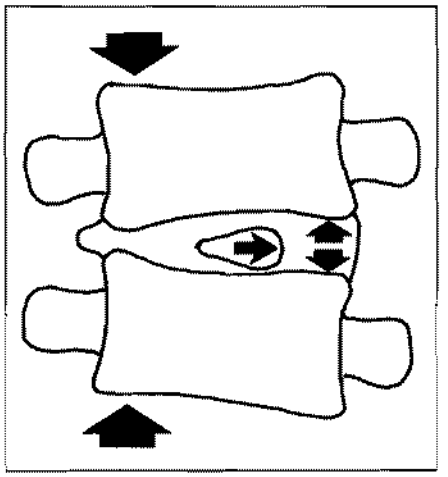

**Fig. 1.** *With offset compressive loading, the nucleus pulposus moves toward the unloaded side, becomes compressed against the inner wall of the annulus, and then acts as a fulcrum that stiffens (but does not bulge) the annulus on the unloaded side. (Courtesy of Forum Medicum Inc.)*

that allows the annulus to tolerate increased loading. Similarly, White and Panjabi have stated that no evidence exists to show that the reduction of intradiskal pressure is therapeutic or relieves pain better than other methods.[45]

Farfan and associates found that the annulus does not tend to fail with compressive loading, but rather as a result of shear loading.[52] They also calculated that a small amount of torsion will produce a relatively large loss of disk volume.[52] Roaf reported that the disk was also subject to injury from horizontal shear forces produced by rotation.[48]

Kramer noted a narrowing of the intervertebral foramina with compression of the nerve roots, caused by an exaggerated cervical and lumbar lordosis associated with a decrease in height of the intervertebral disks.[41(p20)] With hyperextension of the trunk, Cyron et al found that the posterior elements of the spine transmit a substantial part of the load.[53] They concluded that stress fractures leading to spondylolisthesis are likely to be produced by repeated extension under load.[53]


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Holt injected 148 disks in 50 subjects without any history of neck or arm pain or injuries to the cervical spine. In only 10 disks did the contrast medium stay within the annulus.[54] In a similar study of the lumbar spine, Holt found 37% of the disks to be abnormal in 30 subjects with no history of low back pain.[55] Severe pain was produced by the injection of the medium in every subject, indicating an intact and functioning nociceptive receptor system in these abnormal disks.[54,55] Because abnormal disks are commonly found in subjects with no history of pain, pain production is not necessarily an indication of an abnormal disk. Thus, many disks are probably abnormal and degenerated and have some tears in the annulus, but apparently continue to function well and are not usually a source of continuing or even infrequent pain.

The clinical implications of spinal nerve root compression have been well documented[56] and appear to point to disk degeneration as a causative factor. Similar neurological changes, however, can be caused by a stretch of the spinal nerve roots.[57] Nerve roots are more vulnerable to stretch than peripheral nerves because of the absence of perineurium and the parallel nonplexiform arrangement of fibers.[57] During elongation, the cross-sectional area is gradually reduced and a compression deformity of axons and blood vessels is observed. The elastic limit of nerve roots is reached at 15% elongation, and a total mechanical block occurs at or just before the elastic limit.[57] Dorsal root ganglia are more susceptible to mechanical stimulation than axons[58]; therefore, sensory changes may be more common than motor deficits. Traction on nerve roots may also produce a lancinating pain.[59] If the primary etiology of ILBPS is biomechanical, it may also be causing a stretching of the spinal nerve roots and thus mimic disk degeneration. If that primary etiology could also increase shear forces to the disk, degeneration would be facilitated. Thus, in my view, nonspecific back pain may precede disk herniation as a causative factor rather than the disk being the source of the preceding ILBPS.

## Sacroiliac Joints

Most of the research on low back pain has focused on the lumbar spine. Occasional research on the SIJs has generated little evidence of either function or dysfunction. The relative absence of evidence of function or dysfunction of these joints seems to have been interpreted as evidence of the absence of function or dysfunction. The nature of the function of these joints can be determined by analyzing their structure and movement.

### *Structure*

Although the anatomy of the SIJs is well known, the structural mechanics of these joints are less understood. The sacrum has been described as functioning as the keystone of an arch[60,61]; however, the keystone of an arch becomes wedged more tightly as weight is applied from above. The sacrum is actually suspended from the ilia by the dense posterior sacroiliac ligaments and functions as the reverse of a keystone by hanging more deeply between the ilia with increased weight loading[62–65] until it reaches its limit of motion, the posterior superior iliac spines (PSISs) approximate, and further movement of the sacrum downward between the innominates is blocked.[64] Because the sacrum is suspended and thus carried by the ilia, the SIJs are inherently non-weight-bearing joints.

### *Movement*

Various centers of movement of the SIJs have been described by Weisl,[66] Brooke,[67] and Pitkin and Pheasant.[68] Erhard and Bowling have suggested that, for all practical purposes, the only motions permitted are gliding in a ventral and caudal direction and return to the resting position.[69]

In testing movement of the SIJs of a standing subject who flexes the trunk forward in the sagittal plane, the clinician should place his index fingers of each hand on each PSIS and the thumbs together on the sacrum. The PSISs can then be observed to rise and diverge slightly on the sacrum as the innominates rotate anteriorly around the acetabula. In my opinion, this movement is indicative of a transverse axis through or near the central aspect of the SIJ. Mennell[70] and Mennell[71] have described this transverse axis of rotation with flexion and extension of the trunk on the pelvis in the sagittal plane. Bourdillon described an oblique axis through the SIJs with oblique trunk flexion.[72]

In a study of five fresh postmortem pelvises, Lavignolle and colleagues performed a spatial in-vivo analysis of the relative displacements of the iliac bones with respect to the sacrum during asymmetrical movements of the pelvis to simulate normal gait.[73] Because relative displacements were measured, there was no need to stabilize the sacrum to eliminate displacement of the lumbar spine and the lumbosacral junction. They found three different oblique axes of rotation through the pubic symphysis.[73] A phenomenon of anterior unlocking of the SIJs was observed. Evidence for this was the occurrence of forward translation on those axes, on the side of extension as that innominate moved slightly anteriorly and downward on the sacrum. The innominate on the side of flexion moved slightly upward and posteriorly on the sacrum.[73]

### *Function*

The SIJs appear to provide limited accessory motion to decrease stress on the lumbar disks, especially L5-S1. I believe that if flexion of the lumbar spine occurs without the caudal gliding of the sacrum on the ilia, compression stress of the anterior annulus of the lumbar disks would be more rapid and acute. The line of gravity is posterior to the acetabula in the normal standing posture[74,75] and causes a posterior rotation of the innominates around the acetabula,[76] which appears to enhance this caudal gliding of the sacrum with lumbar flexion (Fig. 2).[30] In the lordotic standing posture, the


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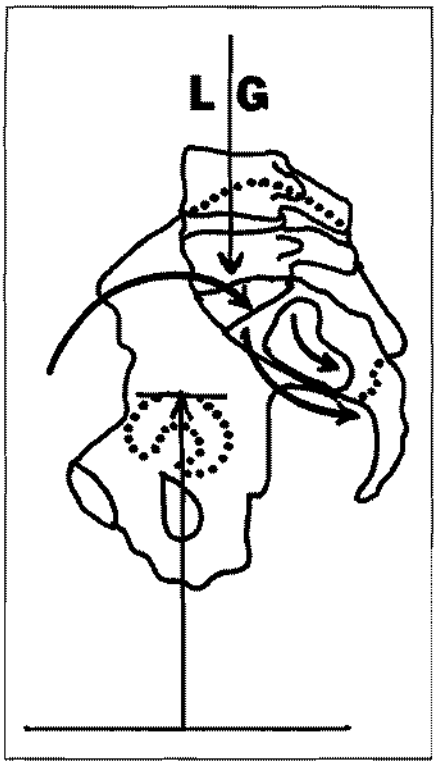


**Fig. 2.** *When the line of gravity (LG) is posterior to the acetabula, the concurrent posterior rotation of the pelvis enhances caudal gliding of the sacrum on the ilia. (Courtesy of Forum Medicum Inc.)*

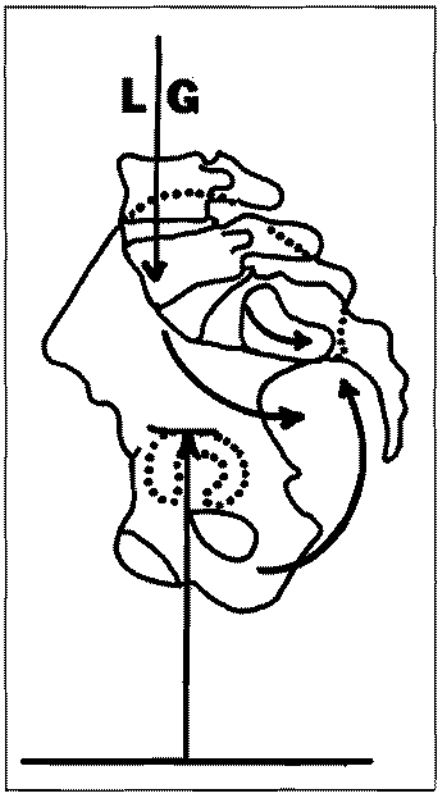


**Fig. 3.** *When the line of gravity (LG) moves anteriorly with anterior rotation of the pelvis around the acetabula, caudal gliding is impaired. (Courtesy of Forum Medicum Inc.)*

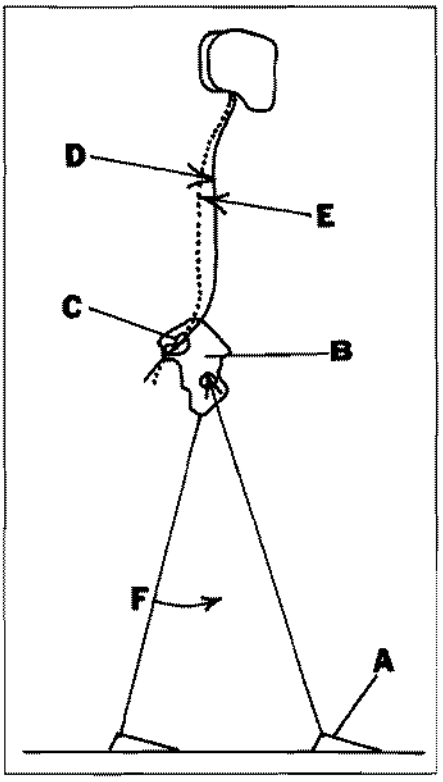


**Fig. 4.** *After initial impact of the foot during gait (A), the pelvis (B) decelerates in the sagittal plane, followed by weight-loading of the upper trunk onto the ipsilateral innominate bone, which is cushioned by the concurrent counterrotation and resilience of the sacroiliac joint (C). The pelvic deceleration slows the anterior inertial movement of the trunk (D) causing a sequential rhythmic flattening of the spinal curves from the sacrum cephalad. A posterior recovery motion (E) helps to control the anterior inertial movement and assists the hip flexors in the initiation of the next step (F). (Courtesy of Forum Medicum Inc.)*

line of gravity is displaced anterior to the center of the acetabula, increasing the pelvic angle and exaggerating the natural curves of the spine.[77] Anterior displacement of the line of gravity also occurs during lifting.[78] When the weight of the upper trunk is displaced anterior to the center of the acetabula, the anterior part of the pelvis tends to rotate downward, raising the posterior pelvis and creating a rotational force anteriorly, in extension, around the acetabula.[76] This force counters the caudal gliding of the sacrum and may limit this accessory motion (Fig. 3).[30]

Mennell noted that rotation of the trunk in the sagittal plane was limited by a loss of function in the SIJs.[71] This finding indicates that the normally functioning SIJs allow a certain increase in trunk rotation. Thus, because rotation of the lumbar spine is limited by the position of the zygapophyseal joints and because an accessory motion in the SIJs allows an increase in rotation, impairment of this accessory motion in the SIJs would increase torsional stress on the disks. One function of this accessory motion in the SIJs, therefore, is probably to decrease torsional stress on the disks with trunk rotation.[30]

In my opinion, the SIJs function subtly, but importantly and dynamically, during ambulation. Normal gait is essentially a controlled fall initiated by an anterior inclination of the trunk. This controlled fall, depending on the degree of anterior inclination of the trunk, governs the speed of the gait from a slow walk to a jog (running speed is not dependent on trunk inclination).[79] During normal gait, the ground reaction force of initial impact momentarily arrests and reverses the anterior inclination of the trunk (Fig. 4).[79,80] The trunk is never actually tilted posteriorly, but moves anteriorly and posteriorly in relation to the mean forward inclination once during each step and twice during each stride.[79] This rhythmic oscillation begins with pelvic deceleration, which causes each vertebra to decelerate in turn, and probably causes a flattening and recovery of the spinal curves with loading and unloading of the disks. Thorstensson et al found the excursion of this oscillation to be about 2 to 2.5 cm at L3 and about 1 to 1.5 cm at C7,[79] which indicates that the spinal curves may function to dampen this rhythmic sacrocranial vertebral oscillation (RSVO) as the movement at C7 was less than that at L3. This oscilla-


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tion serves to control the anterior inclination of the trunk through these brief reversals. The posterior recovery motion begins at two-point support when maximum deceleration of the trunk occurs. The hip flexors then lift the training leg, and the posterior motion of the trunk helps to propel that leg forward with the next step, further conserving the energy expended while walking.

As the trunk inclines anteriorly, the sacrum glides caudally between the innominates and is tethered by the sacrospinous and sacrotuberous ligaments. With the posterior recovery of the trunk, the sacrum glides ventrally.[81] This movement could only take place at the SIJs with the sacrum suspended from the ilia, and it must take place on an oblique axis (Fig. 5). If a dysfunction of the SIJs compromises the movement of the sacrum, then the RSVO must occur on the top of the sacrum rather than within the sacrum, increasing shear forces at the lumbosacral disk. The sacral gliding also appears to cushion impact loading of the femoral head after initial contact as the weight of the upper trunk shifts to the leading leg. In my opinion, an SIJD that compromises that gliding would probably cause an increase of impact loading to the femoral head, which could cause fractures of the trabeculae in the subchrondral bone and eventually arthritis of the hip. Because a loaded motion segment will creep or slowly deform over time and that creep is enhanced if vibration is present,[82,83] concurrent posture faults may also be exacerbated with RSVO. My speculation is that this oscillation also functions to increase nutrition of the disks.

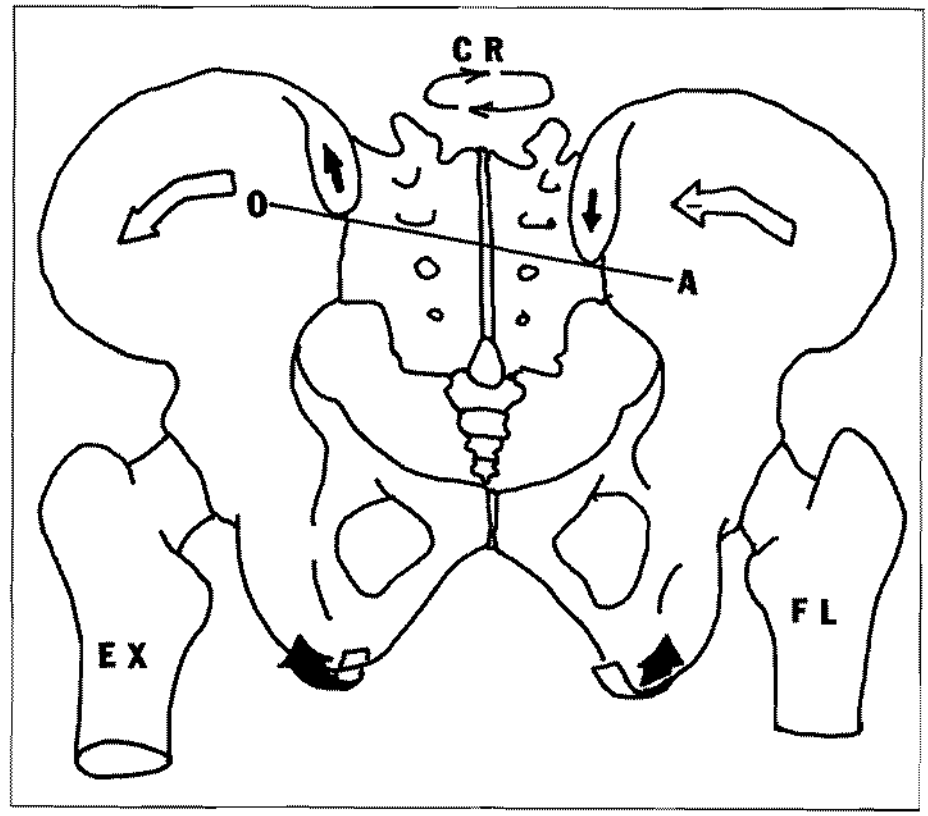


**Fig. 5.** *Posterior view of pelvis and adjacent joints. Movement of pelvis is shown at two-point support with the right leg in flexion (FL) and the left leg in extension (EX).[73] Caudal gliding of the sacrum, which occurs with rhythmic sacrocranial vertebral oscillation, begins at this point and must take place on an oblique axis (OA) if both joints are considered simultaneously. Sacral torsion in the horizontal plane from the counter-rotation (CR) of the trunk also occurs at this time. (Courtesy of Forum Medicum Inc.)*

### Evaluation

Even when a thorough examination based on palpation and movement tests is performed, the results of those tests must be appropriate to and indicative of a precise pathology. Any proposed movement dysfunction must be relevant to anatomy, function, mode of onset, or known biomechanical changes. The low reliability of clinical measurements of the SIJs[84] and unreliable test results[85] indicate, in my opinion, an inappropriate application of the tests; inappropriate interpretation of the test results; and probably a lack of basic understanding of the anatomy, function, or small and complex movements involved.

Palpation of the spine in the assessment of dysfunction is of questionable reliability. Stoddard remarked that

> In spite of the recovery of the patient, I could not detect any improvement in the position of the vertebrae in a large percentage of patients treated. . . . Positional adjustments of vertebrae did not reposition bones. . . . In a majority of cases, positional faults were unimportant and . . . what was achieved by the manipulation was an increased range of movement. . . . Palpating for boney landmarks and positions was a most unsatisfactory method of finding the osteopathic spinal lesion, and began to rely more and more on functional mobility tests.[86(pp107–108)]

Palpation may detect the pelvic obliquity that occurs with unilateral dysfunction of the SIJs, but it is of no value with bilateral dysfunction, which causes no pelvic asymmetry, nor can it be used to determine whether the asymmetry is congenital or acquired. Objective testing involving measurement of movement is "notoriously unreliable."[87]

Currently, many clinicians treating low pack pain assume that the most common type of dysfunction of the SIJs is a unilateral posterior rotation of the innominate on the sacrum.[70–72,88] Platt, although noting that the sacrum was suspended by the sacroiliac ligaments, stated that a primary anterior rotation of an innominate was not possible


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without a destruction of bony or ligamentous tissue and concluded that the primary pelvic lesion was the posterior rotation of one innominate bone on the sacrum.[63] Although the innominate does not appear capable of moving directly anteriorly on the sacrum because of the wedge shape of the sacrum, Platt overlooked the fact that many people believe the innominate can move anteriorly and downward relative to the sacrum because the sacrum narrows caudally.

McConnell and Teall described a condition in which the ilium is forward, the ischium is backward, and the "innominatum is thrown downward" on the sacrum. This causes an apparent lengthening of the limb that can be noticed by comparing the heels when the patient is positioned supine.[89] Chamberlain identified and described this downward rotation and fixation of the innominate bone on the sacrum by using stereoscopic roentgenograms and special positioning techniques. He also found that "the patient's acute sacroiliac symptoms have almost invariably been on the side of the high pubis" when the patient is positioned standing.[24] Chamberlain believed that it was highly unlikely this condition could occur bilaterally, but Swart has described bilateral anterior innominate dysfunction and its correction.[27]

If the SIJD is not properly corrected, the SIJs may become inflamed. Davis and Lentle used technetium-99m stannous pyrophosphate bone scanning with quantitative sacroiliac scintigraphy in 50 female patients with ILBPS (age range = 21–71 years, $\bar{X}$ = 39) and found that 22 patients (44%) had sacroiliitis. Eight of these patients (36%) had unilateral sacroiliitis, and 14 (64%) had bilateral sacroiliitis. Of the 22 patients with abnormal scans, 20 had normal radiographs.[90]

## Influence of Sacroiliac Joint Dysfunction on Idiopathic Low Back Pain Syndrome

From my perspective, I believe that the influence of the action of the SIJs on ILBPS, disk disease, idiopathic arthritis of the hip, scoliosis, spondylolisthesis, and the biomechanics of lifting mechanisms has been generally overlooked or ignored in the literature. Grieve concluded that "either the condition goes unrecognized or, because of authoritarian and intimidating pronouncements about its non-existence, the likelihood of the condition is not included among the many factors for assessment, and a careful comprehensive examination of the joint is not conducted."[88(p284)] Consequently, much of this discussion is presented relative to my own research and experience.

### *Pathomechanics*

Although varying considerably in severity, the common onset of SIJD occurs with an anterior shift of the line of gravity when leaning forward to perform some task (Fig. 3).[76] Because the abdominal muscles are not active in the normal standing posture,[91,92] when the weight of the upper trunk moves over the anterior pelvis, the pelvis rotates either downward anteriorly or anteriorly and obliquely around the acetabula. The severity of the dysfunction may be increased depending on the rapidity of the weight transfer and the amount of weight added to the upper trunk during lifting, bending, or lowering. I believe that, because the posterior ligaments of the sacrum are loosened when the innominates move anteriorly on the sacrum and the thin sheath of anterior sacroiliac ligaments offers only minimal protection, the SIJs are vulnerable to injury and fixation anteriorly.

My own analysis of the pathomechanics of the SIJ is that anterior rotational forces tend to rotate the innominate bones anteriorly and downward around the acetabula while the ilia lift and carry the sacrum upward, changing the relationship of the SIJs to the acetabula (Fig. 6). Because the sacrum is placed within the innominates and is wider anteriorly than posteriorly, the innominate bones rise and diverge on the sacrum where they may become fixated. Although more common bilaterally, this fixation frequently occurs unilaterally, causing a pelvic obliquity and a high iliac crest on the same side when the patient is positioned standing. This high iliac crest, high PSIS, and increased pelvic angle on the involved side may be mistaken for a posterior SIJD. With the patient positioned supine, however, the acetabulum on the involved side will move downward in relationship to the SIJs, causing an apparent lengthening of the leg on that side, which can be noticed by comparing the relative positions of the malleoli in the midline. Because the acetabula also move posteriorly relative to the SIJs with the unilateral lesion, that leg will appear to be shorter than the uninvolved leg when the patient is in the long-sitting position.[9]

In my clinical experience, the most consistent sign that confirms the lesion and corrects the dysfunction is not the apparent difference in leg length, if any, but the manner in which the leg length appears to change with mobilization of the innominates on the sacrum when the patient is positioned supine. I believe that when a suspected anterior dysfunction of the SIJ is corrected with a posterior and upward movement of the innominate on the sacrum, the anterior superior iliac spine (ASIS) moves upward and the ipsilateral PSIS will move caudally and medially on the sacrum. The leg on that side will appear to become shorter, as noted when comparing the relative leg length at the malleoli in the midline. Marking the broadest part of each medial malleolus with a felt-tipped marker makes this movement more obvious, and measurements can be made between these marks. In my experience, carefully palpating, marking, and measuring the location of the PSISs before and after mobilization will demonstrate movement medially and caudally on the sacrum of 1 to 1.5 cm or more. With bilateral dysfunction, the relative leg length is assessed by comparing the malleoli in the middle before and after flexion of each innominate on the sacrum because each leg appears to shorten with the maneuver as each side is corrected.[9,27–30]


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By correlating apparent changes in leg length with measurements of the movements of the PSISs on the sacrum before and after mobilization, I have tentatively identified what appears to be a compromise of the anterior SIJD that could be mistaken for a superior iliac shear or a posterior SIJD. In the position of anterior SIJD, the PSISs move cephalad and laterally on the sacrum as the ASISs rotate anteriorly and downward around the acetabula, indicating that the movement of the sacrum within the innominates is on a transverse axis probably just superior to the center of the SIJ. As the PSISs diverse on the wedge of the sacrum, compression of tissue, resulting in impairment of movement of the SIJ, appears to occur near the new position of the PSIS and may cause a shift in the transverse axis. I hypothesize that the divergence of the PSISs would decrease tissue pressure on the anterior aspect of the SIJs, allowing the anterior aspect of the iliac component of the SIJ to slip cephalad on the anterior aspect of the sacral surface of the SIJ and to pivot on the area of increased tissue pressure. The area of increased tissue pressure becomes a new, but abnormal, axis (Fig. 7), compromising the original anterior SIJD. This condition also could cause the leg to appear to shorten in the supine position, but to lengthen in the long-sitting position.

In my clinical practice, the mechanism of the compromised anterior SIJD is confirmed and corrected by observing the changes in apparent leg length during various maneuvers. A sharp pull on the leg on the affected side in the long axis corrects the anterior shearing and the leg appears to become longer, but the PSISs remain essentially in the same position. The joint then is in a position of anterior dysfunction, which can be corrected by rotating the innominate posteriorly and downward on the sacrum. This makes the leg appear to shorten again. I believe, however, that the PSISs will move caudally and medially on the sacrum. If an attempt at correction of this condition is made while applying sudden traction to the ipsilateral leg with the leg in about 30 degrees of abduction and 20 degrees of hip flexion, the leg will appear to become shorter rather than longer. My rationale is that the traction effort in this position causes the PSISs to move caudally and medially and the anterior innominate to move posteriorly and cephalad on the sacrum toward its resting position. I also believe that this condition can be corrected by grasping the ischial tuberosity and buttock and pulling anteriorly with one hand while pushing downward and posteriorly with the other hand placed on the superior aspect of the iliac crest. My experience indicates that for correction of SIJD—whether compromised or not—the clinician not only must rotate the anterior pelvis upward and posteriorly, but must also strive to move the posterior aspect of the innominates downward and medially on the sacrum at the same time. The patient can self-correct for this condition by flexing the hip and knee with the thigh in horizontal abduction.[30] In my opinion, correction must always be performed bilaterally.

Cibulka and associates have suggested that an SIJD creates an anterior tilt of the innominate on one side and a posterior tilt of the contralateral innominate.[93,94] Their data may also be interpreted in terms of a unilateral anterior SIJD, which also causes a high iliac crest, a high PSIS, and an increased pelvic angle on the same side. The unilateral anterior SIJD can be simply demonstrated by comparing the relative leg length with the patient positioned supine. If the leg

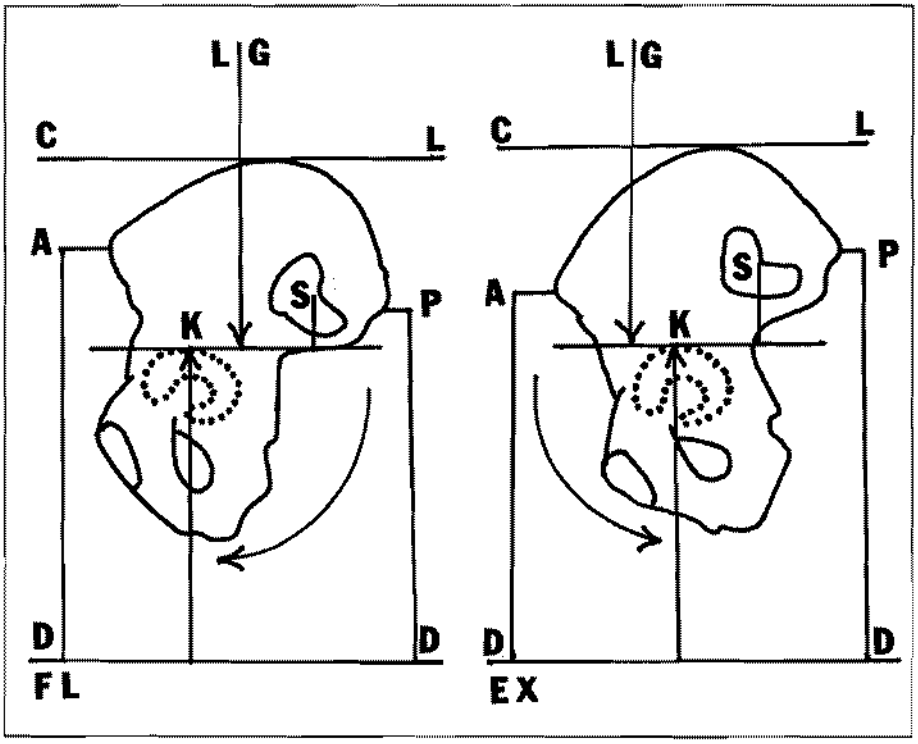


**Fig. 6.** *In the flexion position (FL), the line of gravity (LG) is posterior to the acetabula and causes a posterior rotational force around the acetabula. As the line of gravity moves anteriorly to the acetabula in the extension position (EX), the pelvis rotates anteriorly around the acetabula. The top of the acetabula (K) to the base (DD) remains constant. Although the height of the posterior superior iliac spine (PD) and of the anterior superior iliac spine (AD) changes considerably, the level of the crests of the ilia (CL) may not change much. As the level of the sacroiliac joint (S) rises, apparent leg length is increased in both supine and standing positions. The horizontal distance from K to S becomes shorter; thus, in the patient with sacroiliac joint dysfunction, leg length may be shorter during sitting, but longer when positioned supine. (Courtesy of Forum Medicum Inc.)*


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on the side of the high iliac crest is longer than the other leg when the patient is positioned supine, then the dysfunction was anterior. Flexion of that innominate on the sacrum will cause that leg to appear to shorten and will cause the iliac crests to be level when the patient is positioned standing.[9,28–30]

Although an anterior tilt of the innominate on one side and a posterior tilt of the innominate on the other side occur with the asymmetrical movement of locomotion,[73] I believe there are several reasons why these movements probably do not occur with dysfunction: 1) The common mode of onset occurs when both innominates are moving directly anteriorly and downward or obliquely anteriorly and downward around the acetabula (after which, one side may become compromised); 2) for the innominates to rotate anteriorly enough on one side and posteriorly enough on the other side so that both innominates are dysfunctional would appear to cause more torsion at the pubic symphysis than is structurally available; and 3) because the innominates normally move posteriorly on the sacrum against the strong posterior ligaments and excessive motion posteriorly is effectively blocked,[64] it is unlikely that a posterior dysfunction of the SIJs exists. Unequal heights of the PSISs with concurrent inequality of the innominate tilt can exist in various degrees of SIJD.

### Effect on Passive Straight Leg Raising

Bohannon et al found a constant relationship among PSLR, pelvic rotation, and the pelvic angle. Pelvic rotation occurred in every subject by 9 degrees of PSLR and usually before 4 degrees of PSLR.[95] Thus, any restriction in pelvic rotation caused by an SIJD might affect the PSLR test. Dysfunction on the SIJs can also cause hamstring muscle strain[94] and sciatic pain.[96] I believe, therefore, that PSLR can be used effectively to help assess this dysfunction. Passive straight leg raising may increase pain in an inflamed sciatic nerve, while the simultaneous pull of the hamstring muscles on the innominate bone can cause a posterior rotation force that may ease the pain of an anterior SIJD. Mennell noted that, as the ipsilateral innominate bone moves posteriorly with PSLR, the sacrum is carried posteriorly on the opposite innominate, and contralateral pain in the low back is indicative of an anterior SIJD on the contralateral side.[70] Theoretically, PSLR will tend to slacken the fibers of the contralateral posterior sacroiliac and iliolumbar ligaments. Passive straight leg raising may cause ipsilateral pain in the low back when lowering the leg if the patient is holding back and actively assisting in lowering the leg because the pull of the iliacus muscle tends to move the ipsilateral innominate anteriorly on the sacrum, increasing the pain of an anterior SIJD. This movement may give the impression of pain in the iliacus; however, if the patient stabilizes the anterior pelvis by raising the head and shoulders to contract the abdominal muscles and is then able to lower the leg without discomfort, the source of the pain is the SIJ and not muscle.

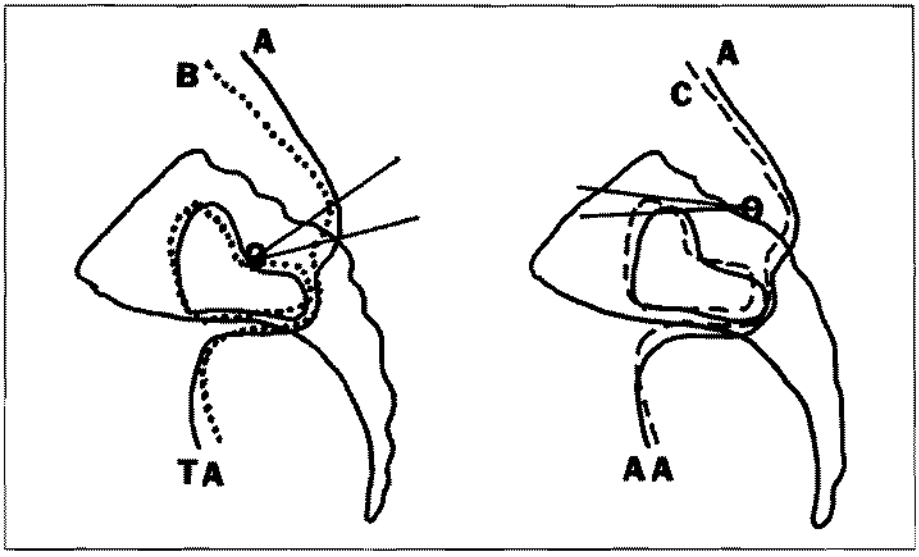


**Fig. 7** *(Diagram TA). In the patient with sacroiliac joint dysfunction, the innominate(s) move on a transverse axis (O) relative to the resting position (A) (solid line) to the position of dysfunction (B) (dotted line). The cephalad and lateral movement of the posterior superior iliac spine (PSIS) on the sacrum spreads the joint, increasing tissue pressure near the PSIS. (Diagram AA) The PSIS then becomes an alternate axis of rotation (O), which may allow the innominate(s) to shift to a second position (C) (dashed line). This shift of the innominate(s) makes correction in flexion somewhat more difficult. (Courtesy of Forum Medicum Inc.)*

Passive straight leg raising may also cause ipsilateral pain in the low back, which I have found indicates the anterior sacral shear that compromises an anterior SIJD.[30]

### Effect on Gait

I believe that SIJ motion plays a critical role in gait, and the following represents my interpretation of events that occur during walking. When the pelvis rotates anteriorly in the horizontal plane, the ipsilateral trochanter is positioned posteriorly and is closely packed to the pelvis with initial impact (Fig. 8A). With ipsilateral extension, the contralateral pelvis rotates anteriorly in the horizontal plane as the ipsilateral trochanter swings anteriorly (Fig. 8C). This movement is controlled by the decelerating force applied by an eccentric contraction of the gluteus maximus muscle, which serves to decrease impact loading of the femoral head. Counterrotation of the upper trunk also serves to help decrease this impact loading on the ipsilateral side as these forces are dissipated in the


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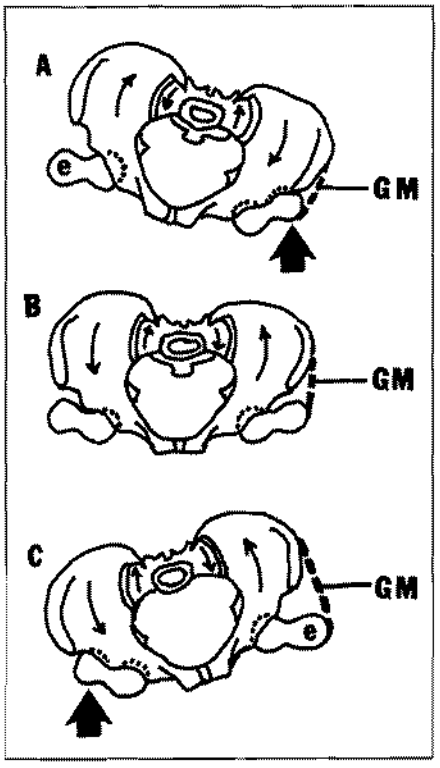


**Fig. 8** *With heel-strike on the left side (A), the trochanter is posterior toward the innominate, the right leg is in extension (e), and the right trochanter is more anterior to the innominate (two-point support). During mid-swing with weight bearing on the left side (B), the gluteus maximus muscle (GM) is elongating. With heel-strike on the right side (C), the right trochanter is posterior toward the innominate, the left leg is in extension, and the gluteus maximus muscle completes deceleration. (Courtesy of Forum Medicum Inc.)*

dynamic elasticity of the posterior ligaments of the SIJs. This counterrotation also probably functions to protect the lumbar disks from torsion shear, which would appear to be markedly increased without this deceleration and dissipation. In support of this hypothesis, Ducroquet et al noted a decrease of pelvic rotation in the horizontal plane in patients with SIJD.[97]

### Effect on Regional Musculature

I have observed in my patients that pain in the buttocks is commonly associated with low back pain, but is not necessarily referred pain or pain in the sciatic nerve. I hypothesize that because the gluteus maximus muscle originates from both the sacrum and the ilium, when the innominate moves anteriorly on the sacrum with anterior SIJD, the conjoint origin is separated, thus also separating the fibers of the gluteus maximus muscle on a line from just inferior to the PSISs to the greater trochanter. A movement of 1 to 1.5 cm cephalad and laterally of the PSISs on the sacrum is not unusual, and I have measured as much as 2.5 cm with correction in a young woman who suffered acute low back pain after a fall.[30]

Bogduk demonstrated that the large mass of spinal extensor musculature has a relatively small origin, principally on the posterior surface of the sacrum.[98] McGill found large forces generated within the large low back extensors, which were transmitted to the aponeurosis and directly to the sacroiliac region, suggesting that connective tissue microtrauma is a factor in clinically observed sacroiliac trauma and pain.[99] He also suggested that microtrauma is often accompanied by a spasm of the extensor muscles. I believe the spinal extensor musculature must play a substantial part in the regular recovery of the spine during RSVO, and if movement of the SIJs is impaired by dysfunction, stresses on the aponeurosis would be increased. Pain in these muscles may be a secondary effect. In comparing electromyographic activity in the lumbar paraspinal muscles of subjects with and without chronic low back pain, Miller found similar levels of integrated EMG activity in both groups of subjects while they performed various tasks. No evidence was found to demonstrate the presence of the reflex-spasm cycle in the subjects with chronic low back pain.[100]

### Associated Abdominal Pain

Occasionally, associated pain will occur in the abdomen at Baer's sacroiliac point, which has been described as being 2 in* from the umbilicus on a line from the umbilicus to the ASIS.[70(p90),72(p24)] Theoretically, torsional strain on the SIJ can modify tenderness at this point, and mobilization of the SIJs can relieve it.[29(p142),79(p152)] Norman injected a local anesthetic into the SIJs and successfully relieved the lower abdominal pain.[101] Wilson called attention to the fact that "unusual radiation of pain from the lower three lumbar vertebral joints has led to the unnecessary removal of pelvic organs in the female and to coccygectomy."[102]

### Association with Pregnancy and Menstruation

Grieve found that sacroiliac strains sometimes follow gynecological and obstetrical operations.[103] Ligamentous laxity accompanies pregnancy in the last trimester[104,105] and to a lesser degree during menstruation and menopause.[105] Postmortem specimens in various stages of pregnancy clearly showed that the increased range of movement is easily recognizable by the fourth month and that at full term the range increased by about 2½ times. In one subject, the anterior margins of the joint could be separated by almost 2 cm.[67] Movement abnormalities of the SIJs and pubic joints are a common cause of persistent postpartum pain, and simple mobilizing techniques localized to the SIJs are very effective in alleviating this pain.[88(p283)]

### Effect on Nerves and Treatment by Injection

An anterior and downward rotation of the innominates on the sacrum may stretch the spinal nerve roots, which in turn may cause neurological changes[57] or lancinating pain.[59] Norman and May treated over 300 patients with injection of local anesthetic into the SIJs, relieving pain immediately in patients who had both sensory changes and an absent Achil-

*1 in = 2.54 cm.


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les reflex.[106] Therapeutic results were obtained by adding hydrocortisone to the anesthetic. Several patients with continuing low back pain following one or two laminectomies were successfully treated by three or four injections.[106] Ray also reported good relief from ILBPS by performing facet nerve blocks and sacroiliac desensitization with needle rhizotomies.[107] In a double-blind study, Ongley and associates successfully treated chronic low back pain and ligamentous insufficiency with manipulation of the SIJs, flexion exercises, and sclerosant injections into the adjacent ligaments to proliferate the production of connective tissue.[108]

### *Pain Production that May Mimic an Increase in Intradiskal Pressure*

Low back pain is frequently precipitated or exacerbated while leaning forward concurrent with an increase in intradiskal pressure[11]; however, if the pain is secondary to SIJD, it can be prevented or relieved by supporting the anterior pelvis with the abdominal muscles prior to leaning forward. Coughing or straining may cause or increase the pain of SIJD. An increase of intra-abdominal pressure probably spreads the innominate bones on the sacrum, stretching the painful tissues even further.[9] If stabilization of the SIJs by lateral compression of the innominate bones (after correction of the SIJD) does not alleviate pain during coughing or straining, then the coughing or straining may be related to an increase in intradiskal pressure or to an injury to the muscles involved in forced expiration.

Mennell described how pain on sitting may arise from the SIJs through a combination of unequal weight distribution and rotation when the ischial tuberosity is fixated, causing an anterior torsional strain on the SIJ.[30(p130)] I have found that pain on sitting caused by SIJD can be differentiated from pain caused by the increase in intradiskal pressure by placing a pad about 2 to 3 cm thick under the upper thighs, just anterior to the ischial tuberosities.[30] This technique lifts the anterior pelvis, causing a corrective force in flexion, and relieves the pain of an anterior SIJD.

### *Effect on Laterally Deviated Spine*

In my clinical practice, I have found that proper mobilization of the SIJs can provide a rapid correction of the laterally deviated lumbar spine and immediate relief of acute low back pain without the use of traction, lateral shift, or other mobilization of the lumbar spine. The laterally deviated spine has been thought to be a shift of the nucleus pulposus within a presumably intact annulus fibrosus,[88(p154)] but may actually be a shift in the sacral dynamics in patients with SIJD.

## Precautions

In my opinion, mobilization of the SIJs may be precise and appropriate. I believe that, because the normally functioning SIJs allow compensatory rotation for the lumbar spine and an SIJD impairs that rotation, a correction of SIJD is possible by forcing a rotation with manipulation.[94,109,110] This technique, however, does not provide a consistent method of determining when a complete correction is made, the joint may be caused to wedge more tightly or to become unstable, and torsional shear of the lumbar disks may be increased. Bragard warns that tearing or rupturing of the articular capsule and its ligaments often results from unphysiologic forced movement of the SIJs.[111] With intervertebral disks under torsion, microscopic failure in the annulus fibrosus begins just before 3 degrees of rotation.[52]

## Summary and Conclusions

I believe a relatively uncomplicated biomechanical dysfunction of the SIJs is consistent with the nature of most ILBPSs. In my view, this dysfunction may cause acute or chronic low back pain and, if compromised, give the appearance of a posterior dysfunction of the SIJs or a superior iliac shear. It can also mimic disk disease by causing pain on leaning forward, pain on sitting, and pain with coughing or sneezing. My clinical observations lead me to conclude that secondary effects of the dysfunction are numerous and varied and give the appearance of a multifactorial etiology.

I have presented an argument that impaired function of the SIJs may alter gait, cause pain with ambulation, and increase shear forces on the disk, and it may exacerbate a spondylolisthesis or cause an unstable segment. If SIJD is left untreated or is treated inappropriately, the patient may eventually lose his or her job and family support and become socially isolated. In my opinion, lack of recognition of SIJD has caused inappropriate testing, inappropriate interpretation of test results, inappropriate treatment, inappropriate research, and development of inappropriate technology. Future research on low back pain should include SIJD as a major contributing factor.


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# Commentary

The "Old Coot Syndrome" is a phrase appearing in the July 1979 issue of *Military Medicine*. The term means that, throughout history, the major resistance to medical progress has been the pressures of medical authority; we quickly learn to cram our clinical experiences into sanctioned molds. Now that the physical therapy profession has evolved into a pillar of the medical community, we are even more at risk for the Old Coot Syndrome. DonTigny presents an intriguing hypothesis that conflicts with our own clinical observations. Because of this discrepancy, we believe his article deserves to be read carefully, lest we pass a mirror and see old coots looking back.

The author hypothesizes that idiopathic low back pain syndrome (ILBPS) stems from a unifactorial, biomechanical lesion: anterior dysfunction of the sacroiliac joint (SIJ). He states, "If each affected structure is examined individually without consideration of the basic lesion, it may give the appearance of a multifactorial etiology." His article contains several areas of controversy that must be addressed.



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**Anterior Dysfunction of the Sacroiliac Joint as a Major Factor in the Etiology of Idiopathic Low Back Pain Syndrome**

Richard L DonTigny

*PHYS THER.* 1990; 70:250-262.

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