2019

Changing the narrative in diagnosis and management of pain in the sacroiliac joint area

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This article was originally published as:
Palsson, T. S., Gibson, W., Darlow, B., Bunzli, S., Lehman, G., Rabey, M., Moloney, N., Vaegter, H. B., Bagg, M. K., & Travers, M. (2019). Changing the narrative in diagnosis and management of pain in the sacroiliac joint area. *Physical Therapy, Early View, Online First*.

Original article available here:
https://doi.org/10.1093/ptj/pzz108

TITLE: Changing the Narrative in Diagnosis and Management of Pain in the Sacroiliac Joint Area

RUNNING HEAD: Changing the Narrative for Sacroiliac Joint Pain

TOC CATEGORY: Musculoskeletal

ARTICLE TYPE: Perspective

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KEYWORDS: Lumbosacral Region, Musculoskeletal Pain, Pain Management, Patient Care, Patient Education, Pelvic Girdle Pain

ACCEPTED: March 10, 2019

SUBMITTED: July 24, 2018

The sacroiliac joint (SIJ) is often considered to be involved when people present for care with low back pain where the sacroiliac joint (SIJ) is located. However, determining why the pain has arisen can be challenging, especially in the absence of a specific cause such as pregnancy, disease, or trauma, where the SIJ may be identified as a source of symptoms with the help of manual clinical tests. Nonspecific SIJ-related pain is commonly suggested to be causally associated with movement problems in the sacroiliac joint(s); a diagnosis traditionally derived from manual assessment of movements of the SIJ complex. Management choices often consist of patient education, manual treatment, and exercise. Although some elements of management are consistent with guidelines, this perspective argues that the assumptions on which these diagnoses and treatments are based are problematic, particularly if they reinforce unhelpful, pathoanatomical beliefs. This article reviews the evidence regarding the clinical detection and diagnosis of SIJ movement dysfunction. In particular, it questions the continued use of assessing movement dysfunction despite mounting evidence undermining the biological plausibility and subsequent treatment paradigms based on such diagnoses. Clinicians are encouraged to align their assessment methods and explanatory models to contemporary science to reduce the risk of their diagnoses and choice of intervention negatively affecting clinical outcomes.

Low back pain (LBP) is the leading cause of disability worldwide.[1] A significant proportion (16%–35%) of these presentations are thought to involve the sacroiliac joint (SIJ) complex.[2-5] The 3 broad categories of SIJ pain are[6] pregnancy-related SIJ pain, specific pathology of the SIJ (eg, spondyloarthropathy or fracture), and SIJ-related pain of other origin.[7, 8] Pain of unknown or nonspecific onset is the focus of this article and will collectively be referred to as nonspecific SIJ-related pain. When a patient seeks care because of pain in the low back, pelvic girdle region, or both, the role of the health care professional is to perform a thorough examination that considers diagnoses of specific pathology, screens for risk of pain persistence, and directs appropriate care.[9]

Traditionally, the SIJ has been considered as part of the diagnostic triage for LBP with clinicians seeking to draw distinction between LBP with or without SIJ involvement. The involvement of the SIJ in low back pain has been simplified into a role as a local source of nociception or as a dysfunctional biomechanical junction (with either too little or too much movement occurring), either becoming painful itself or driving symptoms elsewhere, eg, the lumbar spine.[10] Thus, clinicians have sought to rule in or rule out the SIJ as a nociceptive source and/or implicate SIJ movement dysfunction as the cause for local and/or remote symptoms.

It is important to draw a distinction between SIJ related pain and what is considered to be SIJ movement dysfunction as the use of overlapping terminology may result in confusion.[10] The SIJ can be inferred as a source of local nociception using well-documented pain provocation tests.[11] However, the outcome of these tests does not inform the clinician why the structures are sensitive. Frequently, movement dysfunction of the SIJ is credited with being a driver of increased local tissue sensitivity and subsequent symptoms. However, the biological plausibility of reaching such conclusions based on movement detection and palpation of the SIJ have been questioned for more than 10 years.[10, 12] Nonetheless, this concept and the associated tests are still taught on clinical curricula and are

widespread in clinical practice throughout the world.[13] Thus, the present article aims to review this important topic within the context of current knowledge. Drawing parallels from the LBP literature, the potential unhelpful consequences of diagnostic and management narratives that communicate movement dysfunction as the cause or contributor toward pain will be considered. On the basis of this information, we offer recommendations for practice that align with current evidence.

[H1] Implying SIJ involvement

[H2]Local Tissue Sensitivity at the SIJ

During assessment, pain provocation tests can diagnose the SIJ as a source of local sensitivity.[11] Here, the examiner manually applies mechanical stress either directly to the pelvic girdle (eg, sacral thrust, gapping- and compression tests) or indirectly through the hip, causing a shearing stress (thigh thrust test) or torsion (Gaenslen test) in the SIJ. A more detailed description of each of these tests can be seen in Laslett et al.[11] These useful clinical tools demonstrate good diagnostic validity and are able to discriminate SIJ-related pain[11, 14, 15] from other potential nociceptive sources such as the lower back or the hip. On the basis of the outcome of these tests, clinicians can appropriately interpret positive pain-provocation tests as indicative of an increase in SIJ tissue sensitivity.

SIJ provocation tests do not however provide the clinician with insight as to why these structures are sensitive and are therefore incapable of confirming too little or too great movement. However, mechanical sensitivity is merely 1 component of the pain experience which is produced by the brain in response to perceived threat to body tissue.[16] The evaluation of threat by the brain is a complex process that is not fully understood. However, there is clear evidence this evaluation integrates information from multiple domains, including peripheral nociception.[17] There are many highly innervated components of the SIJ including joint capsule, ligaments and subchondral bone.[18-20] A more

detailed description of the anatomical construct of the pelvic girdle can be found in Vleeming et al.[21] The mechanical stress of these provocation tests may therefore induce symptoms/be familiar to the patient's reports. Nociceptive information from peripheral tissues is important in threat assessment and the subsequent experience of pain.[22] It is sensible to consider the SIJ as a potential source of nociception in light of the rich supply of neural fibers with nociceptive abilities[18, 19] and that these can be involved in the experience of pain.[23, 24] Trauma or other aberrant loading to the intra- and/or extraarticular joint structures[7, 8, 25] or a direct, chemical stimulation[26, 27] is highly likely to stimulate nociceptive fibers. If the SIJ is diagnosed as a source of nociception, further clinical examination is often undertaken with the goal of establishing specific underlying movement dysfunctions of the SIJ. This dysfunction is then suggested as a means of explaining the local sensitization. The assessment of SIJ movement dysfunction is typically done via clinical tests involving movement detection and palpation.[28-30] Consequentially, the diagnostic and therapeutic narrative that is communicated to patients may become extended from mechanical sensitivity at the SIJ to their symptoms being attributable to a specific movement dysfunction of the SIJ. However, coexistence of signs (as determined by SIJ pain provocation tests) and symptoms, and a hypothesized movement dysfunction does not mean these are causally related. In fact, experimental data suggest that the number of positive pain provocation tests is related to pain sensitivity and verbal reports of pain intensity in the SIJ region.[27] In addition, although the sensitivity of SIJ tissues may be validly assessed, determining the presence of a movement dysfunction is considerably more speculative as outlined below.

[H2]Explaining SIJ Pain as a Consequence of SIJ Movement Dysfunction: Is This Plausible?

Purported SIJ movement dysfunctions often have labels such as structural weakness, asymmetry, instability, stiffness or positional faults (eg, torsion, upslip, or downslip) of the joint(s) or associated structures. In this article, these labels are collectively referred to as "movement dysfunctions."

The accuracy of this clinical reasoning process is dependent on biological plausibility and clinical test validity. Specifically, accurate reasoning requires that current knowledge (of the anatomy and biomechanics of the SIJ and the neurobiology of pain) allows for detection and then inference of a causal relationship between the movement dysfunction and the pain. However, this relationship is not substantiated by current knowledge. The SIJ is an inherently stable structure, where very small movement available occurs in 6 degrees of freedom during normal activities. Several features of the articular configuration limit movement to a few degrees of rotation (at most) about a transverse axis.[31, 32] With the sacrum being wedged between the innominate bones, there is reciprocal congruency of irregular articular surfaces and a complex network of intra and extra-articular ligaments. Further contributions to joint stability are provided by gravitational loading upon the sacrum and musculotendinous forces that span the joint (see Vleeming et al[21] for review). With this in mind, it is interesting that movement dysfunction is often thought to indicate a lack of stability. Multiple clinical tests have been described to identify movement dysfunction (see van der Wurff et al[33] for review). However, evidence has been mounting for more than a decade challenging the plausibility of these tests to diagnose a purported movement dysfunction of the SIJ (see Laslett[10] for review). Criticisms include issues such as relying on clinicians manually detecting movements of the SIJ through multiple layers of tissue[34] and that the movements are so small that external detection by manual methods is virtually impossible.[35] The amount of SIJ movement (rotation and translation) has been investigated using radiostereometric analysis; a highly accurate, reliable and appropriate method for 3-dimensional measurement of small articular movements.[36] The Table presents an overview of findings from

radiostereometric analysis studies investigating the articular movements of the SIJ during tests intended to diagnose a movement dysfunction in people with SIJ or pelvic girdle pain. For example, during the standing hip flexion test/Gillet test, SIJ rotation with a mean of 0.2 (SD = 0.5) degrees was observed.[35] Furthermore, the direction of rotations were variable and mean translations were minute (mean = 0.3 [SD = 0.2] mm). Additionally, modelling of posterior superior iliac spine displacement during the standing hip flexion test suggests that the posterior superior iliac spine may move <0.2 mm on the stance side.[37] These very small movements have also been demonstrated recently in vitro.[38] A more recent study has demonstrated equally small SIJ movements during the active straight-leg raise test,[39] which suggests that gravitational deloading does not cause changes in SIJ movement. Despite movements of the SIJ during clinical testing being minute, it has been suggested that clinicians can detect this SIJ motion.[40] However, given the inherent perceptual difficulty in detecting such tiny movement, it is likely that any perception of movement may be attributable to other factors such as soft tissue motion[34] or pain-associated muscle activation[26] as a response to nociceptive activity (see Arendt-Nielsen and Graven-Nielsen[41] for review).

Given these challenges in detection of movement, it is not surprising that tests for movement dysfunction are not reliable. This appears to be the case independent of level of clinical experience[10, 42] or training[43] of the assessor. Furthermore, movement dysfunction tests require accurate identification of relevant anatomic landmarks and assessment of their symmetry and motion during testing. Here, interexaminer agreement (kappa) for identifying the anterior superior iliac spine (Cohen $\kappa = 0.24$) and the posterior superior iliac spine (Cohen $\kappa = 0.08$)[43] has been shown to be only slight to fair.[44] Thus, it seems that these bony landmarks cannot be identified accurately. These data, combined with the data

158 on movement magnitude render tests for detecting motion or position of the SIJ unusable[33, 45] for the
159 valid detection of SIJ movement.
160 We therefore suggest that although clinicians commonly seek to identify movement
161 dysfunctions on the basis of such tests, the weight of evidence has not changed in the last decade and
162 the use of these tests and models of movement dysfunction testing of the SIJ remain unsupported.
163 SIJ-related pain, similar to LBP is multidimensional in nature and there is little evidence to
164 support either the successful identification of, or intervention upon, SIJ movement dysfunctions in the
165 management of this condition.[12, 33] Establishing causality in a clinical setting is extremely difficult. In
166 this case, directly attributing SIJ-related pain to movement dysfunctions causing increased peripheral
167 nociceptive input from SIJ tissues, is a flaw in reasoning; mistaking association for causality. Positive
168 pain provocation tests are likely indicative of increased sensitivity of the tissues,[11] which might to some
169 degree be subsequent to tissue loading. However, this is a reductionist, linear interpretation of the pain
170 experience. The inadequacy of this reasoning is highlighted by recent trials of SIJ denervation
171 procedures outlined below.
172
173 **[H2]Evidence That Nociceptive Activity From the SIJ Contributes to Pain**
174 Pain in general is not a simple tissue-based stimulus response,[22, 46-48] and the emergent pain experience
175 can be described as a response to a sense of threat to the body.[22, 47, 48] Thus, understanding and
176 managing pain are contingent on identifying contributors to an individual's sense of threat.[16] Clearly, it
177 is reasonable to suggest that nociceptive input from SIJ area tissues[23, 24] may contribute to threat
178 perception.[22, 47]
179 Radiofrequency denervation is arguably the intervention most likely to abolish nociception[49] as
180 it is aimed at preventing conduction of nociceptive impulses by ablating the nerves involved.[50] Studies

using this method have shown it to be effective in reducing SIJ pain[51, 52] but not abolishing the pain entirely. Moreover, Juch et al[53] evaluated the effect of radiofrequency denervation of the SIJ in addition to exercise rehabilitation. No clinically important difference was observed in the primary outcome (pain intensity at 3 months after intervention) with the addition of radiofrequency denervation. Together, these data do show that nociceptive activity in and around the SIJ may contribute significantly to SIJ-related pain but this peripheral nociception is not the sole cause of the pain experience. In fact, nociception from the SIJ and surrounding structures appears to represent only a part of a complex, multidimensional experience of pain,[49] suggesting that supraspinal processing of afferent input (nociceptive and nonnociceptive) and other modulatory factors plays an important role in the pain experience.[54] This list of factors contributing to such modulation likely includes internally held beliefs and knowledge regarding fragility/structural integrity/robustness of the area. The above arguments have important implications for clinicians and clinical practice.

[H1] Implications for Clinical Practice

[H2]Should We Dispense With Movement Dysfunction Models for the SIJ?

The purpose of this article is not simply to present a contrarian perspective to a commonly held management paradigm. Rather, the purpose is to highlight the fundamental flaws and potential consequences of explaining SIJ pain through a movement dysfunction lens. If clinical decisions are based on a construct that lacks plausibility and clinical tests lacking in validity and reliability, the entire management paradigm must be questioned. Dispensing with the use of tests for movement dysfunction and associated diagnoses would be consistent with a contemporary understanding of the biomechanics of the SIJ, clinicians' ability to assess this reliably, as well as reflecting the current knowledge underpinning pain perception. As pain is a response to credible threat perception,[16, 55] factors that

increase threat perception are likely to be unhelpful. Changes in motor planning seem to occur immediately after the onset of acute low back[56] and pelvic girdle pain[26] and may therefore be a natural response to the perception of threat. Such changes seem to be influenced by cognitive factors such as fear of movement[57] but the failure of reversing these beliefs may result in unfavorable loading and thereby the maintenance of pain (see Hodges and Moseley[58]).

Pain associated with sensitized SIJ-related structures can be diagnosed accurately with high levels of sensitivity (94%) and specificity (78%).[11] However, unsubstantiated pathoanatomical explanatory models such as structural weakness, abnormality or instability may undermine a person's perception of the reversibility of symptoms and promote movement-related fear. More importantly, such pathoanatomical explanatory models may undermine the person's perception of reversibility of symptoms by promoting movement-related fear through unintended reinforcement of perceptions of threat and damage in people with pain.[59-61] With nonspecific SIJ-related pain, this raises an important issue as labels of SIJ movement dysfunctions are not biologically plausible nor verifiable with valid or reliable clinical tests. We argue that clinicians must consider the potentially harmful effect of implicit and explicit messages of fragility that they deliver through assessment and management based on a movement dysfunction paradigm.

[H2]Mind One's Words: Avoid Communicating Fragility Messages

Patients' beliefs, particularly their understanding of the cause and nature of their pain are considered increasingly important features of the pain experience[59, 62-64] and may influence the pathway to pain persistence.[65, 66] For example, in LBP patients those who received radiology report findings had poorer outcomes than a control group who did not receive such pathoanatomical information.[67] When an individual experiences pain (in their SIJ area or elsewhere), typically there is an attempt to make sense

of the pain; often this is done by forming a representation of it[68] on the basis of 5 key belief dimensions. A representation of SIJ-related pain based on pathoanatomical beliefs might look like the following:

1. Identity beliefs describing explanatory and prognostic labels: “I have an unstable pelvis”[12]
2. Beliefs about potential causes: “I have pelvic pain because I have a weak core”[12]
3. Beliefs about consequences “My pelvis goes out of place”[12]
4. Beliefs about perceived self-control over pain: “I should stop when I feel any pain”[12]
5. Expectations of how long the pain will last: “I will always have a weakness now so I must be careful”[12]

These beliefs are informed by society around the individual (ie, from observing the experiences of others and the media) as a biomedically based explanations for pain are overwhelmingly prevalent in society.[69] Therefore, people may present for treatment with these unhelpful biomedical and pathoanatomical beliefs already well established. However, evidence suggests that health care professionals may play a dominant role in the development/reinforcement of these beliefs.[66] For example, a recent study demonstrated that most people view their persisting spinal pain as being driven by structures that are “physically defective”[70] and worryingly, the majority (89%) of the 130 participants indicated they learned this from health professionals. Although clinicians may not intend to frame messages in this way, these data demonstrate that unhelpful pathoanatomical models can be (mis)interpreted and remembered by patients.

Pain beliefs inform coping behavior,[68] and perceptions of fragility based on pathoanatomical explanations are linked to an avoidance behavior that, in turn. can sustain pain and disability in a fear avoidance cycle.[62] Thus, clinicians need to carefully consider the influence that pathoanatomical explanations and labels suggesting structural weakness, abnormality or instability have on patient

beliefs, behaviors and emotional responses.[61, 64, 66, 71] Believing that one is unable to hold the body together because of a lack of "core stability"[60, 66] and an innominate bone that "slips out of place" is likely to give rise to guarding and avoidance behaviors, which can themselves sustain pain[25]; in addition, such catastrophic beliefs are highly distressing.[25] A recent, longitudinal observational study (n = 2891) found that emotional distress in the acute stages of low back pain increased the number of subsequent primary care consultations.[72] Thus, not only on an individual level, but also from a health care service delivery point of view, it is essential that we move away from the use of nonplausible, pathoanatomical diagnoses and explanations that may drive perceived threat and distress.

[H2]Helping Patients Make Sense of Their Pain

It is both a challenge and a duty of contemporary clinical practice to avoid reinforcing negative beliefs either explicitly (by explanations eg, "Your pelvis is unstable") or implicitly (by treatment choices, eg, "You need to build up your core muscles"). Rather, clinicians should seek to provide explanations that help the patient re-conceptualize the pain experience by addressing key belief dimensions.[73] For example, patients presenting with positive pain provocation tests of the SIJ may have it explained to them that their spine is a strong structure and that the pain they are experiencing is due to increased sensitivity of the SIJ structures (identity beliefs). A multitude of factors may influence the sensitivity of the SIJ structures such as the adoption of provocative movement behaviors, fear or vigilance (cause beliefs) which may sustain pain and disability (consequence beliefs). Strategies to address these mechanisms, eg, movement control or cognitive reframing (control/treatment beliefs) may enhance their functional capacity with pain control using short- and long-term goals (timeline beliefs).

[H2]Aligning Treatment Rationale With Explanation of Pain

For all types of spinal pain, there exist different treatment approaches with various degrees of effectiveness. It appears that treatment choice commonly depends on clinician preferences, independent of whether these are supported by contemporary guidelines.[30] A further concern is that interventions predicated on addressing movement dysfunction may contradict one another, which results in mixed messaging to the person with SIJ pain. For example, manual interventions purporting to increase the movement of the SIJ seem incongruous with prescribing home-exercises focusing on increasing "stability" (often prescribed for people with pelvic girdle pain).[30] Aligning the rationale for treatment with current evidence is likely to provide a more consistent message.

Using manual therapy (when indicated) may be explained using known neurophysiological mechanisms including activation of endogenous descending inhibition, changes in the neurobiological milieu in the periphery and changes in muscle activity.[74] Such explanations may be placed in the context of empowering the patient to move and engage in an active and mutually determined rehabilitation process. This may address the domains outlined above (labeling, cause, control, consequences and timeline) more constructively than other inaccurate models relating to movement (increased/decreased) or correcting joint position. On a similar note, an alternative rationale for the prescription of exercise, as opposed to arguing for stability changes in the SIJ, can be consistent with the idea that pain is multidimensional and is more indicative of sensitivity as opposed to damage or joint dysfunction. Therefore, the rationale to patients for using exercise could include an explanation of how sensitive tissues respond well to physical load (as seen in eg, the management of knee and hip osteoarthritis[75, 76]). This is likely due to the involvement of endogenous pain inhibitory systems.[77]

The purpose of this article is not to advocate for or against any given treatment approach or modality. Rather, using nonspecific SIJ-related pain as a model, we encourage clinicians to do the following:

1. Explain how pain works tailored to the individual presentation (encompassing a biopsychosocial model)
2. Constructively address unhelpful/aberrant health beliefs
3. Promote reassurance regarding structural integrity of the pelvis/SIJ
4. Design and discuss a management plan that is aligned with points 1 to 3

[H1]Conclusion

Sacroiliac joint movements during clinical testing are imperceptibly small, and clinical tests used to diagnose movement dysfunctions are not supported by contemporary evidence. Although an assessment of pathoanatomical processes should not be disregarded, the degree to which they contribute to the pain experience is questionable given that nociceptive input from peripheral tissues represents only 1 potential contributor to the pain experience, regardless of pain location. On the basis of this information, there is a need for a paradigm shift in clinical reasoning, as assessing, diagnosing, and assigning causality of pain to movement dysfunction of the SIJ is disputed by available evidence.

Education plays a vital role in patient care management, and clinicians should carefully consider their role in perpetuating nonplausible pathoanatomical diagnoses, which may be harmful. There is a need to align the assessment, management and messaging associated with pain in the SIJ region with contemporary evidence. Clinicians should specifically aim to dispel potentially unhelpful misperceptions regarding SIJ movement dysfunctions. Further, they should address unhelpful beliefs about structural fragility instead of reinforcing these either explicitly with pathoanatomical labels or implicitly with treatment rationales.

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Funding

There are no funders to report.

Disclosure

The authors completed the ICJME Form for Disclosure of Potential Conflicts of Interest and reported no conflicts of interest.

References

1. Vos T, Abajobir AA, Abate KH, et al. Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. *The Lancet.* 2018;390(10100):1211-1259.
2. Maigne J, Planchon C. Sacroiliac joint pain after lumbar fusion. A study with anesthetic blocks. *European Spine Journal.* 2005;14(7):654-658.
3. Katz V, Schofferman J, Reynolds J. The sacroiliac joint: A potential cause of pain after lumbar fusion to the sacrum. *Journal of Spinal Disorders & Techniques.* 2003;16(1):96-99.
4. Liliang P-C, Lu K, Liang C-L, Tsai Y-D, Wang K-W, Chen H-J. Sacroiliac joint pain after lumbar and lumbosacral fusion: Findings using dual sacroiliac joint blocks. *Pain Medicine.* 2011;12(4):565-570.
5. Schwarzer AC, Aprill CN, Bogduk N. The sacroiliac joint in chronic low back pain. *Spine.* 1995;20(1):31-37.
6. Vleeming A, Albert H, Östgaard H, Sturesson B, Stuge B. European guidelines for the diagnosis and treatment of pelvic girdle pain. *European Spine Journal.* 2008;17(6):794-819.
7. Chou LH, Slipman CW, Bhagia SM, et al. Inciting events initiating injection-proven sacroiliac joint syndrome. *Pain Medicine.* 2004;5(1):26-32.
8. Visser LH, Nijssen PGN, Tijssen CC, van Middendorp JJ, Schieving J. Sciatica-like symptoms and the sacroiliac joint: clinical features and differential diagnosis. *European Spine Journal.* 2013;22(7):1657-1664.
9. Foster NE, Anema JR, Cherkin D, et al. Prevention and treatment of low back pain: evidence, challenges, and promising directions. *The Lancet.* 2018;*[Epub ahead of print]*.
10. Laslett M. Evidence-based diagnosis and treatment of the painful sacroiliac joint. *Journal of Manual & Manipulative Therapeutics.* 2008;16(3):142-152.
11. Laslett M, Aprill CN, McDonald B, Young SB. Diagnosis of sacroiliac joint pain: validity of individual provocation tests and composites of tests. *Man Ther.* Aug 2005;10(3):207-218.
12. Beales D, O'Sullivan P. A person-centered biopsychosocial approach to assessment and management of pelvic girdle pain In: Jull G, Moore A, Falla D, Lewis J, McCarthy C, Sterling M, eds. *Grieve's Modern Musculoskeletal Physiotherapy*. 4 ed: Elsevier; 2015:488-495.
13. Lee D. The pelvic girdle: a look at how time, experience and evidence change paradigms. In: Jull G, Moore A, Falla D, Lewis J, McCarthy C, Sterling M, eds. *Grieve's Modern Musculoskeletal Physiotherapy*. 4 ed: Elsevier; 2015:495-500.
14. van der Wurff P, Buijs EJ, Groen GJ. A multitest regimen of pain provocation tests as an aid to reduce unnecessary minimally invasive sacroiliac joint procedures. *Archives of physical medicine and rehabilitation.* Jan 2006;87(1):10-14.
15. Szadek KM, van der Wurff P, van Tulder MW, Zuurmond WW, Perez RSGM. Diagnostic validity of criteria for sacroiliac joint pain: A systematic review. *The Journal of Pain.* 2009;10(4):354-368.
16. Moseley GL, Butler D. *Explain Pain Supercharged*. Adelaide: Noigroup Publications; 2017.
17. Apkarian AV, Hashmi JA, Baliki MN. Pain and the brain: Specificity and plasticity of the brain in clinical chronic pain. *Pain.* 12/13 2011;152(3 Suppl):S49-S64.
18. Szadek KM, Hoogland PVJM, Zuurmond WWA, De Lange JJ, Perez RSGM. Possible nociceptive structures in the sacroiliac joint cartilage: An immunohistochemical study. *Clinical Anatomy.* 2010;23(2):192-198.
19. Szadek KM, Hoogland PV, Zuurmond WW, de Lange JJ, Perez RS. Nociceptive nerve fibers in the sacroiliac joint in humans. *Regional Anesthesia and Pain Medicine.* 2008;33(1):36-43.

20. McGrath MC, Zhang M. Lateral branches of dorsal sacral nerve plexus and the long posterior sacroiliac ligament. *Surgical and Radiological Anatomy.* 2005;27(4):327-330.

21. Vleeming A, Schuenke MD, Masi AT, Carreiro JE, Danneels L, Willard FH. The sacroiliac joint: an overview of its anatomy, function and potential clinical implications. *Journal of anatomy.* Dec 2012;221(6):537-567.

22. Moseley GL. Reconceptualising pain according to modern pain science. *Physical Therapy Reviews.* 2007/09/01 2007;12(3):169-178.

23. Murakami E, Tanaka Y, Aizawa T, Ishizuka M, Kokubun S. Effect of periarticular and intraarticular lidocaine injections for sacroiliac joint pain: Prospective comparative study. *Journal of Orthopaedic Science.* 2007;12(3):274-280.

24. Nacey NC, Patrie JT, Fox MG. Fluoroscopically Guided Sacroiliac Joint Injections: Comparison of the Effects of Intraarticular and Periarticular Injections on Immediate and Short-Term Pain Relief. *American Journal of Roentgenology.* 2016/11/01 2016;207(5):1055-1061.

25. Hodges PW, Smeets RJ. Interaction between pain, movement, and physical activity: short-term benefits, long-term consequences, and targets for treatment. *The Clinical journal of pain.* Feb 2015;31(2):97-107.

26. Palsson TS, Hirata RP, Graven-Nielsen T. Experimental Pelvic Pain Impairs the Performance During the Active Straight Leg Raise Test and Causes Excessive Muscle Stabilization. *The Clinical journal of pain.* Jul 2015;31(7):642-651.

27. Palsson TS, Graven-Nielsen T. Experimental pelvic pain facilitates pain provocation tests and causes regional hyperalgesia. *Pain.* 2012;153(11):2233-2240.

28. Byrd E, May S, Marsden J. The sacroiliac Joint: a survey of the current practice in the United Kingdom. *Physiotherapy.* 12// 2017;103, Supplement 1:e26.

29. Hodges C, Maxwell B. A time series survey of physical therapist assessment of sacro-iliac joint dysfunction in the United States. *Physiotherapy.* 2015;101:e579-e580.

30. Beales D, Hope JB, Hoff TS, Sandvik H, Wergeland O, Fary R. Current practice in management of pelvic girdle pain amongst physiotherapists in Norway and Australia. *Manual Therapy.* 2015/02/01/ 2015;20(1):109-116.

31. Sturesson B, Selvik G, Uden A. Movements of the sacroiliac joints. A roentgen stereophotogrammetric analysis. *Spine.* Feb 1989;14(2):162-165.

32. Goode A, Hegedus EJ, Sizer P, Brismee J-M, Linberg A, Cook CE. Three-Dimensional Movements of the Sacroiliac Joint: A Systematic Review of the Literature and Assessment of Clinical Utility. *The Journal of Manual & Manipulative Therapy.* 2008;16(1):25-38.

33. van der Wurff P, Hagmeijer RH, Meyne W. Clinical tests of the sacroiliac joint. A systematic methodological review. Part 1: Reliability. *Man Ther.* Feb 2000;5(1):30-36.

34. McGrath MC. Palpation of the sacroiliac joint: An anatomical and sensory challenge. *International Journal of Osteopathic Medicine.* 2006/09/01/ 2006;9(3):103-107.

35. Sturesson B, Uden A, Vleeming A. A radiostereometric analysis of the movements of the sacroiliac joints in the reciprocal straddle position. *Spine.* Jan 15 2000;25(2):214-217.

36. Kibsgård TJ, Røise O, Stuge B, Röhrl SM. Precision and Accuracy Measurement of Radiostereometric Analysis Applied to Movement of the Sacroiliac Joint. *Clinical Orthopaedics and Related Research.* 2012;470(11):3187-3194.

37. Bagg MK, Wand BM, Hirschberg J. Pelvic landmark displacement as a function of sacroiliac joint movement: a kinematic study. Paper presented at: 9th Interdisciplinary World Congress on Low Back and Pelvic Girdle Pain, Singapore2016; Singapore.

38. Hammer N, Scholze M, Kibsgård T, et al. Physiological in vitro sacroiliac joint motion: a study on three-dimensional posterior pelvic ring kinematics. *Journal of anatomy.* 2018;0(0).

39. Kibsgård TJ, Röhrl SM, Røise O, Sturesson B, Stuge B. Movement of the sacroiliac joint during the Active Straight Leg Raise test in patients with long-lasting severe sacroiliac joint pain. *Clinical Biomechanics.* 2017/08/01/ 2017;47:40-45.

40. Hungerford BA, Gilleard W, Moran M, Emmerson C. Evaluation of the Ability of Physical Therapists to Palpate Intrapelvic Motion With the Stork Test on the Support Side. *Physical Therapy.* 2007;87(7):879-887.

41. Arendt-Nielsen L, Graven-Nielsen T. Muscle pain: sensory implications and interaction with motor control. *The Clinical journal of pain.* May 2008;24(4):291-298.

42. Kmita A, Lucas NP. Reliability of physical examination to assess asymmetry of anatomical landmarks indicative of pelvic somatic dysfunction in subjects with and without low back pain. *International Journal of Osteopathic Medicine.* 2008/03/01/ 2008;11(1):16-25.

43. Fryer G, McPherson HC, O'Keefe P. The effect of training on the inter-examiner and intra-examiner reliability of the seated flexion test and assessment of pelvic anatomical landmarks with palpation. *International Journal of Osteopathic Medicine.* 2005;8(4):131-138.

44. Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics.* Mar 1977;33(1):159-174.

45. Stovall BA, Kumar S. Anatomical landmark asymmetry assessment in the lumbar spine and pelvis: a review of reliability. *PM & R : the journal of injury, function, and rehabilitation.* Jan 2010;2(1):48-56.

46. Loeser JD, Melzack R. Pain: an overview. *The Lancet.* 1999;353(9164):1607-1609.

47. Moseley GL, Butler DS. Fifteen Years of Explaining Pain: The Past, Present, and Future. *The journal of pain : official journal of the American Pain Society.* Sep 2015;16(9):807-813.

48. Zusman M. Forebrain-mediated sensitization of central pain pathways: 'non-specific' pain and a new image for MT. *Man Ther.* May 2002;7(2):80-88.

49. Bagg MK, McAuley JH, Moseley GL, Wand BM. Recent data from radiofrequency denervation trials further emphasise that treating nociception is not the same as treating pain. *Br J Sports Med.* Jan 19 2018;Epub ahead of print.

50. Ostelo RW, Deyo RA, Stratford P, et al. Interpreting change scores for pain and functional status in low back pain: towards international consensus regarding minimal important change. *Spine.* Jan 01 2008;33(1):90-94.

51. Cohen MDSteven P, Hurley MDPDRobert W, Buckenmaier MDChester C, Kurihara RNC, Morlando RNB, Dragovich MDA. Randomized Placebo-controlled Study Evaluating Lateral Branch Radiofrequency Denervation for Sacroiliac Joint Pain. *Anesthesiology.* 2008;109(2):279-288.

52. Patel N, Gross A, Brown L, Gekht G. A Randomized, Placebo-Controlled Study to Assess the Efficacy of Lateral Branch Neurotomy for Chronic Sacroiliac Joint Pain. *Pain Medicine.* 2012;13(3):383-398.

53. Juch JNS, Maas ET, Ostelo R, et al. Effect of Radiofrequency Denervation on Pain Intensity Among Patients With Chronic Low Back Pain: The Mint Randomized Clinical Trials. *Jama.* Jul 04 2017;318(1):68-81.

54. Ossipov MH, Morimura K, Porreca F. Descending pain modulation and chronification of pain. *Current opinion in supportive and palliative care.* 2014;8(2):143-151.

55. IASP. *Part III: Pain Terms, A Current List with Definitions and Notes on Usage*. Seattle: IASP Press; 1994.

56. Arendt-Nielsen L, Graven-Nielsen T, Svarrer H, Svensson P. The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. *Pain.* 1996;64(2):231-240.

57. Karayannis NV, Smeets RJ, van den Hoorn W, Hodges PW. Fear of Movement Is Related to Trunk Stiffness in Low Back Pain. *PloS one.* 2013;8(6):e67779.

58. Hodges PW, Moseley GL. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. *J Electromyogr Kinesiol.* 2003;13(4):361-370.

59. Darlow B, Dean S, Perry M, Mathieson F, Baxter GD, Dowell A. Easy to Harm, Hard to Heal: Patient Views About the Back. *Spine.* Jun 01 2015;40(11):842-850.

60. Bunzli S, Smith A, Watkins R, Schutze R, O'Sullivan P. What Do People Who Score Highly on the Tampa Scale of Kinesiophobia Really Believe?: A Mixed Methods Investigation in People With Chronic Nonspecific Low Back Pain. *The Clinical journal of pain.* Jul 2015;31(7):621-632.

61. Sloan TJ, Walsh DA. Explanatory and diagnostic labels and perceived prognosis in chronic low back pain. *Spine.* Oct 01 2010;35(21):E1120-1125.

62. Vlaeyen JW, Linton SJ. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain.* Apr 2000;85(3):317-332.

63. Young Casey C, Greenberg MA, Nicassio PM, Harpin RE, Hubbard D. Transition from acute to chronic pain and disability: a model including cognitive, affective, and trauma factors. *Pain.* Jan 2008;134(1-2):69-79.

64. Bunzli S, Smith A, Schutze R, O'Sullivan P. Beliefs underlying pain-related fear and how they evolve: a qualitative investigation in people with chronic back pain and high pain-related fear. *BMJ Open.* Oct 19 2015;5(10):e008847.

65. Linton SJ. A review of psychological risk factors in back and neck pain. *Spine (Phila Pa 1976).* May 01 2000;25(9):1148-1156.

66. Darlow B, Dowell A, Baxter GD, Mathieson F, Perry M, Dean S. The Enduring Impact of What Clinicians Say to People With Low Back Pain. *The Annals of Family Medicine.* November 1, 2013 2013;11(6):527-534.

67. Kendrick D, Fielding K, Bentley E, Kerslake R, Miller P, Pringle M. Radiography of the lumbar spine in primary care patients with low back pain: randomised controlled trial. *BMJ.* 2001;322(7283):400.

68. Leventhal H, Brissette I, Leventhal EA. The common-sense model of self-regulation of health and illness. In: Cameron LD, Leventhal H, eds. *The Self-Regulation of Health and Illness Behaviour*. London: Taylor & Francis/Routledge; 2003:42-65.

69. Darlow B, Perry M, Stanley J, et al. Cross-sectional survey of attitudes and beliefs about back pain in New Zealand. *BMJ Open.* May 23 2014;4(5):e004725.

70. Setchell J, Costa N, Ferreira M, Makovey J, Nielsen M, Hodges PW. Individuals' explanations for their persistent or recurrent low back pain: a cross-sectional survey. *BMC Musculoskeletal Disorders.* 2017;18:466.

71. Darlow B. Beliefs about back pain: The confluence of client, clinician and community. *International Journal of Osteopathic Medicine.* 2016;20:53-61.

72. Traeger AC, Hubscher M, Henschke N, et al. Emotional distress drives health services overuse in patients with acute low back pain: a longitudinal observational study. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society.* Sep 2016;25(9):2767-2773.

73. Bunzli S, Smith A, Schutze R, Lin I, O'Sullivan P. Making Sense of Low Back Pain and Pain-Related Fear. *The Journal of orthopaedic and sports physical therapy.* Sep 2017;47(9):628-636.

74. Bialosky JE, Bishop MD, Price DD, Robinson ME, George SZ. The mechanisms of manual therapy in the treatment of musculoskeletal pain: A comprehensive model. *Manual Therapy.* 2009/10/01/ 2009;14(5):531-538.

75. Fransen M, McConnell S, Harmer AR, Van der Esch M, Simic M, Bennell KL. Exercise for osteoarthritis of the knee. *Cochrane Database of Systematic Reviews.* 2015(1).

76. Fransen M, McConnell S, Hernandez-Molina G, Reichenbach S. Exercise for osteoarthritis of the hip. *The Cochrane database of systematic reviews.* Apr 22 2014(4):Cd007912.
77. Koltyn KF, Brellenthin AG, Cook DB, Sehgal N, Hillard C. Mechanisms of Exercise-Induced Hypoalgesia. *The journal of pain : official journal of the American Pain Society.* 2014;15(12):1294-1304.

520 **Table.**

521 Overview of Studies Testing Movements in the Pelvic Girdle (Sacroiliac Joints and Pubic Symphysis) Using

522 Radiostereometric Analysis[a]

523

Movement	Sample	Side (n)	Measurement	Rotation (Degrees) About Cardinal Axes			Helical Axis	
				x-Axis	y-Axis	z-Axis	Rotation (°)	Translation (mm)
Standing to standing with left hip maximally flexed[35]	n = 22 (4 females) with diagnosed sacroiliac joint syndrome	Left (21)	Mean	−0.2	0.2	0.2	0.6	0.3
			Range	1.0 to 0.5	−0.7 to 0.8	−0.3 to 0.9	0.2 to 1.4	0.1 to 1.0
			SD	0.4	0.4	0.3	0.4	0.2
		Right (20)	Mean	−0.2	−0.1	0.1	0.6	0.3
			Range	−1.4 to 0.2	−0.8 to 0.5	−0.4 to 0.8	0.2 to 1.8	0 to 2.2
			SD	0.4	0.4	0.3	0.4	0.4
Standing to standing with right hip maximally flexed[35]	n = 22 (4 females) with diagnosed sacroiliac joint syndrome	Left (20)	Mean	−0.1	0	0.1	0.7	0.3

			Range	−1.0 to 0.7	−1.1 to 1.8	−0.3 to 1.2	0.1 to 1.8	0 to 0.7
			SD	0.5	0.5	0.4	0.5	0.2
		Right (22)	Mean	−0.2	−0.2	−0.2	0.7	0.3
			Range	−0.7 to 0.2	−1.0 to 0.9	−0.8 to 0.5	0.2 to 1.2	0 to 0.8
			SD	0.3	0.5	0.3	0.3	0.2
Active straight-leg raise of right lower limb[39]	n = 12 (11 females) with pelvic girdle pain attributed to sacroiliac joint fusion	Left (12)	Mean	−0.8	−0.2	0.3	1	0
			Range	−1.3 to −0.3	−0.8 to 0.5	−0.1 to 0.7	0.5 to 1.4	−0.1 to 0.1
			SD	0.3	0.3	0.2	0.3	0.1
		Right (12)	Mean	0	−0.1	0	0.7	0
			Range	−1.0 to 0.5	−0.8 to 0.5	−0.5 to 0.5	0.3 to 1.4	−0.3 to 0.3
			SD	0.4	0.3	0.2	0.4	0.1

[a]The included studies were performed on individuals with non–pregnancy-related pelvic girdle pain and measured the absolute differences in static positions of the sacroiliac joint in a neutral position and then in various positions of weight bearing and non–weight bearing.