Prolotherapy for the Thoracolumbar Myofascial System

Bradley D. Fullerton, MD[a,b,*]

KEYWORDS

• Prolotherapy • Thoracolumbar fascia • Interspinous ligament • PRP • Ultrasound • Low back pain • Biotensegrity

KEY POINTS

- Prolotherapy has focused on entheses as a key source of chronic low back pain, even without clear diagnosis of enthesopathy.
- Treatment has traditionally been guided by anatomic knowledge and careful palpation. Dynamic ultrasonography can visualize tissue pathology previously identified only by detailed palpation.
- Prolotherapy cannot be fully understood without knowledge of biotensegrity and fascial anatomy.
- Biotensegrity provides the framework for a fundamentally different understanding of biomechanics, musculoskeletal diagnosis, and treatment.
- Detailed case studies of chronic pain resolution can provide proof of concept evidence for prolotherapy in the treatment of low back pain.

Video content accompanies this article at http://www.pmr.theclinics.com.

PROLOTHERAPY: WHAT IS IT?

In the 1958 edition of his classic book on prolotherapy, Hackett described the treatment as a strengthening of "the weld of disabled ligaments and tendons to bone by stimulating the production of new bone and fibrous tissue cells…"[1] The 1939 research of Kellgren,[2] showing sciatic-like referral from the interspinous ligaments of the lumbar spine (**Fig. 1**), led Hackett to focus on ligaments as a source of pain

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[a] Private Practice: ProloAustin, 2714 Bee Cave Road, Suite 106, Austin, TX 78746, USA; [b] Department of Internal Medicine, Texas A&M College of Medicine, Health Professions Education Building, 8447 Riverside Parkway, Bryan, TX 77807, USA
* Private Practice: ProloAustin, 2714 Bee Cave Road, Suite 106, Austin, TX 78746.
E-mail address: drbdf@aol.com

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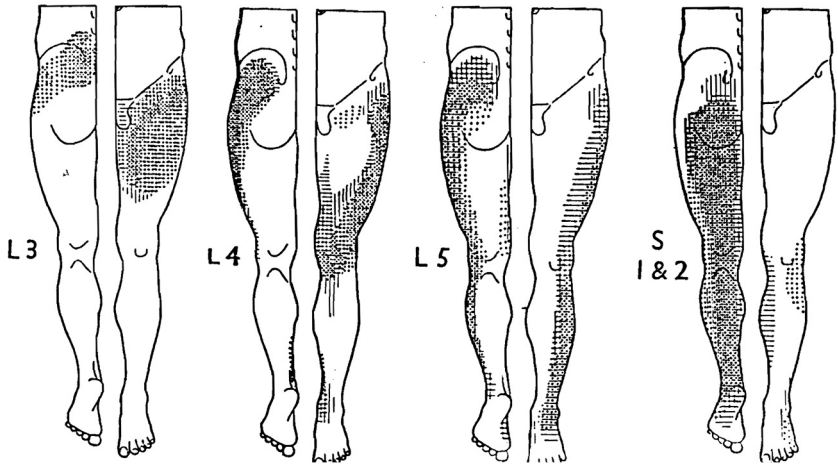

Fig. 1. A composite image adapted from Kellgren's original article showing referral patterns induced by injecting 0.1 mL to 0.3 mL of 6% saline into the interspinous ligament. At each level, the left image is a posterior view, whereas the right image is an anterior view. (*From* Kellgren JH. On the distribution of pain arising from deep somatic structures with charts of segmental pain areas. Clin Sci 1939;4(35):38; with permission.)

referral. He reasoned that stimulating repair in these structures could resolve long-standing low back and sciatic pain. He also performed his own experiments documenting the referral patterns of sacroiliac ligaments into the lower extremities (Fig. 2).[3]

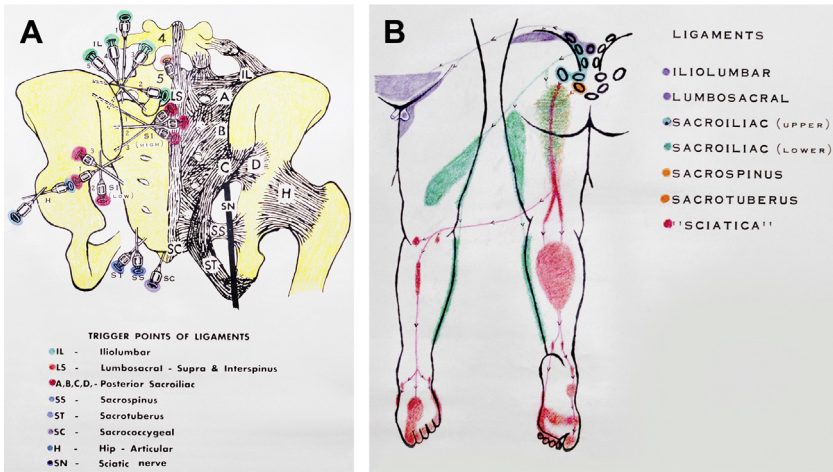

Fig. 2. A composite image of Hackett's original color diagrams documenting pain referral patterns with hypertonic saline injection into various ligaments supporting the sacroiliac joint. (*A*) indicates injection location whereas (*B*) indicates the referral patterns of those injections. (*From* Hackett GS, Hemwall GA, Montgomery GA. Ligament and tendon relaxation treated by prolotherapy. 5th edition. Madison (WI): Hackett Hemwall Patterson Foundation; 1993. p. 26–36. Available at: http://hhpfoundation.org/; with permission.)

As a result, the practice of prolotherapy has developed as a process of widespread injection of enthesis points using pain and crepitus to palpation as a guide to diagnosis and injection location. Yet, many of the anatomic structures injected have no known documented pathology and thus are not included in standard differential diagnosis. This has made study design difficult and opened the technique to significant criticism. The narrow focus on the disk as the most common cause of back pain continues to this day even in regenerative medicine circles.[4] If Kellgren and Hackett were correct, however, there are many tissues of the spine that are still minimized as potential sources of pain and, more importantly, as sources of abnormal biomechanics, which could contribute to degenerative spine conditions.

In 1995, *SPINE: State of the Art Reviews* published a volume on prolotherapy in the lumbar spine and pelvis. In that volume, Mooney, an orthopedic spine surgeon and one of the founding editors of the journal *Spine*, reported on his own experience of "the bias in the scientific community against innovative concepts" such as prolotherapy.[5] He touched on the difficulty of diagnosis in orthopedic medicine versus orthopedic surgery. He stated, "in contrast to fractures, each problem is somewhat different as to the location of the weak link." He encouraged readers to be open to new words and concepts such as "tensegrity … and particularly the integrated fascial system."

FASCIAL ANATOMY AND BIOTENSEGRITY

In the more than 20 years since that publication, this integrated fascial system has received extensive attention from fields, such as osteopathy and manual therapy/bodywork. In 2007, the first Fascia Research Congress met as an effort to bring together connective tissue research scientists and a diverse community of body workers.[6] In 2012, Willard and colleagues[7] published a thorough examination of the anatomy and function of the thoracolumbar fascia (TLF), which included a transverse section of the layers and sublayers of this structure. This image (**Fig. 3**) facilitates understanding of ultrasound images as discussed later in the case studies. This standardization of nomenclature for the TLF also reveals how this system integrates input from epaxial (spine), hypaxial (abdomen), and appendicular (the 4 extremities) myofascial systems. In 2015, Stecco[8] authored the first anatomic text focused on the continuity of the myofascial system throughout the body; she advocates for a slightly different nomenclature of the TLF.

The underlying biomechanics of this integrated fascial system have been explored by Stephen Levin, an orthopedic surgeon and systems scientist.[9] Levin expanded on concepts from futurist and architect Buckminster Fuller, who proposed the term, *tensegrity* (a combination of tension and integrity), to describe a class of constructs known as *floating compression* by the sculptor Kenneth Snelson (Video 1).

To understand structural stability with minimal mass, Fuller explained that "tension is primary and comprehensive, and compression is secondary and local."[10] Levin argues that traditional biomechanics, which involve lever forces and ultimately shear, will never fully explain the intrinsic mobility with stability of biologic structures. Levin proposed the term, *biotensegrity*, as the application of these concepts to biologic structures. In biotensegrity structures, there are no bending moments and no shear forces; thus, they have the lowest energy costs.

In the same volume of *SPINE: State of the Art Reviews*,[11] Levin stated that the sacrum and all other bones should be considered as compression-bearing structures, which float in a continuous network of tension supplied by fascia, muscles, ligaments, and other connective tissue. Thus, the soft tissues are the primary frame of the body, whereas the bones are islands within that continuous frame.

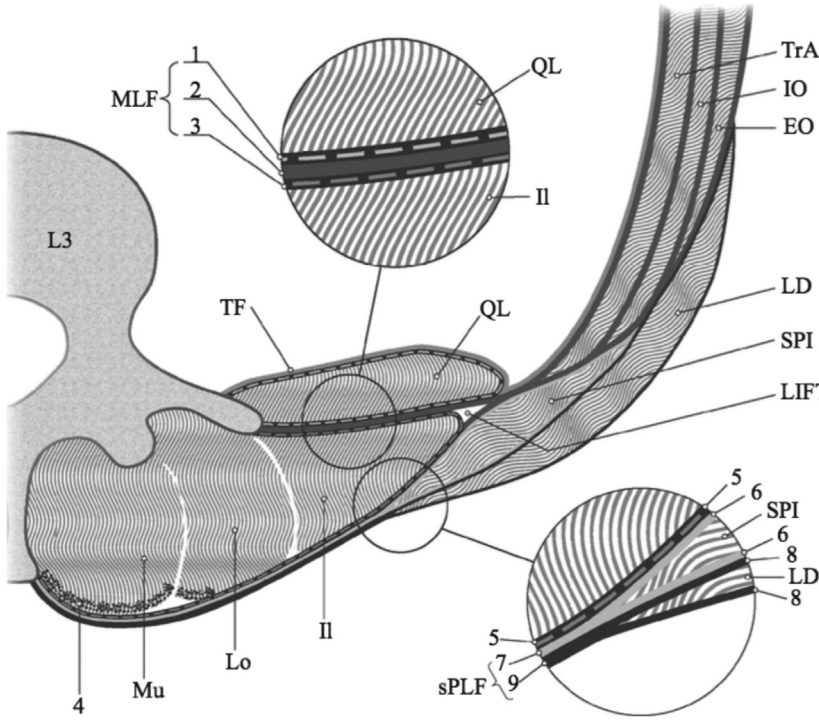

Fig. 3. Transverse view of TLF layers: This diagram correlates with an axial view of the traditional lumbar MRI. The patient is supine and the view is from caudal to cephalad of the left side. Willard and colleagues state, "This is a transverse section of the PLF and MLF and related muscles at the L3 level. Fascial structures are represented such that individual layers are visible, but not necessarily presented to scale." The details of each sublayer are beyond the scope of this review. Structures identified in the 2 case reports include hypaxial myofascia (the transversus abdominis [TrA] muscle and quadratus lumborum [QL]), epaxial myofascia (the 3 paraspinal muscles, multifidus [Mu], longissimus [Lo], and iliocostalis [Il], which are deep to the PLF; all provide input into the aponeurosis of the erector spinae muscles [Apo ES] indicated as #4 and by a stippling pattern), and appendicular myofascia (latissimus dorsi [LD], serratus posterior inferior [SPI], and lower trapezius, which is not depicted at this level). The MLF is a combination of the hypaxial and epaxial myofascia. These layers are magnified in the upper circle of the diagram. The PLF is a combination of the epaxial and appendicular myofascia. These layers are magnified in the lower circle of the diagram. The LIFT is a combination of all 3 myofascial layers. The lateral raphe (not labeled) connects the abdominal wall muscles to the TLF; it includes the LIFT and tendon of the TrA. sPLF is superficial layer of PLF. IO is internal oblique. EO is external oblique. (*Adapted from* Willard FH, Vleeming A, Schuenke MD, et al. The thoracolumbar fascia: anatomy, function and clinical considerations. J Anat 2012;221(6):517; with permission.)

Biotensegrity structures are also fully integrated so that forces are instantaneously transferred through the entire structure. If the tension in the structure is continuous, any force applied to the structure deforms the entire structure. The structure itself then becomes a far more rapid form of communication than nerve conduction. When there is a loss of tension somewhere in the system, this communication is interrupted resulting in abnormal muscle recruitment and altered mobility. The task for the prolotherapist then becomes finding the loss of tensional integrity in the soft tissue frame and stimulating repair at the loss of tensegrity.

BIOTENSEGRITY-BASED ANATOMY AND BIOMECHANICS—A SUMMARY

1. Muscles are not structures that pull on bones to cause movement.
2. Bones float in a variable tension network consisting of muscle and fascial elements.
3. Fascial continuity provides passive tension and stored energy in the form of prestress.
4. Muscle fibers within the fascial continuity provide dynamic tension.
5. Thus, bones move (or, in the setting of outside forces, remained stable) when the tension around them changes.

CLINICAL APPLICATION OF BIOTENSEGRITY PRINCIPLES—A SUMMARY

1. Construct—focus on tensional continuity, which provides instantaneous, body-wide communication
2. History—past trauma is a loss of prestress in the tensegrity system
3. Signs/symptoms—muscle hyperactivity is compensation for loss of prestress (or loss of tensional continuity)
4. Diagnosis—use dynamics (in physical examination and ultrasonography) to find a loss of tensional continuity in the myofascia
5. Regenerative treatment—applied at the loss of tensional continuity (which may or may not be the location of a pain generator)

DETAILED CASE REPORTS AS PROOF OF CONCEPT

The complexity of biotensegrity structures and the complexity of prolotherapy treatments make large well-designed studies difficult. A recent review found only 2 studies of prolotherapy in low back pain that met criteria for inclusion.[12] With the large number of connective tissue structures as possible sources of pain and instability, even agreement on diagnosis among experienced practitioners can be difficult. These difficulties should not deter examining the decades of positive clinical reports concerning prolotherapy. It may be that the underlying pathology treated by prolotherapy would be better understood by detailed study of individual patients rather than starting with large controlled studies.

Case 1—Chronic Back Spasm Related to Injury of the Posterior Layer of the Thoracolumbar Fascia/Aponeurosis of the Erector Spinae Muscles/Interspinous Ligament/Multifidi Tendon

A 48-year-old white man with a greater than 10-year history of waxing and waning, activity-dependent low back pain and spasm without gluteal or lower extremity referral. Approximately 15 years prior to presentation he had an episode of his back locking up when flexed. He did not seek treatment and was able to recover over several days' time. These symptoms resolved and he was able to complete an iron man triathlon in the years after that. The onset of his chronic symptoms was gradual and initially seemed minor. Eventually he saw a chiropractor and had adjustments that did not affect his back pain. He sought consultation with an orthopedic spine surgeon; a lumbar MRI showed degenerative disk disease in the lower lumbar spine. These records were unavailable for review. In the year prior to his recurring back spasm, he was playing table tennis and felt a "pull" in his low back. Since that time, he has had daily low back pain, and exercise has been limited by recurring left lumbar muscle spasm. He is unable to complete lumbar strengthening exercises or participate in aerobic activities due to these back spasms.

Physical examination is remarkable for visually prominent left thoracolumbar paraspinals during single-leg stance. He is unsteady with single-leg squat bilaterally. In bipedal standing, there is a palpable deviation of the upper lumbar, posterior spinous processes toward the right. In supine position, he is unable to maintain straight leg raise (SLR) against resistance bilaterally. In prone position, there is palpable crepitus and tenderness on the left side of the upper lumbar spinous processes but none on the right. This correlated with ultrasound findings (Video 2). In prone he has difficulty but can partially maintain SLR against resistance bilaterally. With prone hip external rotation against resistance, he is unable to maintain stability at the midlumbar spine. His spine rotates at that location when testing either right or left. Otherwise, lower extremity strength, sensation, and muscle stretch reflexes are normal. There are no neural tension signs and lumbar range of motion is only mildly limited in flexion.

Ultrasound examination is focused at midline lumbar spine with linear-array high-frequency transducer. This revealed a cortical pit at the L1 posterior spinous process with thickening of the posterior layer of the TLF (PLF) on the left compared with the right (**Fig. 4**). With sonographic palpation (Video 3), the left longissimus lumborum

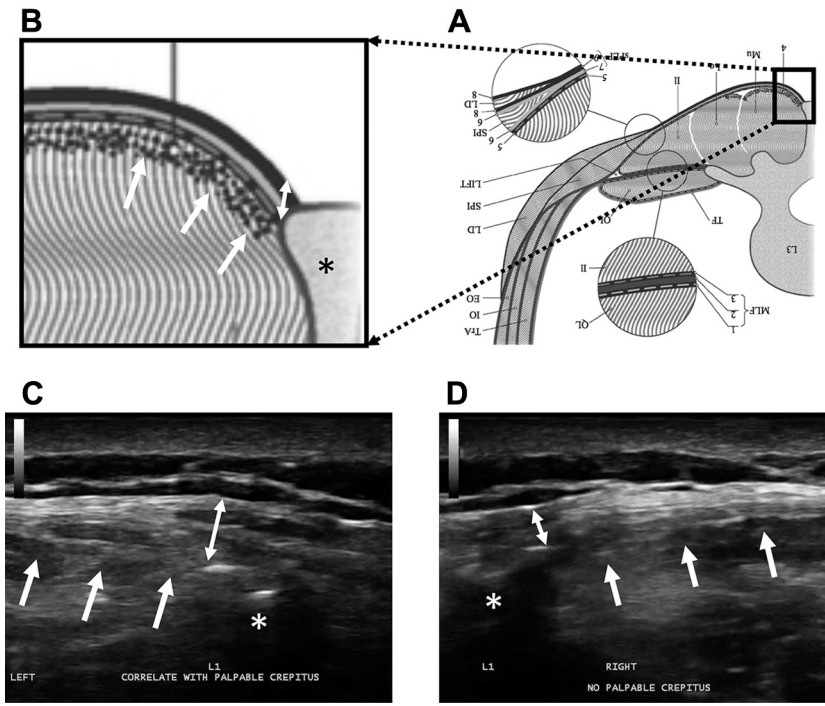

Fig. 4. PLF and Apo ES ultrasound. (*A*) is the same diagram as shown in **Fig. 3**; however, it has been reversed to correlate with the view seen on ultrasound when the patient is in the prone position. The view is still from caudad to cephalad viewing the left side. (*B*) Magnified view of the same diagram correlating with the black box in (*A*). (*C*, *D*) Ultrasound images of the patient in case 1. In *B, C* and *D*, single-head arrows point to the Apo ES, which is irregular and poorly defined on the left compared with the right. In *B, C* and *D*, double-head arrows indicate the thickness of the PLF at the insertion into the L1 posterior spinous process, with cortical pit indicated by (*asterisk*). The left PLF is thickened and heterogenous compared with the right. ([*A, B*] *Adapted from* Willard FH, Vleeming A, Schuenke MD, et al. The thoracolumbar fascia: anatomy, function and clinical considerations. J Anat 2012;221(6):517; with permission.)

fibers partially collapse and translate laterally. The right longissimus lumborum fibers maintain stability with compression and do not translate laterally (**Fig. 5**). At lower lumbar levels, the multifidi appeared partially torn at the spinous process and blending with the aponeurosis of the erector spinae muscles (Apo ES). This is seen most clearly with compression at L5 (Video 4).

Treatment involved initial injection of a solution of 0.3% lidocaine and 15% dextrose at the tissues of interest under ultrasound guidance, then repeat of physical examination maneuvers to evaluate response. This was then repeated for other structures until the patient was pain-free and had normal dynamic testing (**Box 1**). The same structures are then injected with a total of 4-mL low–white blood cell, low–red blood cell, autologous platelet-rich plasma (PRP) derived from 60 mL of whole blood.

At follow-up 2.5 months after procedure, the patient reported significant improvement in his chronic pain. He described having no back pain the day after the procedure and states, "that was really interesting." Then the pain returned to its normal baseline; 6 weeks to 8 weeks later, he noticed the episodes of activity-limiting pain and spasm were decreasing. In discussing the back pain, he reported, "I always feel it" but "it doesn't get in my way now." He has been able to run up to 2 miles without increase in back pain and estimates overall improvement at 50%.

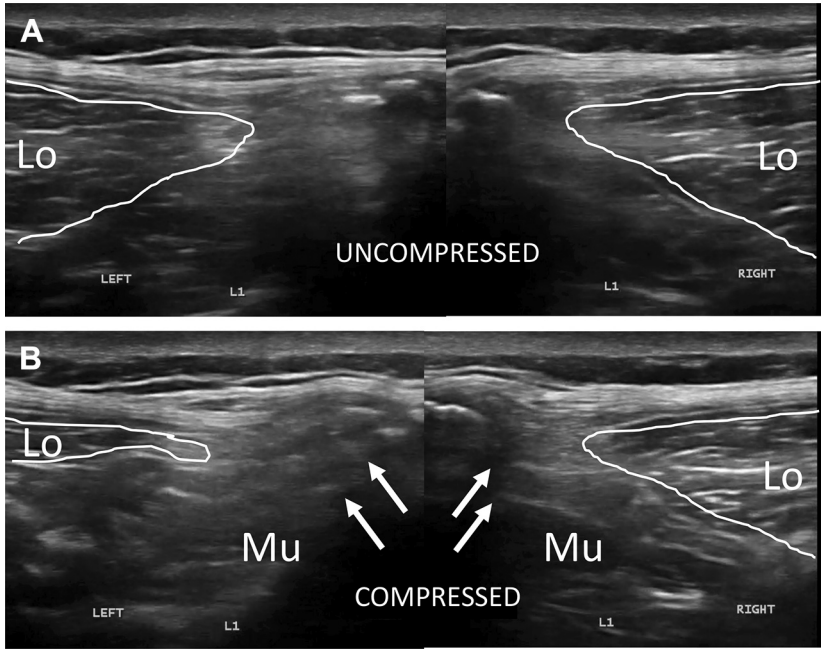

Fig. 5. Longissimus at L1 uncompressed and compressed with labels. (*A*, *B*) Composites of still images taken from Video 3 with the posterior spinous process at the midline. Longissimus (Lo) is outlined in white. At upper lumbar levels the longissimus fibers extend more medially overlying the multifidus (Mu). In (*A*), the uncompressed left side is farther from the spinous process than the uncompressed right. In (*B*), the left longissimus compresses easily and translates farther lateral compared with the right. The arrows point toward the blending of multifidus tendon into the Apo ES, which are also more heterogenous and hypoechoic on the left compared with the right.

Box 1
Case 1, structures injected

1. Left PLF and Apo ES at L1 and L2 spinous processes (Video 5)
2. L1-2 and L2-3 supraspinous and interspinous ligaments (Video 6)[13]
   a. After injection of 1 and 2, supine and prone SLR strength against resistance was normal and without pain
   b. Prone hip external rotation strength testing showed improved control of lumbar rotation.
3. Left multifidus insertions at L3-5 posterior spinous processes (Video 7)
   a. After injection 3, prone hip external rotation strength testing showed complete stability of lumbar rotation.

On examination, there is no palpable deviation of the upper lumbar, posterior spinous processes toward the right in bipedal standing. There is no tenderness to palpation and minimal crepitus in this area. He had full strength with prone SLR against resistance. There was still mild lumbar rotation with prone hip external rotation against resistance. Ultrasonography showed clear improvements in tissue organization and resistance to sonographic compression (Video 8). The plan was to continue increasing activity to tolerance and follow-up in 2 months if pain returned or progress plateaued.

He did not return for follow-up. At phone follow-up 2.5 years' posttreatment, he reported that the pain and spasm had continued to reduce over the several months after the last appointment. He has had no back spasms, even with increased activity, over the past 2.5 years. He says he is approximately 75% better compared with before treatment and has no limitations from his back pain.

Case 1 discussion

Prolotherapy in the lumbosacral spine is a purposefully broad injection pattern that seems imprecise to those unfamiliar with the technique. In the lumbar spine, common injection locations include the posterior and lateral surfaces of the posterior spinous processes, the lateral and posterior surfaces of the transverse processes, facet joint capsules, iliolumbar ligament insertions at the ilium, and sacroiliac ligaments indicated on the Hackett diagram (see **Fig. 2**). A similar standardized injection pattern was used in a randomized controlled study by Yelland and colleagues.[14] The conclusion of this article states, "in chronic nonspecific low back pain, significant and sustained reductions in pain and disability occur with ligament injections, irrespective of the solution injected or the concurrent use of exercises." Although this study was performed more than 12 years ago, ligament injection to treat chronic nonspecific low back pain is offered by few physicians. Understanding ligament and other connective tissue pathology may motivate more physicians to learn this method.

The approach described in this case is a hybrid of traditional prolotherapy injection location (informed by history and physical examination findings) with ultrasound diagnosis and ultrasound guidance to clarify the pathology traditionally treated with prolotherapy. The laxity in the Apo ES and the overlying PLF could have resulted from the acute episode of the pull while playing table tennis and/or the chronic, repetitive trauma of his exercise regimen with breakdown of collagen fibers. Using tensegrity theory, the recurring muscle spasms in this patient are interpreted as an attempt by the body to reintroduce prestress into these fascial layers. The laxity in these collagen layers would conceivably change the length-tension relationship of the muscles that insert into these layers. This would alter the timing of muscle recruitment and cause repetitive abnormal stresses to other tissues, such as the disks, over time.

In the author's experience, prolotherapy to the interspinous ligament (as in this case) can result in remarkable clinical improvements. The misunderstood anatomy of the interspinous ligaments may provide some explanation for this observation. Classic anatomy texts[15,16] describe the interspinous ligaments as connecting adjacent posterior spinous processes. As described by Willard,[13] however, the interspinous ligament fibers are in parallel with the spinous processes; thus, they connect the supraspinous ligament to the ligamentum flavum. The fanlike fibers are narrow as they blend with the ligamentum flavum and become broader as they blend with the supraspinous ligament. During lumbar flexion, this arrangement transmits tension from the TLF/supraspinous ligament to the ligamentum flavum, resulting in elevation of the ligamentum flavum away from the spinal canal (**Fig. 6**). Because the ligamentum flavum also blends with the facet capsules, the interspinous ligament transmits tension to those capsules as well. This may have profound implications in the understanding of ligamentum flavum buckling on MRI and the development of degenerative lumbar stenosis.

If injection of dextrose and PRP stimulates formation of collagen and return of prestress to the system, the muscle spasms should gradually reduce in the months after treatment as in this case. If the tissue diagnosis and injection location or correct, the clinical results should be long lasting, as in this case and the study by Yelland and colleagues[14]

Case 2—Recurring Sciatica Versus Pseudosciatica from Injury to Lumbar Interfascial Triangle and Gluteus Minimus/Medius

A 52-year-old woman with a 5-month history of right low back and gluteal pain referring to posterior lateral thigh and calf. This started after performing a headstand in

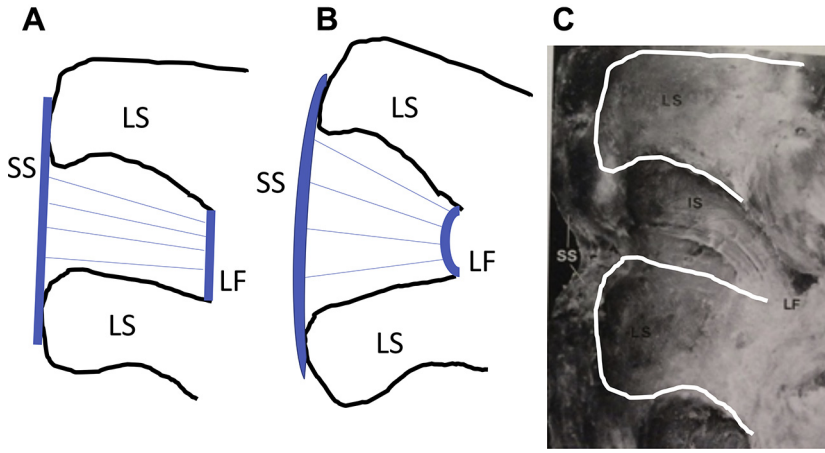

Fig. 6. Interspinous ligament anatomy and biomechanics. (*C*) A gross anatomy picture from Willard; the original image has been adapted by outlining the lumbar posterior spinous processes (LS) in white. (*A*) Shows the spine in neutral; (*B*) shows the spine in flexion. The thick lines correspond to the supraspinous ligament (SS) and ligamentum flavum (LF); the thin lines represent the fan shaped pattern of the interspinous ligament (IS) fibers. Note that in flexion, the IS fibers serve to transmit tension from SS to LF resulting in elevation of the LF away from the spinal canal. ([*C*] *Adapted from* Willard F. The muscular, ligamentous and neural structure of the lumbosacrum and its relationship to low back pain. In: Vleeming A, Mooney V, Dorman T, et al, editors. Movement, stability and low back pain: the essential role of the pelvis. New York: Churchill Livingstone; 1997. p. 7; with permission.)

yoga while pulling the legs up over her head; 2 months prior to this office visit, she had been diagnosed by a board-certified physiatrist with right lumbosacral radiculopathy; MRI indicated "Right paracentral disk herniation at L5-S1 inferior extrusion contacting the right S1 nerve root, Schmorl's node at the inferior aspect of the L5 vertebral body." She was given a "prednisone taper" and referred for lumbar extension–based therapy, which had been partially effective.

She also recalled 2 prior injuries. Six years previously, when learning to wakeboard, she was resisting being pulled up into a standing position when she felt a "pop" in her right low back and immediate pain and collapsed into lumbar and hip flexion. She had to be carried from the shore line to her car and required evaluation in an emergency room. She received an unknown injection and recovered over several days. Five years previously, she flipped over the handlebars of her mountain bike landing on the right low back causing significant abrasion and permanent scar near the posterior superior iliac spine.

At my initial evaluation, she continued to have sciatic pain (**Fig. 7**A) with an equivocal right SLR, absent right Achilles reflex, and normal strength and sensation. She was referred for physical therapy with focus on core strengthening and progressed well yet without resolution. She managed her chronic low back/hip pain using a lumbar pillow when sitting and remained active, including extended hiking vacations over the next 15 months. At that time, she had recurrence of the severe symptoms with sciatic referral (**Fig. 7**B) after performing a yoga strengthening exercise involving supine position, 90° hip flexion, and then maximal lumbar rotation side to side. She initially sought evaluation with the previous physician, who found recurrence of the positive SLR and recommended epidural steroid injection or evaluation for surgery on the herniated disk.

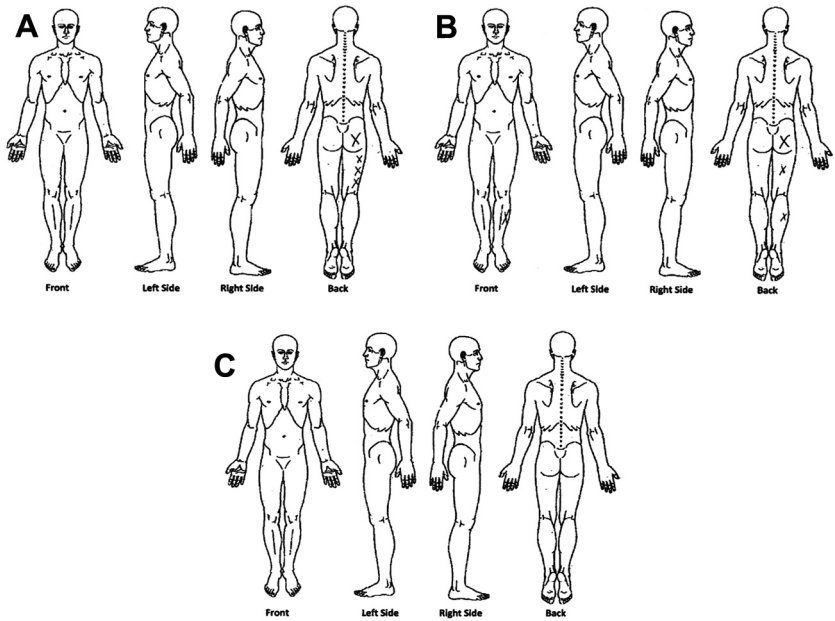

Fig. 7. Sequential pain diagrams of patient presenting with diagnosis of radiculopathy. (*A*) At the initial evaluation. Pain improved with physical therapy and she returned more than 1 year later with severe exacerbation. (*B*) At the time of representation and treatment with dextrose/PRP prolotherapy. (*C*) At 3-month follow-up posttreatment.

She sought a second opinion at my office, where physical examination was remarkable for standing posture with mild hip flexion; limited active lumbar range of motion in flexion, extension and left lateral flexion; unstable right single-leg squat; weak hip abduction and extension; positive right SLR at 35°; and tenderness to palpation at the right quadratus lumborum, right lateral gluteal muscles/ilium, and right gluteus medius tendon at the posterior facet of the greater trochanter. Sensation and lower extremity muscle stretch reflexes were normal.

Ultrasonography of the TLF system revealed a loss of tensional integrity in the right longissimus lumborum, quadratus lumborum, middle layer of the TLF (MLF) (**Fig. 8**, Video 9), lumbar interfascial triangle (LIFT), and lateral raphe at L4 (**Fig. 9**, Video 10). Ultrasonography of the right gluteal muscles revealed a loss of tensional integrity in the proximal musculotendinous junction of the gluteus medius, origin of the posterior part of the gluteus minimus from the ilium, and an intrasubstance partial tear of the gluteus medius tendon at the posterior facet of the greater trochanter.

Treatment involved initial injection of a solution of 0.3% lidocaine and 15% dextrose at the tissues of interest under ultrasound guidance and then repeat of dynamic physical examination maneuvers to evaluate response. This was then repeated for other structures until the patient was pain-free and had normal testing (**Box 2**). The same structures are then injected with a total of 10-mL low–white blood cell, low–red blood cell, autologous PRP derived from 120 mL of whole blood.

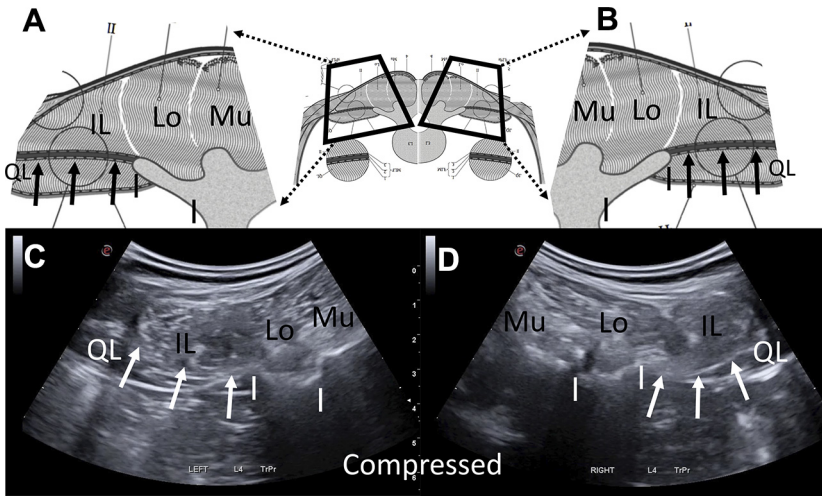

Fig. 8. Axial ultrasound images with compression at the L4 transverse process (TrPr). The center of (*A*, *B*) is a composite image using the transverse view of the TLF from Willard and colleagues, showing left and right sides of a prone patient. The lateral parts of (*A*) and (*B*) are a magnified image corresponding to the black trapezoids at the center, as indicated by the black dotted arrows. The trapezoids represent the section visualized on the curved-array ultrasound images in (*C*, *D*) (*left* and *right*, respectively). The length of the L4 TrPr is demarcated by the vertical white lines in the lower images and vertical black lines in the upper images. The MLF attaches to the tip of the TrPr (a common location of injection in prolotherapy); it is indicated by the white arrows in the lower images and the black arrows in the upper images. Note the poor definition of the compressed MLF and quadratus lumborum (QL) on the right compared with the left. Other muscles identified in *A–D* include multifidus (Mu), longissimus (Lo) and iliocostalis (IL). See Video 9 showing compression at this location. ([*A, B*] *Adapted from* Willard FH, Vleeming A, Schuenke MD, et al. The thoracolumbar fascia: anatomy, function and clinical considerations. J Anat 2012;221(6):517; with permission.)

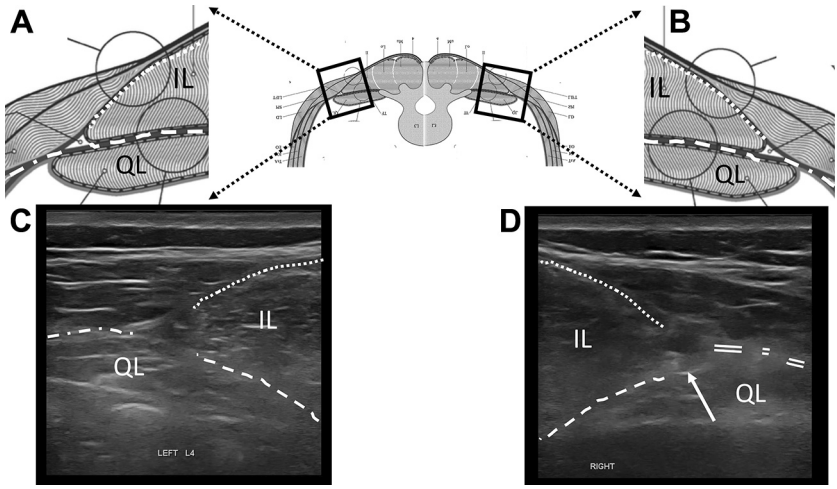

Fig. 9. Axial ultrasound images of bilateral LIFT at L4. The center of (*A*) and (*B*) is a composite image as in Fig. 8. The lateral parts of (*A*) and (*B*) are a magnified view corresponding to the black squares at the center, as indicated by the dotted arrows. The black squares represent the linear-array ultrasound images in (*C*) and (*D*) (*left* and *right*, respectively). The dotted white lines correspond to the PLF. The dashed white lines corresponding to the MLF. The dot–dash white lines correspond to the tendon of transversus abdominus (note the *right side* in [*D*] is a *wider line* indicating the poor definition compared with the *left* in [*C*]). These lines terminate at the 3 apices of the LIFT, which is also poorly defined on the right compared with the left. The white arrow points to the part of the LIFT that appears partially torn in Video 10, a video of compression in these locations. IL, iliocostalis muscle.

Over the next month, she experienced gradual reduction in sciatic pain and increase in walking tolerance. By 6 weeks' posttreatment, her sciatica was resolved and she had no pain with sitting or driving. At 3-month follow-up, she reported that she no longer thought about using her lumbar pillow during sitting and that she was pain-free with all activities (Fig. 7C). Right single-leg squat, SLR, and hip abduction/extension strength were all normal.

Box 2
Case 2, structures injected

1. Right lateral raphe from abdominal wall to LIFT and MLF at L4-5 (Video 11)
   a. After numbing here, supine SLR improved from 35° to 45° hip flexion with less radicular pain. Patient also reported decreased pain with lumbar flexion and increased stability with right single-leg squat.
2. Right gluteus medius musculotendinous junction
3. Right posterior part of the gluteus minimus origin at the ilium
   a. After numbing structures 2 and 3, there was no change in hip abduction strength. SLR was still positive at 45° hip flexion.
4. Right gluteus medius tendon at lateral and posterior facets of the greater trochanter
   a. After numbing structure 4, hip abduction strength was 5/5 and supine SLR was negative to 90°.

Case 2 discussion

Typical examination prior to palpation-guided prolotherapy includes a detailed palpation of the sacroiliac ligaments and the enthesis of all gluteal muscles, including the iliac crest, posterior superior iliac spine, lateral sacrum, lateral ilium, and greater trochanter. If tenderness is found, injection is performed at these locations as well. Most experienced prolotherapists have resolved cases of chronic low back pain with and without sciatica by treatment of mainly hip structures. What tissue pathology is being treated in these cases?

It is well known that trigger points in gluteus medius and minimus muscles can refer to the low back, lateral thigh, lateral leg, and foot.[17] During traditional prolotherapy technique, the gluteus medius and minimus are needled extensively when injecting the lateral ilium origins. The tendons are also routinely injected at the greater trochanter. The development of ultrasonography has allowed diagnosis and injection within myofascial structures that do not include attachment to bone, such as the LIFT and gluteus medius musculotendinous junction in this case.

This case illustrates the need to address multiple structures to resolve chronic referred pain, like the concept of a double-crush injury to the nerve. The diagnosis of radiculopathy was not necessarily incorrect. The forces that created the disk herniation and potential dynamic impact to the nerve root could have resulted from the multiple soft tissue injuries she sustained prior to the development of the radiculopathy. Once both injuries were addressed, the dynamics affecting the nerve root may have changed enough for her to become asymptomatic.

SUMMARY/FUTURE DIRECTIONS

Biological structures diffuse forces in ways that are not fully understood by biomechanical theory. Biotensegrity theory argues that forces are distributed to all the tissues of the body regardless of the trauma. If accurate, this seems to open all collagen fibers of the body to the level of potential diagnoses after trauma. Increasing knowledge of fascial anatomy is advancing understanding of previously ignored connections, such as the myofascial expansions described by Stecco.[8] Thus, the limitation of tissue diagnosis to small regions within the spine, such as the disk or facets, inevitably miss some of the loss of tension caused by trauma. Refinement of diagnosis as described in these cases will allow physicians to commonly diagnose injuries to soft tissue structures, such as LIFT, PLF, Apo ES, MLF, and the seemingly endless other soft tissue structures of the spine. More thorough understanding of how forces are transferred through these tissues in normal motion and in trauma will allow more rapid diagnosis of structures currently considered unimportant. Then, it may be possible to identify common patterns of myofascial injury and degeneration that predate commonly diagnosed degenerative conditions of the spine and extremities, such as disk degeneration and facet arthropathy.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found online at https://doi.org/10.1016/j.pmr.2017.08.010.

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