Short Leg Syndrome:
A Common Cause of Low Back Pain

Kendi Hensel, DO, PhD[1] & Paul Crapo, OMS III[2]

[1]Associate Professor, Department of OMM, TCOM-UNTHSC
[2]TCOM-UNTHSC

Discrepancies in leg length are extremely common among the general population. Most people have few if any problems from a leg length inequality, but for some it can cause low back pain and other symptoms that are collectively termed Short Leg Syndrome. Low back pain stemming from Short Leg Syndrome is a common presentation that is missed too often in emergency departments and primary care clinics, because its prevalence and diagnostic findings are not well known by these providers. This paper reports a case of a 27 year-old Caucasian male with acute low back pain who first presented to an emergency department and was subsequently seen in a primary care clinic three times before being correctly diagnosed with Short Leg Syndrome. The prevalence and pathophysiology of Short Leg Syndrome are discussed. Obstacles hindering practitioners from making the correct diagnosis and solutions to those obstacles, such as encouraging the uniquely positioned Osteopathic community to assist in these efforts, are also discussed.

INTRODUCTION

A short leg, or limb length inequality (LLI), can occur because one leg is anatomically shorter than the other (an anatomic LLI), or occur because of somatic dysfunction (a functional LLI). Astoundingly, the estimated prevalence of anatomic LLIs nears 90%,[1] according to a 2005 review that compared research from 1970 to 2005 using Medline, CINAHL, and MANTIS databases. Not only did the author find that the prevalence of anatomic LLIs nears 90%, but that the average LLI is 5.22 millimeters (mm), that 14.8% of people have a LLI greater than 10 mm, and 2.6% have a LLI greater than 20 mm.[1] These figures are similar to statistics cited in a 1983 article comparing patients with chronic low back pain to symptom-free patients. In the symptom-free group, 15.6% had a LLI of 10 mm or more, and 2.2% had a LLI of 20 mm or more.[2] But the research on prevalence fails to answer the question: how large does a LLI have to be to cause symptoms? The data is conflicting on this point. Some articles say a LLI must reach approximately 5 mm before clinically apparent symptoms are produced, others 11 mm, and still others say as high as 30 mm.[2,3,4] In truth, it is unlikely that symptoms develop at a specific asymmetry threshold. Rather, symptoms can be absent or present in patients with a 5 mm LLI or 30 mm LLI, and the presence of symptoms is more likely related to how much time the patient spends standing or walking and the vigor of their physical activity.[1,2] Regardless, in a typical population where 90% of people have some degree of anatomic LLI, and when 50% of these LLIs are 5.22 mm or greater, it is prudent for practitioners to keep Short Leg Syndrome (SLS) high on their differential for patients presenting with low back pain (LBP).

SLS can be defined as an anatomic LLI that causes symptoms. Symptoms commonly include LBP, but may also include a shooting pain down the leg (sciatica), as well as pain over the sacroiliac joint, hip, outer thigh, knee, shin, ankle, and plantar fascia stemming from sacroiliac joint strain, greater trochanteric bursitis, iliotibial band strain, chondromalacia, shin splints, medial ankle synovitis, and medial plantar fasciitis respectively. Asymmetric landmarks involving the medial malleoli, anterior superior iliac spines (ASIS), posterior superior iliac spines (PSIS), iliac crests, and lumbar vertebrae almost invariably accompany SLS. The following predictable physical exam findings are found on the side with the shorter leg: 1.) superior medial malleolus, 2.) inferior ASIS, 3.) superior PSIS, 4.) inferior iliac crest, and 5.) contralateral side bending and ipsilateral rotation of the lumbar spine (Table 1).[5,6,7,8,9] Opposite findings are found on the contralateral side of the LLI. With the exception of the medial malleoli, these changes occur in an effort to equalize the length of both legs. The innominate on the side of the LLI rotates anteriorly, thus lengthening the short leg, while the innominate on the contralateral side of the LLI rotates posteriorly, shortening the longer leg. The sacral base tilts toward the side with the LLI, dropping the iliac crest on the same side, causing the lumbar spine to side bend away and rotate toward the side with the short leg.

The diagnosis of SLS is clinical. Physicians should look for the above physical exam findings in patients with LBP, or other symptoms suspicious of SLS. However, caution is advised to avoid misdiagnosing SLS in a patient with a functional LLI. A functional LLI is an appreciable leg length discrepancy caused by somatic dysfunction that is usually the result of poor lower limb mechanics, such as excessive foot pronation. Not surprisingly, limb lengths equalize once the functional LLI is resolved, assuming leg lengths were equal in the first place. With the exception of the medial malleoli, exam findings on a functional LLI will be exactly opposite to that found in an anatomic LLI (Figure 1).[10] Treatment for functional LLIs

varies depending on what caused the dysfunction. The treatment for a functional LLI stemming from excessive foot pronation is custom orthotics that corrects lower limb mechanics.

The best method to quantify anatomic LLIs is controversial. Commonly, practitioners measure from the medial malleoli of the short leg to its corresponding ASIS. However, due to disproportionate compensatory changes from patient to patient, this measurement has been criticized as inaccurate.[11,12] A second method is to measure the entirety of both limbs with plain radiographs, called a scanogram. Most current literature cites this as the most accurate method.[2] A third method used by the osteopathic and podiatric communities is to measure the declination of the base of the sacrum using radiography.[13]

Once the diagnosis is confirmed, various approaches exist to treat SLS, but all involve equalizing leg lengths through one method or another. The most common method is the use of a heel lift. Initial therapeutic heel lift size is also a matter of some debate. Some literature suggests starting with either a 1/16 or 1/8-inch lift depending on the health and age of the patient.[14] Other articles recommend starting out with a heel lift half the size of the LLI.[14] In either case, a patient typically begins wearing heel lifts smaller than the LLI itself to give the body time to decompensate. Patients then wear progressively larger heel lifts until the lift reaches the size of the LLI. Surgical approaches are reserved for severe cases, and include techniques such as the Ilizarov distraction method and subtrochanteric femoral shortening osteotomy.[15,16]

PATIENT PRESENTATION

A 27-year-old Caucasian male presented to the emergency department complaining of LBP. In the patient's history of present illness (HPI), he stated the pain had been present for four years, but that two months ago it worsened after he started working at a department store lifting and storing merchandise in the back of the store. The pain was sharp with no radiation, numbness, tingling, urinary retention, incontinence, bowel problems, or diaphoresis. He rated the pain a five out of ten. The symptoms were aggravated by movement and relieved by rest. Plain radiographs were taken of the patient's back which were negative for any acute bony abnormality. The patient was discharged home on cyclobenzaprine and meloxicam and told to follow up with a local resident clinic in one week.

At the resident clinic the next week, the patient's pain was still unrelenting. He was taught stretching exercises, given ibuprofen to replace meloxicam, and told to follow up in one week.

A week later, the patient's pain was still not better, despite being compliant with the exercises and medications. Ibuprofen was continued, cyclobenzaprine was discontinued, and nabumatone was added. Plain lumbar radiographs were re-ordered, a urinalysis was performed, and blood tests were taken. The patient was told to follow up in one week to discuss the results.

Another week later, the patient's symptoms were still present, matching the HPI recorded in the emergency department, except his pain was less severe. His past medical history consisted of chronic low back pain. His past surgical history was positive for a tumor removal from his right knee. His medications included ibuprofen 800 mg twice daily and nabumetone 500 mg twice daily. He denied knowledge of any drug allergies. The patient had recently quit smoking. His family history was negative. A review of systems

Physical Exam Findings in Short Leg Syndrome

Anatomic Landmarks (on the side of the short leg)	Physical Exam Findings
Medial Malleolus	Superior
Anterior Superior Iliac Spine (ASIS)	Inferior
Posterior Superior Iliac Spine (PSIS)	Superior
Iliac Crest	Inferior
Lumbar Spine	Contralateral side bending, ipsilateral rotation

FIGURE 1:
Innominate Changes in Anatomic & Functional Limb Length Inequalities
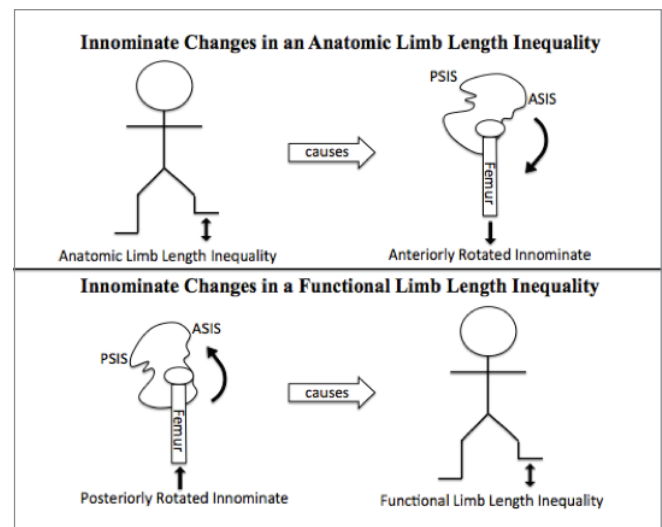
Innominate changes seen in limb length inequalities. The top part of the figure shows how the innominate rotates anteriorly to compensate for an anatomic limb length inequality. The bottom part of the figure shows how somatic dysfunction producing a posteriorly rotated innominate can cause a functional limb length inequality.

was unremarkable except for that noted in the HPI. The physical exam showed normal sensation, motor function, gait, stance, and reflexes. A standing and supine osteopathic structural exam was performed which showed the following: tender to palpation lumbar paraspinal muscles, a superior left medial malleolus, an inferior left ASIS, a superior left PSIS, an inferior left iliac crest, and side-bent right rotated left lumbar vertebrae from L2 to L5. The patient's pubic symphysis was then gapped and landmarks were rechecked. His medial malleoli were almost symmetric, but the rest of the findings were nearly identical. Lumbar soft tissue and HVLA were performed and the patient felt slightly better. The lumbar radiographs, urinalysis, and blood tests were all negative, and they were discussed with the patient. At this point, heel lifts were not initiated. The patient was told to follow up in one month.

DISCUSSION

Several points regarding the treatment of this patient merit discussion. First, each of the doctors involved likely did not have SLS high on their differential diagnosis when they encountered this patient with low back pain. More than 85% of patients who present to primary care with LBP have a non-emergent and nonspecific cause,[17] the vast majority of which come from some type of strain on the structures that comprise the back itself (muscles, ligaments, tendons, disks, etc.).[18] Although it is unknown what percent of patients have low back pain due to SLS, knowing that the prevalence of LLIs in the general population is 90%, with a mean of 5.22 mm, and that clinical symptoms can be present in patients with LLIs as low as 5 mm,[1] should cause providers to place SLS high on their differential diagnosis for a patient with LBP. Had this been the case, the patient might have been diagnosed earlier. Second, it took almost one month before at least a sufficient structural exam was performed on the patient. Ideally, the patient would have been evaluated with a structural exam at his first presentation. The structural exam should have at least included a standing forward flexion test, and an evaluation of the medial malleoli lengths, ASISs, PSISs, iliac crests, and lumbar spine. For the reader's benefit, an additional section at the end of the article has been added on how to perform an adequate structural exam *(see Appendix, page 28)*.

Performing a good structural exam on a patient with LBP is analogous to performing a good cardiovascular exam on a patient with chest pain. Third, the practitioners involved were probably ill-equipped to correctly diagnose SLS. Most doctors have never been taught what physical exam findings to look for in SLS *(Table 1)*. Put together, these three aspects regarding this patient's several doctor-patient encounters make it easy to understand why a patient with SLS might be misdiagnosed. However, had the patient been diagnosed earlier, even in the emergency department, the correct treatment plan could have been initiated, and the interim pain, time, and money spent could have been avoided or minimized. The exact costs that SLS incurs on society are not known, but can be surmised when considered in the context of LBP. For LBP, 90 billion dollars of healthcare related expenses were spent nationally in 2010 (without factoring in lost opportunity cost such as days missed at work).[19] Additionally, of 291 conditions considered in the Global Burden of Disease 2010 Study, LBP ranked first in years lived with disability (YLDs) and sixth in the total burden of disease (Disability Adjusted Life Years or DALYs).[20] It seems reasonable to assume that if providers worldwide knew how to appropriately diagnose SLS, at least a small proportion of these costs would be reduced, given the prevalence and magnitude of anatomic LLIs in the general population.

CONCLUSION

SLS is an often missed diagnosis of LBP by providers. This can change if providers adequately educate themselves about the syndrome. On what specifically should they educate themselves? On two items: 1.) the prevalence of anatomic LLIs in the general population, and 2.) how to diagnose SLS from its characteristic physical exam findings. Regarding the prevalence, providers should remember that of the nonspecific 85% of LBP they will see, a substantial proportion will likely be due to SLS, given that 90% of the general population has an anatomic LLI, with a mean of 5.22 mm, and that clinical symptoms can be present in patients with LLIs as low as 5 mm. Regarding the diagnosis of SLS, providers should remember to perform adequate structural exams that at least include a standing forward flexion test, an evaluation of the medial malleoli lengths, the ASISs, PSISs, iliac crests, and the lumbar spine to determine if the following pattern of landmarks is appreciated on the side of the short leg: 1.) superior medial malleolus, 2.) inferior ASIS, 3.) superior PSIS, 4.) inferior iliac crest, and 5.) contralateral side bending and ipsilateral rotation of the lumbar spine. Additionally, disseminating this information to change SLS from being a commonly missed diagnosis of LBP to a common diagnosis of LBP will take considerable effort. The Osteopathic community is uniquely situated to help. Consider their focused education and diagnostic training in musculoskeletal complaints. Given these characteristics, they likely have the greatest potential to educate their colleagues across the nation on how to correctly diagnose SLS, thus, responsibility falls largely on their shoulders. Further research should be done to determine the proportion of patients with LBP that stems from SLS, the financial and other societal costs on the general population due specifically to SLS, and effective ways at disseminating knowledge on how to diagnose SLS effectively to the general medical community.

APPENDIX:

HOW TO PERFORM AN OSTEOPATHIC STRUCTURAL EXAM

Performing a structural exam on patients is critical to diagnose and treat them correctly, yet many providers do not remember how to perform one adequately. The purpose of this appendix is to teach clinicians how to perform a simple yet thorough osteopathic structural exam. Content contained herein has been summarized from the second edition of *The Atlas of Osteopathic Techniques*, by Alexander S. Nicholas and Evan A. Nicholas, 2012. Generally, a structural exam should include the following four main components, normally performed in the order provided:

    I. Osteopathic Static Musculoskeletal Examination

    II. Spinal Regional Range of Motion Testing

    III. Osteopathic Layer-by-Layer Palpation

    IV. Intersegmental Motion Testing

I. Osteopathic Static Musculoskeletal Examination

The goal of the static portion of the exam is to determine potential somatic dysfunction by identifying obvious structural asymmetries. To perform the static exam, visualize the patient from the anterior, posterior, and lateral views; then determine landmarks and compare symmetry.

Note or compare the following major landmarks from the ANTERIOR VIEW:

- Midgravitational line
- Head position in relation to shoulders and body
- Levelness of eyebrows
- Levelness of eyes
- Deviation of nasal bones and/or nose
- Angles of mouth
- Deviation of mentum
- Levelness of shoulders
- Depth of shoulders (anteroposterior relation)
- Thoracic symmetry
- Iliac crests
- Rotation of anterior superior iliac spine (ASIS)
- Levelness of patellae
- Pronation or supination of feet

Note or compare the following major landmarks from the POSTERIOR VIEW:

- Midgravitational line
- Head position in relation to shoulders and body
- Mastoid processes
- Neck to shoulder angles
- Levelness of shoulders
- Depth of shoulders (anteroposterior relation)
- Position of scapulae
- Erector spinae muscle prominence(s)
- Levelness of iliac crests
- Rotation of posterior superior iliac spine (PSIS)
- Levelness of greater trochanters
- Achilles tendons shape

Note or compare the following major landmarks from the LATERAL VIEW:

- Lateral midgravitational line connecting:
    - External auditory canal
    - Lateral head of humerus
    - Third lumbar vertebra
    - Anterior third of the sacrum
    - Greater trochanter of the hip
    - Lateral condyle of the knee
    - Lateral malleolus
- Head position in relation to shoulders and body
- Sternal angle
- Lordosis of cervical spine
- Kyphosis of thoracic spine
- Lordosis of lumbar spine
- Lumbosacral angle
- Flexion or extension of hips
- Flexion or extension of knees
- Arch of feet

II. Spinal Regional Range of Motion Testing

The purpose of spinal regional range of motion testing is to determine potential somatic dysfunction in the components of the body that cause motion around the cardinal axes of motion (flexion, extension, side bending, and rotation). To perform this section of the exam, physicians should test active and passive range of motion in the cervical, thoracic, and lumbar spine in flexion, extension, side bending, and rotation. It is easiest to test the cervical and thoracic spine while the patient is seated and the lumbar spine while the patient is standing. Examiners should look for asymmetries when comparing left and right, as well as any significant increase or decrease in range of motion when compared to normal range of motion values. Importantly, normal range of motion values vary depending on the source (in some cases significantly), therefore, it is incumbent for physicians to use their clinical judgment in deciding whether an increase or decrease in range of motion represents somatic dysfunction or a healthy patient with an acceptable outlying range of motion value. Clinicians desiring specific values may reference chapter three in the second edition of *The Atlas of Osteopathic Techniques* (2012), which compares three different sources for normal spinal range of motion values.

III. Osteopathic Layer-by-Layer Palpation

The layer-by layer examination has eight components, which are:

1. Observation
2. Temperature
3. Skin topography and texture
4. Fascia
5. Muscle
6. Tendon
7. Ligament
8. Erythema friction rub

1. Observation

Before touching the patient, visualize the area being examined first for signs of somatic dysfunction. See if there are any visual signs of trauma, erythema, swelling, fullness, diaphoresis, abnormal hair patterns, nevi, follicular eruptions, etc.

2. Temperature

Metabolic changes from trauma, infection, or even chronic fibrotic effects may generate heat which can be sensed with the wrists or hands. To evaluate, place either the volar aspect of the wrist or dorsal aspect of the hypothenar eminence of the hand a couple inches above the skin being tested. Do this over the area of interest and over the paraspinal areas.

3. Skin topography and texture

Somatic dysfunction may cause an increase or decrease in the humidity, oiliness, thickening, roughening, etc. of the skin. This can be sensed as the pads of the fingers are applied to the area being examined, light enough that the fingernail beds do not blanch.

4. Fascia

Fascia may "bind" or tighten when somatic dysfunction is present. To sense this, place the hands over the area to be tested and apply just enough pressure that the fingernail beds blanch. Move the hands superiorly, inferiorly, left, right, clockwise, and counter-clockwise, to evaluate for areas of ease or restriction.

5. Muscle

Acute and chronic muscle injuries may cause somatic dysfunction that can be deduced through palpation. Acute changes have a boggy feeling overlying the muscle, while the muscle itself may feel like it is contracted, rigid, or hard. Chronic changes feel ropey or stringy. To determine, place the hands over the area to be examined and apply pressure deeper than that applied to the fascia.

6. Tendons

Damaged tendons may undergo fibrous thickening, or changes in their elasticity. Palpate tendons from their bony attachments to their continuation with the muscle belly.

7. Ligaments

Ligaments can cause somatic dysfunction by being too lax, causing joint laxity, or too tight, causing joint restriction. Some ligaments are more amenable to palpation than others. If able, palpate ligaments in the area of concern.

8. Erythema friction rub

The purpose of this test is to discover paraspinal areas with autonomic changes that cause segmental dysfunction. To perform, place the pads of the second and third digits over the paraspinals and stroke downward two to three times. Evaluate for redness at each spinal segment.

IV. Intersegmental Motion Testing

Intersegmental motion testing refers to articulatory motion in the spinal facets or at any joint. Most often, the goal of motion testing is to obtain a specific diagnosis. In some instances, a specific diagnosis is unable to be ascertained (such as the standing flexion test). Providers should focus motion testing not only on the area of the patient's complaint, but also on other areas where the body may be compensating for the original somatic dysfunction. Intersegmental motion testing includes tests that aid in the diagnosis of Short Leg Syndrome and leg length inequalities such as individual lumbar spinal segment motion testing and the standing flexion test. The details on spinal motion testing as well as other joint motion testing is beyond the scope of this appendix. Those who desire to know more may reference chapter five of the second edition of *The Atlas of Osteopathic Techniques*, 2012.

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