Editorial Commentary: The Pelvis is the Lowest Vertebral Level: Diagnostic Approach to Hip-Spine Syndrome

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Abstract: The human pelvis represents a wonderful example of apparent idealistic simplicity overwhelmed by realistic complexity. Traditionally, the pelvis has been termed a "ring" linking the lower extremity to the spine via the sacroiliac joint. In essence, the pelvis is the lowest vertebral level—"the hip bone's connected to the spine bone." Thus, the law of parsimony seemingly applies in the diagnosis and management of both arthritic and nonarthritic hip and spine disorders in isolation or combination. However, an inverse Occam's razor is much more likely. The layered theory of hip disorders illustrates how a base osteochondral layer (femoroacetabular impingement syndrome, ischiofemoral impingement from either the lesser trochanter or greater trochanter, arthritis), a static inert soft-tissue layer (labrum, capsule, ligament), a dynamic soft-tissue layer (muscle, tendon), and a neurokinetic chain layer all interact and can lead to hundreds, if not thousands, of different combinations of primary and secondary symptom sources. Although correlation does not equal causation, intuitively and overly simplistically, a stiff painful hip can transfer stress across the pelvic ring to the spine, causing back pain. Alternatively, 2 separate symptom sources could be present at the same time. Biomechanical stress transfer can occur from flexion-based (e.g., femoroacetabular impingement syndrome) or extension-based (e.g., ischiofemoral impingement) problems. The diagnosis of hip-spine syndrome in patients becomes really complicated usually really fast, encompassing the hip joint, peritrochanteric space, deep gluteal space, pelvis and pelvic floor, sacroiliac joint, and lumbosacral spine—and don't forget mental health and the mind controls the musculotendinous system in these challenging, often frustrated, patients. Static imaging findings necessitate dynamic symptom correlation, especially via pertinent values including pelvic incidence; pelvic tilt; sacral slope; lumbar lordosis; femoral and acetabular version; cam, pincer, and dysplastic morphologies; and leg length. Judicious diagnostic injections can greatly assist in clinical symptom interpretation. Successful treatment requires consideration and management of the primary etiology and pertinent secondary downstream effects. When a patient's hip hurts, one should always look at the patient's back; when a patient's back hurts, one should always look at the patient's hip.

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The pelvis is the bony linkage connecting the spine and the lower extremities. In fact, the pelvis can be thought of as the lowest vertebral level, locked in place by a quantitative value known as the pelvic incidence (PI).[1] The PI represents the fixed, position-independent sum of sacral slope and pelvic tilt. Although these latter 2 values are position dependent, the unchanging PI has a significant impact on symptoms in patients with hip pain and those undergoing hip preservation surgery. Because the pelvis is an osseous ring, biomechanical abnormalities at the hip joint, such as cam morphology associated with femoroacetabular impingement syndrome, may transfer stress across this ring to the lumbosacral spine. Similarly, whereas a number of different types of extra-articular impingement have been studied, ischiofemoral impingement (IFI) has also exhibited significant stress transfer through the ring to the lumbosacral spine.[2] Conversely, spine etiologies may transfer stress to the hip and lower extremity. Most

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of the literature discussing this hip-spine syndrome[3] has concerned the instability risk after total hip arthroplasty (THA), with minimal representation of patients with nonarthritic, non-THA hips. This is why "Hip-Spine Syndrome in the Nonarthritic Patient," by Vaswani, White, Feingold, and Ranawat,[4] is such a valuable contribution to the literature.

The term "hip-spine syndrome" was first used in a 1983 publication by Offierski and MacNab[3] to describe concomitant degenerative changes in both the hip joint and lumbar spine. Although "the chicken or the egg" analogy comes to mind, we must remember that "correlation does not equal causation."[1] The hip pathology may cause the spine problem, or vice versa. However, it could simply be 2 completely separate, non-causative things going on at the same time. Intuitively, the causation theory makes sense: A stiff, painful arthritic hip is coupled with the pelvis, which is the bottom vertebral level, and attempted motion at the hip may necessitate adjacent joint and/or vertebral level motion to compensate for the hip stiffness. Now, if we apply that concept to the nonarthritic hip, one can easily reason that hip stiffness conditions may subsequently transfer stress to the spine. Both cam morphology and pincer morphology mechanical blockages to further hip motion that will transfer pelvic stress across the sacroiliac joint into the lumbosacral spine. I have had several AANA educational opportunities at the Orthopaedic Learning Center in which cadaveric hip motion was investigated with the intent of observing stress transfer to the spine. It is blatantly obvious that even in hips without cam or pincer morphology, if one takes the hip far enough, it can impinge.[5-7] If it can impinge, it can transfer stress to the spine.

Nonarthritic cam and pincer lesions are primarily sagittal-plane, flexion-based pathomorphologies with resultant mechanical compression in the deep anterior pelvis transferring stress posteriorly. On the contrary, nonarthritic extension-based pathologies (e.g., IFI—lesser trochanter, excessive femoral anteversion, overtightened iliofemoral ligament capsular plication, narrow-offset THA, reduced offset after hip fracture fixation) result in mechanical tension in the deep anterior pelvis transferring stress posteriorly. Osteoarthritis of the hip, with a flexion contracture due to the anterior capsule and/or iliopsoas, may result in mechanical tension transferring stress posteriorly. Mechanical tension in the front of the pelvis results in premature coupling, locking the leg to the pelvis. Thus, any attempt to extend the hip beyond this tension threshold leads to secondary downstream compensatory pathology.

Loss of hip extension presents a challenging diagnosis and management, with multiple different primary and secondary downstream concurrent symptom sources. This leads to an incredibly complex patient presentation, often with frustrated individuals who have seen multiple doctors, have undergone multiple diagnostic tests, have tried multiple therapeutic interventions (including injections and surgical procedures), and spent significant sums of money with sometimes prohibitive financial investments into their health care, to no successful avail. This is analogous to the circuitous route patients used to take to reach a femoroacetabular impingement syndrome diagnosis, based on data published over 10 years ago.[8] Some of these patients will present to the office never having heard of IFI before, despite the obvious apparent diagnosis based on the history, examination, and imaging. Some patients will present to the office with a chief complaint of IFI on a magnetic resonance imaging scan, but their symptoms and examination are clearly something else. We must remember that one should treat patients not magnetic resonance imaging scans—this helps avoid VOMIT (victims of modern imaging technology) and BARF (brainless application of radiographic findings).[9] This is especially important when treating the posterior hip with IFI owing to the high prevalence of IFI (84%) in cadaveric specimens.[10] The skilled clinician should be able to recognize the proverbial "incidentaloma" and avoid an unnecessary workup.[11] Awareness is improving; diagnostic workup is improving; and treatments are in their infancy but are multifaceted and improving.

In their article, Vaswani, White, Feingold, and Ranawat[4] do a nice job discussing PI within the context of the relation between the hip and spinopelvic alignment. The low PI pelvis exhibits loss of lumbar lordosis, requiring anterior pelvic tilt to maintain sagittal balance, which can increase impingement symptoms. The low PI prevents any posterior pelvic tilt to open the acetabular cup and maintain sagittal balance. This may be seen in hip arthroscopy patients in whom impingement still occurs after a correct cam or pincer lesion correction. This may be seen in patients without any impingement morphology at all. The high PI pelvis has excessive lumbar lordosis, requiring posterior pelvic tilt to maintain sagittal balance, which can improve impingement symptoms. This may be the reason that some patients, including nonarthritic elite athletes, with cam morphology and labral injury may be asymptomatic[12] or minimally symptomatic in youth and even into later years.[13] Thus, hip and spine surgeons should be cognizant of this important relation, but "if you don't measure it, you can't manage it." A lateral sacral plain radiograph, EOS biplanar fluoroscopy, or the scout view from a pelvic computed tomography scan will yield this simple, but essential, value. The "take-home point" of the article by Vaswani, White, Feingold, and Ranawat[4] should be this: When one thinks of the hip, one should also always think of the spine; when one thinks of the spine, one should also always think of the hip.

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