

## Anterior Femoral Glide Syndrome

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I am not a chiropractic philosophy writer, but once a year my editor cuts me a little slack. If I am a good chiropractor, it's because of my failures. I learn the most from the patients who I couldn't help initially: the tough cases, the ones in which I wrack my brain to figure out what I am missing.

These tough cases, the ones that require you to think out of the box, are the ones that push you, if you are willing. It's easy to just keep adjusting them, work out the same trigger points, and convince yourself that one more or 10 more treatments will solve the problem, but that doesn't usually work. A recent study tells us that we can predict by the second visit which patients will respond best, at least in cases of lower back pain.<sup>1</sup> The study implies to me that when the patient is not improving quickly, you have to look with new eyes. What I would suggest is that you go back, re-examine them and think of the whole kinetic chain, maybe even the patient's biochemistry and underlying emotional pieces.

At the bare minimum, I believe we need to be experts not only at manipulation, but also at addressing soft tissue and rehabilitation. If the goal is to restore more normal motion, thus improving function and reducing pain, all three of these pieces are necessary.

An accurate musculoskeletal diagnosis includes not only what joint is not moving, but also what joint is moving too much, what specific tissues are the pain generators, and what movement or lack of movement is stressing the pain generator. Shirley Sahrmann states, "A joint develops a directional susceptibility to movement, which then becomes the 'weak link' and most often the cause of pain."<sup>2</sup>

We have to become more evidence-based. At the same time, we shouldn't become handcuffed to what is absolutely certain. Musculoskeletal research is difficult to do, and its particularly difficult to look at the big picture: the integration of the multiple factors that can contribute to ongoing or recurrent pain syndromes. As Craig Liebenson states, "Lack of evidence of effectiveness is not the same as evidence of ineffectiveness. According to Lewit, we work at the level of acceptable uncertainty."<sup>3</sup>

Many of you have followed my interest in the hip joint. I am grateful for the contribution of Lucy Whyte Ferguson, DC.<sup>4</sup> Even using trigger-point work and manipulation of the hip with the wishbone maneuver, as outlined by Dr. Whyte Ferguson, I found too many patients' hip problems recurring, and too many for whom I could not consistently restore normal motion.

I was introduced to a model from Sahrmann that made sense of the two patterns I have seen in the hip. She calls it the anterior femoral glide syndrome, and mentions an internally rotated version and an externally rotated version. Sahrmann's model talks about what accessory movements are dysfunctional. In anterior femoral glide syndrome, the proximal femur moves improperly during hip flexion. Instead of gliding posterior to provide room for the flexion, it glides anterior, jamming into the anterior hip capsule and causing pain and limitation of flexion. What causes this? Sahrmann talks about familiar muscular imbalances. The hamstrings are too tight and are not balanced by the

gluteus maximus. During hip extension, the hamstrings create a bowstring effect, pushing the femoral head forward. The posterior structures around the hip are too tight, contributing to the anterior motion. The psoas is weak and long, allowing the forward motion and not stabilizing the hip up into its socket. This view of the psoas is consistent with Sean Gibbon's point of view. He sees the psoas as a local and global stabilizer, likely to be inhibited.

Sahrmann looks at two versions of this hip joint problem. In the first, which she describes as more common, the femur tends to be medially rotated. This tends to occur more in females and goes with genu valgum, pronation, and anteversion of the hip. In this pattern, there is a clear dominance of the tensor fascia latae (TFL) over the gluteus medius, thus pulling the hip into medial rotation. The hip external rotators are likely to be weak. The medial hamstrings are dominant over the lateral hamstrings. Whenever the patient stands on the affected leg, the hip is internally rotated; the hip rotates more easily into internal rotation than external rotation. If you have the patient step up onto a step, you'll see a sudden medial rotation motion at the knee on the involved side.

The second version is the one I am more familiar with, perhaps because it is my own pattern. It involves an externally rotated femur which lacks medial rotation. It's more of a male problem, and is consistent with a more rigid overall structure, one that may include a supinated foot. Sahrmann states that the groin pain is more medially located in these patients. These are the patients for whom the wishbone maneuver, a mobilization with eccentric muscular activity, seems to be most effective.<sup>5</sup>

In both of these anterior femur problems, the pain is likely to start in the groin and then spread to the whole hip. These patterns can be the missing link in lumbar and sacroiliac conditions, as well as with lower extremity problems.

Psoas, Psoas, Psoas

Weakness of the psoas is an important factor in these anterior hip patterns. Sahrmann talks about this, but her book is seven years old now, and there is new research and new methods to rehab the psoas. I recommend you read Sean Gibbons' long article on the psoas.<sup>6</sup> I used to think of the psoas as an overactive hip flexor. I now think of the psoas as an inhibited lumbar spine and hip stabilizer. If the psoas is not able to contract in a timely manner, the femoral head will drop anterior and lateral, jamming into the hip capsule. Psoas weakness also plays into lumbar instability patterns. Gibbons' model for testing the psoas involves testing lumbar stability and seeing if psoas contraction can change the palpatory feel of lumbar hypermobility. I suspect that we can test the psoas in a different way, testing the effects of psoas weakness on the insertion rather than the origin.

Here is my hypothesis about another way to test the psoas. If the patient has a hard time flexing the involved hip, teach them to contract the psoas, ideally both supine and side-lying. Have them fire the psoas for a few repetitions, correcting for substitution and not overfiring. Local stability exercises often feel so mild that the patient wants to work too hard. To understand this model of how to rehab the psoas, see my description below, Gibbons' article, and/or print the handout from my Web site.<sup>7</sup>

First, the patient has to understand the exercise and be able to do it at least close to correctly. Then have them hold this mild psoas contraction supine, and simultaneously flex the hip, either passively or actively. If this makes the hip easier to flex, with less of a groin pinch, it shows that the psoas is dysfunctional, and needs rehab. I love to show my patients exercises that make an immediate difference in their symptoms or signs. It's the best rehab motivator I know.

The basic exercises to retrain or recruit the psoas are deceptively simple. Suck the hip gently up into the socket, primarily using the psoas. The doctor should initially provide a gentle traction down the long axis of the hip to increase proprioception. Here's the tricky part: You have to suck the femur up into its socket without hiking the hip (which indicates overactivity of the iliocostalis lumborum and/or the quadratus lumborum). You have to activate the psoas without overactivating the TFL and/or the rectus femoris.

While lying supine, raising the upper body up onto the elbows may help take out the hip hikers. Externally rotating the thigh may help take out the TFL and/or rectus femoris. It's OK initially to fire the rest of the inner core while activating this, using the pelvic floor (Kegel), the lower abs and the multifidi. The local stabilizers are all going to inherently co-contract.

The goal, ideally, is to isolate the psoas as much as possible. Another good position for psoas rehab is side-lying, drawing the hip into the socket. In this position, the key is to control pelvic rotation. Personally, I found these exercises somewhat difficult to learn but rewarding. Once you have the basic motion down, you can integrate it into more global hip flexion.

### Another Hip Mobilization Method

One aspect of this problem is that as the distal part of the femur flexes, the proximal convex femur head in the concave acetabulum has to rotate and glide in an inferior direction. If this doesn't happen, you get that feeling of jamming in the groin. One way to correct this is via the Mulligan concept.<sup>8</sup> Mulligan's model basically says to find a direction of passive pressure (applied by either the doctor or the patient) that allows more joint motion. Repeat that assisted motion over and over to reset the neuromuscular system.

In this case, a superior-to-inferior pressure on the proximal hip usually allows for easier hip flexion. You can provide this with manual pressure; use a wide belt to pull the proximal hip inferior. You can follow this up with home self-mobilization procedures. The patient can use the heel of their own hands, pressing inferior while they lift the leg. The patient also can use a belt, strapped from the involved groin down to the opposite foot, to provide the same superior-to-inferior pressure while flexing the hip.

These simple techniques often have dramatic benefit. Try them out. Have fun integrating them into your practice; your patients will thank you for it.

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