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Peritrochanteric Hip Pain: An Interventional Orthopedic Medicine Approach

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INTRODUCTION:

I spent my entire professional career pondering and studying this phenomenon of the "peritrochanteric hip pain syndrome". Many years ago prior to attending medical school and practicing as a chiropractor and overseeing an orthopedic rehabilitation program we tried countless therapy and exercise protocols which includes multiple physical therapy modalities, electrical muscle stimulation, ultrasound, ice, iontophoresis, stretching, strengthening, etc. For the most part these methods failed and patients continued to suffer with this persistent lateral hip pain. In the early 1990s we began to use steroid injections in the various bursa of the hip and combined this with our rehabilitation and therapy programs. The steroid injections provided significant temporary relief, but the symptoms always returned. Frustrated with the clinical outcomes of our treatment our group decided to begin doing biomechanical studies on this syndrome and try to get to the root of the problem. Fortunately, we had a biomechanical and gait lab that allowed us to complete some sophisticated studies on these patients.

Our first discovery was a common biomechanical fault that most individuals have; that we identified that is probably the most common source of this problem. In the early 1990s we also began to experiment with various orthopedic regenerative medicine strategies, which we will also discuss within the context of this article. Over time our understanding of this condition improved as well as the technological advancements of regenerative medicine, until today we have a host of options for management of this rather complex problem. This has taken 25 years of clinical experience and thousands of empirical trials of treatment to come to the level of understanding we have today. Despite this and despite all of our frequent success with treating this condition there are still patients that we encounter that present significant challenges. There will be some proprietary technologies that we utilize in difficult cases that we have not published and therefore we will withhold discussion of some of the more "higher tech" methods that can be used to treat this condition. However I will share the basic principles that we have learned in regards to cause, evaluation and management of this most difficult condition.

BASIC HIP ANATOMY… WHERE IS THE PAIN COMING FROM?

We begin our discussion with a brief review of hip anatomy. It is important to point out that the hip joint is a ball and socket joint and the top of the femur is capped with an important hyalin cartilage, colored in pink in the picture to the right. This cartilage is of critical importance. It is the erosion of this articular cartilage that heralds the arthritic condition of the hip. The femur has large bony protuberances utilized for powerful muscle attachment. The largest of these bony protuberances is the greater trochanter which is labeled in the picture to the left.

The greater trochanter is actually a very important part of hip anatomy and is at the heart of this discussion. The trochanter is a large bony prominence upon which important muscles and tendons attach. Some of the various muscles attaching to the greater trochanter are seen in the picture to the right. I commonly reference these various muscles attaching onto the greater trochanter as the "HIP ROTATOR CUFF." There are many similar functions to this rotator cuff as there is in the shoulder rotator cuff. There are also of course distinct differences as well.

WHAT IS A HIP BURSA?
As one can see in the picture to the right there are multiple muscles that attach onto or around the greater trochanter. Some of these muscles generate tremendous

force and when injured or inflamed can cause significant pain and disability. The hip joint is quite mobile and because of this mobility there can be friction between all of the muscle and tendon attachments to the hip. To eliminate this friction we have bursa dispersed between tendons or between tendons and bone to reduce friction. Imagine taking a deflated balloon and placing several drops of oil within the balloon and tying the top of the balloon off. As you now hold the balloon between your hands and rub your hands over the balloon the oil inside the balloon lubricates its inner surface and reduces friction.

This anti-friction device created by the balloon is exactly how a bursa works. A bursa is a thin sac where a small amount of lubricating fluid is contained within it. These are dispersed under tendons and between tendons and bone throughout your hip. There can be as many as 20 bursa dispersed around the hip. The picture to the right demonstrates just a few. The most important two bursa of the hip is the greater trochanteric bursa and the bursa underneath the gluteus medius tendon or better known as the gluteus medius bursa. It is common place for these two bursae to become inflamed and irritated with chronic stress to the hips soft tissues resulting in significant hip pain. It is actually much more common to have pain from the tendons, muscles and bursa of the hip than it is to have arthritis pain of the hip. Typically if I were to see 10 individuals with hip pain, 9 of those individuals would have pain from the muscles, tendons and bursa and only 1 would have pain from arthritis. It is also very common for individuals to be misdiagnosed in regards to the source of their hip pain. I have had individuals come in for a stem cell consult for hip arthritis and much to their surprise they were not in need of stem cell therapy for an arthritic hip, but had pain primarily secondary to a problem associated with the muscles, tendons and bursa around the greater trochanter. Directing the appropriate treatment improves their condition without treating any hip arthritis condition. Diagnosis is critically important. Occasionally an individual may have an underlying arthritic hip but again the pain is primarily arising from the tendons and bursa and not the arthritic hip joint. So again diagnosis is crucial

THE BIOMECHANICAL LINK TO CHRONIC PERITROCHANTERIC HIP PAIN:
It may seem onto you that this entire problem of your hip actually does not begin in the hip but rather in the foot. We were to discover that the problem begins when your foot strikes the ground. In the 1990s frustrated about the source of this problem we began to utilize our gait analysis laboratory to evaluate patients with this problem of persistent buttock and peritrochanteric hip pain. We were to find out that consistently on examination of the foot and biomechanics of gait that a consistent anatomical and biomechanical profile began to emerge which was our first real break into uncovering the cause of what we were seeing clinically.

Before I can discuss this condition and its treatment we will discuss some basic anatomy and biomechanical facts that you will need to understand.

FETAL FOOT DEVELOPMENT:
We begin this discussion actually reviewing some important information about the foot anatomy and biomechanics which is of critical importance if you want understand where your hip pain may be coming from. The little feet that we see in a new born actually do not start out like the picture noted on the right, but rather as small limb buds with the feet and toes in the vertical plane as seen in the picture to the left. During fetal development the lower leg and foot goes through a series of rotations as the limb grows. Eventually your foot becomes oriented in the horizontal plane which is the normal foot position. Many of us during fetal development never complete all of these rotations to bring the foot into the perfect horizontal plane. If your foot specifically your forefoot (the portion of your foot in front of your ankle) does not finish all of its rotations it will leave your forefoot in a slight angulated position as seen in the picture to the left. It is essential that

you understand the concept of “forefoot varus” if you ever want to get to the heart of the problem. If the foot fails to complete its full series of rotations you will end up with the forefoot in a position that is slightly tilted off the horizontal plane. If it is tilted in the plane noted in the picture to the left we call this a “forefoot varus”. As noted in the picture to the right if your forefoot excessively rotated you would end up with a forefoot in the plane of a forefoot valgus. It is extremely rare to find a foot that over rotates and develops into a “valgus position”. A common condition in many individuals foot structure is that they fail to complete all of the rotation necessary to be completely horizontal and this failure of rotation leads to a condition which we call forefoot varus. This is a very common clinical finding.

So what? Why is this foot development issue even important when discussing chronic hip pain? When you are born with a forefoot varus your forefoot in relationship to your rear foot is similar to the picture noted below on the left. But this picture does not show you how your foot would appear when you place your foot to the ground and become weight bearing. You actually become a “over pronator”. As soon as the forefoot varus becomes weight bearing your foot overpronates when you walk. This is where we began to get an understanding of how this condition initially develops.

When you walk on 2 feet with a forefoot varus you will not walk on the lateral edge of your foot but will be required to over pronate your foot and ankle in order to get your
big toe to the ground. A critical component of the human gait is to bring the toe to the ground when your foot strikes the ground. In these individuals with a “forefoot varus” they must overpronate in order to accomplish that as seen in the picture to the right. Notice in this sequence of pictures the individual on the left stands in an over pronated position while the other individual to the right stands in a neutral position. It is this over pronated position that begins the pathology as the individual walks in this position for their entire life. So what does foot pronation do to the hip? Therein lies the big question !!

As shown in the picture below an individual that overpronates excessively internally rotates the tibia (shinbone) and also excessively internally rotates the femur (the thigh bone and hip). With every step there is a subtle increase in internal rotation of the leg that alters the entire biomechanics of the human gait. This is something that goes undetected and typically individuals are completely aware of this through their life until we identify the
anomaly on clinical examination.

THE MUSCES OF THE HIP ROTATOR CUFF:

There are a whole host of muscles that begin in the pelvis and wrap around and attached to the greater trochanter of the hip. A number of these muscles are external hip rotators.
This means that if you lift your leg up and rotate your hip outwards it is the contraction of these muscles that will accomplish the task. However, the muscles that externally rotate the hip do not function as external hip rotators during the human bipedal gait. In fact they do something quite different. They instead function to slow down or “break” excessive internal rotation! In other words during the time when you bring your body weight over your foot and the foot overpronates the tibia and femur excessively internally rotate and these muscles must lengthen and contract to slow this movement down. We call this type of muscle contraction an eccentric contraction. In an eccentric contraction the muscle is contracting and lengthening to resist a load. A concentric contraction would be just the opposite where the muscle is shortening to move a load like a biceps curl.

There are a number of muscles involved that are at play. There are 2 important muscles that we will emphasize during this discussion. One of the critical muscles we need to focus on is the gluteus medius muscle. Although many of us that have studied anatomy know the gluteus medius is a muscle that is a hip abductor (elevating the hip away from your body). In fact this occurs from only a portion of the muscle. If you look at the muscle carefully noted in the

picture to the left there seems to be 2 parts of the muscle. The portion more anterior or towards the front is the portion of the muscle that abducts the hip. But as you move more posterior you see a more fan-shaped portion of the muscle which is in a position to function similar to the piriformis muscle which is in the position noted in the triangle colored in green. In addition to the increased stress applied to this muscle with an individual who overpronates this muscle is also responsible for holding your pelvis level which does so by lifting the majority of the weight of your trunk and upper body with each step. This is what holds your pelvis level so you can swing the opposite foot before your heel strikes the ground. There is tremendous stress applied to this muscle and tendon with every step of the human gait. So now we understand that an individual who excessively pronates applies significant stress to the hip rotator cuff especially the gluteus medius and piriformis muscles.

Most individuals take 70 million steps every 10 years. In each of these steps an individual with compromised foot and lower leg mechanics will apply excessive stress to the muscles attaching to the trochanter of the femur and to the tendons and associated bursa. The next step in understanding what transpires with these patients is to understand the pathophysiology of tendinosis. So now we will address the pathophysiological process that tendons undergo with cumulative stress. Many of us are familiar with rotator cuff tendon problems in the shoulder. You need to realize that similar tendon problems can develop in the hip as well when the tendons are placed under excessive stress over years.

THE PATHOPHYSIOLOGY OF TENDINOSIS & TENDINOPATHY:

This section of the discussion is rather sophisticated. I will attempt to simplify this discussion but I also do want to provide current research evidence to our understanding of tendinopathy especially for individuals with a science background or healthcare providers who may be interested in our approach and methodology. In general the term “tendinopathy” suggests that the patient presents with long- standing localized activity-related pain which has had a poor response to conservative treatment.

We have discussed the source of continued stress that is applied to the tendon attachment to bone predominantly overlying the greater trochanter. With cumulative stress there are pathologic changes that occur where there is gradual lysis of college and fibers, focal hypervascularity and changes in the tissue which we call metaplasia.

This process occurs in 3 stages basically the initiation of injury of tendon attachment to bone, failed healing and clinical presentation. In the picture to the left this is a microscopic view of tendon attachment to bone. Small fibrous attachments blend into the bone. When tendon undergoes continued stress the local cells making up the tendon react to the cumulative stress by undergoing what we previously described as “metaplasia”. This is a process where the cells begin to change to another lineage. Tendon cells become cartilage cells. Cartilage likes cells begin to appear in multiple locations where the tendon is under most stress. Cartilage cells function differently than tendon cells and therefore a number of changes begin to appear in the tendon tissue.

Remember the tendon normally has a job of providing tensile strength against loads. Cartilage cells have a completely different job. Cartilage cells lay down a substance called glucosaminoglycan. This mucoid substance deposits within the tendon causing the characteristic thickening of the tendon that we see on ultrasounds and MRIs in individuals with tendinopathy. In addition cartilage cells can also deposit calcium hydroxyapatite, which is why people with tendinopathy can also develop calcium deposits within the tendon. The changes at the tendon attachments ultimately began as an area of degeneration and subsequent small micro-tears that fail to heal. The failed healing leads to focal vascularity of the connective tissue which deposits a poor grade of connective tissue with poor tensile strength. In addition a number of biochemical changes also are seen where there is an expression of substances called cytokines that furthers the progression of degeneration as well as stimulates pain.

The mucoid degeneration that occurs at the tendon attachment explains what we see on ultrasound examination as well as MRIs.[1,2] It is very likely these pathologic changes create a gradual mechanical weakness and higher susceptibility to the small tendon insertion tears that we see early on in the course of disease which can ultimately lead to symptomatic tendinopathy.[3,4] Interestingly, we see clinically women present 7 to 1 more commonly over men with tendinopathy of the hip. We see that research literature suggests that women are in fact more susceptible to tendinopathy than their male counterparts.[5-8] The pathological changes in the tendon attachments can be seen in both the bone attachment sites as well as in the tendon structure itself.9

We utilized ultrasound imaging of the tendons, soft tissues and joints as part of our initial evaluation of all tendinopathy involving the hip, shoulder and other joints. The advancement of ultrasound technology has revolutionized the ability for subspecialty trained physicians not only to identify very specific findings insoft tissues and tendons, but also to target those soft tissue structures and tendons for injection and specialized interventional procedures.

Tendinopathy exhibits a host of characteristic findings that can easily be seen under ultrasound or MRI. An example would be focal areas of tendon thickening or swelling as well as localized hypoechoic (dark areas) on the ultrasound image at the attachment of the tendon or within the tendon.[10] Tendinopathy can show up as a signal abnormality on MRI and an echo abnormality on ultrasound because of the accumulation of water retaining proteoglycans that are part of the tendinopathy pathophysiology.[6,9] A few examples of ultrasound abnormalities involving tendons which include tendinopathy, calcific tendinosis and degenerative tear of the tendons are shown below.

When we investigate tendons that demonstrate tendinopathy there are host of findings that are seen under the microscope such as changes within the collagen matrix, hypercellularity within the region of tendinopathy, increased blood vessels or hypervascularity, but what we do not see is the presence of a lot of inflammatory cells.[2,11] Focal areas of cellular death (apoptosis) can be seen which may also explain some of the imaging findings.[12] Because of various expression of cytokines which are chemicals that can alter various inflammatory responses cause the release of degradative enzymes which also further causes a deregulation of cellular activities within the tendon.[13-15] Again as previously described changes in the cellular environment of the tendon which includes the appearance of cartilage like cells within the tendon also leads to calcification.[16,17] All of these pathological changes which we have not discussed in total make the tendon susceptible to mechanical stimulation and strain that cause the production of pro-inflammatory mediators.[18-21]

The process of tendon healing occurs in basically 3 phases: The initial phase involves an inflammatory response with the influx of cellular elements. Second, during the degenerative phase tendon cells which likely originated in the tissue around the tendon and just within the tendon migrate to the repair site and proliferate.[22,23] The last stage is the clinical presentation as the tendon becomes symptomatic. Chronic pain can occur thereafter which can be resistant to treatment. The physiology of tendon healing is extraordinarily complex. It involves numerous substances that stimulate a cascade of cellular activities that promote healing. Insidiously during the phase of degeneration one can see micro-ruptures in the tendon attachment to bone. Because of the various degenerative changes there is a failed healing response that ultimately leads to the persistent pain as previously described.

I have reviewed briefly some of the pathophysiology involved in tendinopathy in this article so you can begin to understand why we take the approach and specific technologies that we utilize in the treatment of this condition. We will be referencing some of the pathophysiology when we began to describe the treatment methods.

CAN I USE NONSTEROIDAL ANTI-INFLAMMATORY MEDICATIONS?

Many individuals with chronic peritrochanteric hip pain and bursitis use NSAIDs (nonsteroidal anti- inflammatory drugs) which are either prescribed by a physician or taken as an over-the-counter medication. Many individuals ask me whether or not they should or could use anti-inflammatory medications. The answer to that question is somewhat complicated. Let us take into consideration some of the research findings published in the literature. Interestingly, the use of NSAIDs is a double edge sword. In other words there are some beneficial effects and there are unfortunately deleterious effects. As previously stated tendon cells migrate to the area of injury and proliferate and synthesize substances important for healing. Some NSAIDs have an inhibitory effect on this process.[24] It's felt that prostaglandin E2 (PGE2) which anti-inflammatory medications inhibit may be important for early tendon healing such as control of vascular flow. Reducing PGE2 has some beneficial effects by reducing enzymes like metalloproteinases, but the normal matrix remodeling would also be affected thereby contributing to a failed tendon healing.25 Other concerns have been raised by researchers evaluating the effect of NSAIDs that could impede early healing process of an injured tendon–reduce tendon strength.[26-30] NSAIDs have been reported to inhibit collagen synthesis in various cell types and therefore are postulated to exert a negative impact on tissue healing.[31,32] Ibuprofen for example reduces the cellularity of the tendons and also inhibits tendon cell migration to the sites where they need to migrate to in order to stimulate proliferation and healing.[33,34] So, the NSAIDs that we commonly use for overuse injuries, sports injuries, and the aches and pains such as trochanteric hip pain could potentially contribute to tendinopathy.[25]

SO, WHAT EXACTLY IS CAUSING MY HIP PAIN?

So, we have introduced the reader to some basics of anatomy, biomechanics, and pathophysiology of tendinopathy. So now let's put all this together. We have discussed the fact that there are host of different tendons that begin in the pelvis and attach to the greater trochanter of the femur. We have also addressed the fact that there are bursae between the tendons or under the tendons and have discussed their function. We have also introduced the fact that there are specific types of foot structure and alterations in biomechanics of gait that can contribute to increasing stress on the tendons that attach to the greater trochanter.

Typically when an individual develops persistent pain over the greater trochanter there are degenerative changes that occur within the tendon and a "TENDINOPATHY" develops. Because of the degenerative changes over time there is a poor healing potential which leads to pain that becomes resistant to conservative treatment. Again, this is because of the pathophysiology involved his tendinopathy. Remember, we have addressed the fact that the tendon attachment to bone becomes altered and degenerated. We can see this alteration in small micro-tears and change in the architecture of the tendon through MRI and ultrasound examination. The degenerative changes result in a weakened and cheap grade of connective tissue that appears as an incomplete healing response at the attachment of the tendon to bone. This results in a vicious cycle of micro-tearing and reinjurie and failed tendon healing. As we have stated the connective tissue attachment of the tendon to bone becomes altered. There is a presence of a hypervascular, cheap grade of connective tissue with poor healing potential. Alterations within the attachment lead to chronic persistent pain. In addition to the tendinopathy most individuals also developed persistent bursitis under the tendons that are involved. This is more than likely related to the chronic increased tension to the external hip rotator cuff muscles or a response secondary to the production of pro-inflammatory substances in the area.

We have also stated that on occasion within the degenerative tendon, cartilage like cells begin to appear and can deposit mucoid substances called proteoglycans within the tendon that make the tendon become thickened. These cartilage like cells can also deposit calcium within the tendon. Calcification within the tendon represents a more extensive degeneration. Calcification within the tendon alters the mechanical behavior and reduces tensile strength of the tendon subjecting the tendon to further tears especially near or around calcification. The presence of calcification can complicate the clinical presentation of tendinopathy and we often treat this aggressively. We utilize ultrasound guided procedures to decalcify the tendon which will be addressed in this

article. Occasionally the degenerative tendinopathy can weaken the tendon and spontaneous tears can develop within the tendons attaching to the greater trochanter. This would especially be true with the gluteus medius tendon which we have addressed previously.

So in summary the cause of pain is often secondary to the development of tendinopathy within the tendon attachment to bone typically overlying the greater trochanter of the femur. These individuals can also be susceptible to microscopic tears, but failing to heal larger tears can sometimes develop.

CAN YOU REMOVE CALCIUM FROM THE TENDON NONSURGICALLY?
Actually we have developed a method of the calcification of the tendon. We have specifically developed two technologies that we utilize. The first is a method to chelate the calcium that can result in removal of the calcium deposit. There are also methods of simply injecting saline into the calcium and dissolving it and then aspirating the calcium. This is called an ultrasound guided percutaneous tendon decalcification. We also utilize platelet rich plasma injected within the calcification. We have found that the growth factors contained within platelets alters the cartilage cells ability to deposit calcium as well as causes a decalcification of the tendon. We utilize ultrasound to specifically place the needle tip at the site of calcification as shown in the picture to the right.

CLINICAL PRESENTATION OF PERITROCHANTERIC HIP PAIN CAUSED BY DEGENERATIVE TENDINOSIS:
Symptoms of chronic trochanteric hip pain can be unrelenting. Some individuals can find relief with sitting and rest or others the pain can be constant. An individual can have a low-grade pain that is always present that worsens when upright. Because of the tension on the tendons and bursa of the hip, typically symptoms worsen with walking. Another characteristic is that individuals find it difficult to lie on the side of involvement in order to sleep. They often find themselves avoiding lying on that side or awakening at night when they accidentally roll over onto that side.

A critically important phenomenon with regard to symptoms of this condition is important to understand. That is the referred pain that one can experience from this syndrome. It is very common for pain from the gluteus medius and gluteus medius bursa to radiate into the leg. Clinicians recognized this pattern as the L5 dermatomal pattern. Pain from the gluteus medius and the bursa of the hip can simulate sciatica! I have seen patients who have been seen by numerous specialists where abnormalities on MRI were identified and the patient was undergoing epidural injections and treatment for sciatica and was not responding when in fact they did not have any issues with their back but rather the hip. Injection of a diagnostic local anesthetic at the tendon and bursa completely relieved the leg pain.

CONDITIONS THAT SIMULATE PERITROCHANTERIC TENDINOPATHY:
There are multiple referred pain zones from the lumbar spine and pelvis that refer out into the hip. Radicular pain syndromes from nerve root compression such as from herniated disks. Painful discs in discogenic back pain syndromes can refer out over the hip and make the muscles very tender. The sacroiliac joint can refer pain over the hip. Multiple muscles have referred pain phenomenon over the hip. Physicians who were evaluating pain overlying the trochanter of the hip must be intimately aware of the various syndromes that can simulate peritrochanteric tendinopathy and bursitis.

COMBINED SYNDROMES: PERITROCHANTERIC HIP PAIN WITH OTHER BACK PAIN PROBLEMS:
It is actually quite common to see an individual with multiple pain generators as we have discussed in many of our other articles. Patients with peritrochanteric hip pain from tendinopathy and bursitis is no different. I see patients with pain arising from the lumbar spine facet joints, discs, nerves, sacroiliac joints, etc. all at the same time. It will be important to have all of this “sorted out”. This requires a very detailed and careful history and physical assessment. It will

occasionally require diagnostic blocks or treatment directed to the various pain generators to sort out complicated cases. But it is typically the rule rather than the exception that individuals that present with peritrochanteric tendinopathy and bursitis typically have other pain generators and other problems which is why this can confuse some clinicians who did not take the time to sort out the multiple pain generators that can present in these types of cases.

FOOT ORTHOTICS & THE PERITROCHANTERIC PAIN SYNDROMES:
We previously described how a common modality or intrinsic structure of the foot can often be the primary source for patients with external hip rotator cuff tendinopathy. We specialize in biomechanical analysis and examination of foot function as well as the casting and fabrication of foot orthotics within our practice. In this clinical situation it is sometimes inadequate to use a simple “arch support”. It is not a matter of supporting the arch but it is a matter of placing a post to correct forefoot varus.

If you notice the wedge under the varus forefoot in the picture to the left is the correction that is necessary. Most orthotic casting procedures are done in such a way that this portion of the foot and the position of the first ray is not well evaluated and the orthotic device created does not take into consideration the varus position and the first ray. We take pain staking efforts in performing orthotic casting and fabrication to take this into consideration especially with patients suffering from peritrochanteric syndromes. Part of the examination that patients with peritrochanteric hip pain undergo is a careful foot and biomechanical assessment including gait analysis.

We find individuals with obesity are more commonly affected with peritrochanteric tendinopathy. Interesting enough it is not universal and we do find individuals of ideal body weight who still have the syndrome. But obesity clearly makes a contribution to the increased stress applied to these tendons in addition to the other biomechanical stressors that we have already described. So part of the treatment strategy is obviously weight loss.

TREATMENT OF PERITROCHANTERIC TENDINOPATHY: AN ORTHOPEDIC REGENERATIVE MEDICINE APPROACH:
Corticosteroids injected into the hip bursa and around the tendon typically only provide short-term transient relief and ultimately we began utilizing strategies to stimulate connective tissue repair rather than corticosteroids. We began this many years ago.

I have addressed regenerative injection therapy on this website. We addressed this topic in detail in the article “Regenerative Injection Therapy and Pain Medicine”. I personally began using a technique known as “prolotherapy” which is a method of treating tendinopathy and ligamentous instability of joints. The old method utilizes dextrose sugar as a stimulus to promote collagen proliferation at the attachments of tendon to bone and ligament to bone. I still utilize this method to this day. Prolotherapy can and does work with many of these peritrochanteric tendinopathy problems, the only issue is the number of sessions required to get good treatment outcome. Frustrated with the length of time that it would take to help patients with these more complicated hip pain syndromes I begin searching for other alternatives. In approximately 1993 myself, a medical pathologist and veterinarian began work in animal research exploring the healing potential of platelets and their platelet derived growth factors as a stimulus to heal tendons that have undergone tendinopathy and insertional tears. Today this technique is called PRP (platelet rich plasma). When we first began using this many physicians question are use of this but today it is one of the hottest topics in sports medicine and treatment of many soft tissue conditions. It turns out the growth factors that are contained in platelets are capable of reversing much of the pathology that we have described with regard to tendinopathy. We utilize your own blood to obtain these platelets and concentrate them for therapeutic use.

With a simple blood draw your blood can be centrifuged and processed in such a way to remove the red cells from your blood and the majority of your plasma, which is the fluid that your blood circulates in. We concentrate the platelets and once transferred to a syringe can be a “cellular transplant” that can be injected in ligaments, tendons, and within arthritic joints. Again, this has

become extremely popular amongst physicians doing orthopedic medicine and orthopedic surgery. Once platelets can be concentrated we utilize ultrasound technology ultrasound imaging to direct the needle to the focal areas of tendon and soft tissue pathology where the cells can be deployed to stimulate their local effect. We have utilized platelet rich plasma to treat peritrochanteric tendinopathy for many years and have probably accumulated more experienced in most utilizing this method of treatment.

I would like to say that we “cure” everyone with a single platelet rich plasma injection but unfortunately such is not the case. We started this discussion with the description of the complexity of this problem. Later we described the complex pathophysiology and tissue changes within the tendon that are at the root of the chronicity. The reason why we addressed these issues was to discuss how complex treatment can be. One of the first regenerative medicine strategies that we have used in our practice was the use of platelet rich plasma. Currently, we have other more advanced options that we will not address on this website since part of this technology is proprietary and we are conducting studies with its use.

WHAT CAN I EXPECT FROM PLATELET RICH PLASMA INJECTION TREATMENT?
Again we have been using this method of treatment to treat peritrochanteric hip pain syndromes for many years. For us this dates back to the 1990s. We have used multiple methods of cellular preparation and laboratory techniques and we also had to experiment on countless occasions using various techniques to learn how to use this method of treatment effectively. There are several key elements that the patient needs to understand when undergoing this method of treatment.

First, this is not a corticosteroid injection where you will experience immediate symptomatic relief the following day from a treatment. The growth factors contained within platelets stimulate a cascade of healing events and actually set off localized pain that can last for days or weeks following the injections. Occasionally an individual may have pain for a protracted period of time. Yes, you do have to put up with some increased pain for a period of time as a part of the treatment process. We are working with some new technology that may resolve the majority of that problem but this will not be discussed in the context of this article. We will be happy to discuss these more proprietary methods when we consult patients individually.

To treat the postinjection flare we typically recommend ice packs and Tylenol although periodically we find it can be helpful to use a short-term course of opioid medications such as hydrocodone. We do this for a short period of time and do not typically recommend nonsteroidal anti-inflammatory medications since it may interfere with potential healing as we have described previously. One has to realize the complexity of the pathology and also realize that many times it cannot be done in a single treatment. It is surprising despite providing all of the information we do with regards to the pathophysiology, the extent of tendon degenerative change, and all of the precipitating factors how many individuals show up expecting to have some “magical” injection treatment on a one-time basis that will resolve all their problem. The connective tissue repair requires soft tissue engineering strategies using tissue growth factors and sometimes more sophisticated methods of treatment including biomechanical correction, lifestyle changes, etc. We are for the most part quite successful in treating this disorder and we have accumulated decades of experience in doing so. This article hopefully has provided some foundational knowledge and a means of better understanding the peritrochanteric hip pain phenomenon and its underlying pathophysiology.

REFERENCES:

1. Kannus P, Józsa L. Histopathological changes preceding spontaneous rupture of a tendon. A controlled study of 891 patients. J Bone Joint Surg Am 1991, 73(10):1507-1525.
2. Movin T, Gad A, Reinholt F, Rolf C. Tendon pathology in long-standing achillodynia. Biopsy findings in 40 patients. Acta Orthop Scand 1997, 68:170-175.

3. Järvinen M, ózsa L, Kannus P, Järvinen T, Kvist M, Leadbetter W. Histopathological findings in chronic tendon disorders. Scand J Med Sci Sports 1997, 7(2):86-95.
4. Tallon C, Maffulli N, Ewen S. Ruptured Achilles tendons are significantly more degenerated than tendinopathic tendons. Med Sci Sports Exerc 2001, 33(12):1983-1990.
5. CA. W, GJ. H, RE. M, SF. L. An epidemiological analysis of overuse injuries among recreational cyclists. Int J Sports Med 1995, 16(3):201-206.
6. Knobloch. K, Schreibmueller. L, Melle.r R, Busch. KH, Spies. M, Vogt P. Superior Achilles tendon microcirculation in tendinopathy among symptomatic female versus male patients. Am J Sports Med 2008, 36(3):509-514.
7. Hart D, Kydd A, Reno C. Gender and pregnancy affect neuropeptide responses of the rabbit Achilles tendon. Clin Orthop Relat Res 1999, (365):237-246.
8. Holmes G, Lin J. Etiologic factors associated with symptomatic achilles tendinopathy. Foot Ankle Int 2006, 27(11):952-959.
9. Movin T, Kristoffersen-Wiberg M, Shalab iA, Gad A, Aspelin P, Rolf C. Intratendinous alterations as imaged by ultrasound and contrast medium-enhanced magnetic resonance in chronic achillodynia. Foot Ankle Int 1998, 19(5):311-317.
10. McLoughlin R, Raber E, Vellet A, Wiley J, Bray R. Patellar tendinitis: MR imaging features, with suggested pathogenesis and proposed classification. Radiology 1995, 197(3):843-848.

11. Rolf C, Fu B, Pau A, Wang W, Chan B. Increased cell proliferation and associated expression of PDGFRbeta causing hypercellularity in patellar
12. Lian Ø, Scott A, Engebretsen L, Bah rR, Duronio V, Khan K. Excessive apoptosis in patellar tendinopathy in athletes. Am J Sports Med 2007, 35(4):605-611.
13. Fu S, Chan B, Wang W, Pau H, Chan K, Rolf C. Increased expression of matrix metalloproteinase 1 (MMP1) in 11 patients with patellar tendinosis. Acta Orthop Scand 2002, 73(6):658-662.
14. Fu S, Wang W, Pau H, Wong Y, Chan K, Rolf C. Increased expression of transforming growth factor-beta1 in patellar tendinosis. Clin Orthop Relat Res 2002, (400):174-183.
15. Nakama L, King K, Abrahamsson S, Rempel D. VEGF, VEGFR-1, and CTGF cell densities in tendon are increased with cyclical loading: An in vivo tendinopathy model. J Orthop Res 2006, 24(3):393-400.
16. Uhthoff H. Calcifying tendinitis, an active cell-mediated calcification. Virchows Arch A Pathol Anat Histol 1975, 366(1):51-58.
17. de Mos M, Koevoet W, van Schie H, et al. In vitro model to study chondrogenic differentiation in tendinopathy. Am J Sports Med 2009, 37(6):1214-1222.
18. Bosch U, Zeichen J, Skutek M, Albers I, van Griensven M, Gässler N. Effect of cyclical stretch on matrix synthesis of human patellar tendon cells. Unfallchirurg 2002, 105(5):437-442.
19. Wang J, Jia F, Yang G, et al. Cyclic mechanical stretching of human tendon fibroblasts increases the production of prostaglandin E2 and levels of cyclooxygenase expression: a novel in vitro model study. Connect Tissue Res 2003, 44(3-4):128-133.
20. Li Z, Yang G, Khan M, Stone D, Woo S, Wang J. Inflammatory response of human tendon fibroblasts to cyclic mechanical stretching. Am J Sports Med 2004, 32(2):435-440.
21. Yang G, Im H, Wang, JH. Repetitive mechanical stretching modulates IL-1beta induced COX-2, MMP-1 expression, and PGE2 production in human patellar tendon fibroblasts. Gene 2005, 363:166-172.
22. Russell JE, PR. M. Collagen synthesis during primate flexor tendon repair in vitro. J Orthop Res 8:13–20.1990.
23. Gelberman R, Amiel D, F. H. Genetic expression for type I procollagen in the early stages of flexor tendon healing. J Hand Surg 17A:551– 558. 1992.
24. Riley G, Cox M, Harrall R, Clements S, Haleman B. Inhibition of tendon cell proliferation and matrix glycosaminoglycan synthesis by nonsteroidal anti-inflammatory drugs in vitro. Journal of Hand Surgery (British and European Volume, 2001) 26B: 3: 224-228.
25. Chan K, Fu S. Anti-inflammatory management for tendon injuries-friend or foes? Sports Medicine, Arthroscopy, Rehabilitation, Therapy & Technology Sports Medicine, Arthroscopy, Rehabilitation, Therapy & Technology 2009, 1:23 p 1-3.

26. DS. M. Comparative study of ibuprofen and aspirin in soft-tissue injuries. Rheumatol Rehabil
1974;13:141-147.
27. Nishimura K, Nakamura R, diZerega G. Ibuprofeninhibition of postsurgical adhesion formation: a time and dose response biochemical evaluation in rabbits. J Surg Res. 1984;36:1:15-124.
28. Kulick M, Smith S, Hadler K. Oral ibuprofen: evaluation of its effect on peritendinous adhesions and the breaking strength of a tenorrhaphy. J Hand Surg [Am] 1985;11:110-120.
29. Ferry S, Dahners L, Afshari H, al. e. The effects of common anti-inflammatory drugs on the healing rat patellar tendon. Am J Sports Med. 2007;35:1326-1333.
30. Cohen D, Kawamura S, Ehteshami J, al. e. Indomethacin and celecoxib impair rotator cuff tendon-tobone healing. Am J Sports Med 2006;34:262-269.
31. Nishi O, Nishi K, Fujiwara T, al. e. Effects of diclofenac sodium and indomethacin on proliferation and collagen synthesis of lens epithelial cells in vitro. J Cataract Refract Surg. 1995;21:461-465.

32. Matsumoto H, Fujii A. Tenidap, an anti-inflammatory agent, inhibits DNA and collagen syntheses, depresses cell proliferation, and lowers intracellular pH in cultured human gingival fibroblasts. J Pharmacol Exp Ther. 2002;300:668-672.
33. Tsai W, Hsu C, Chang H, Lin Y, Lin M, Pang J. Ibuprofen inhibits tendon cell migration in a process that is probably mediated by the down-regulation of paxillin. J Orthop Res. 2006;24:551- 558.
34. Tsai W, Hsu C, Chang H, Yu-Chun Lin Y, Lin M, Pang J. Ibuprofen Upregulates Expressions of Matrix Metalloproteinase-1, -8, -9, and -13 without Affecting Expressions of Types I and III Collagen in Tendon Cells. J Orthop Res 2010;28:287-291.