

3. Theory for subjective assessment

What is the importance of taking a good history?

A good subjective history from the patient is critical in tendinopathy, because it can masquerade as many other conditions. For example, in the Achilles area, there are four or five different diagnoses that might exist, and you have to be essentially sure as a clinician that you are actually dealing with a tendinopathy. If the patient history is inadequate, you can label symptoms as a tendinopathy, when they are not, and this, in turn, will lead you to the wrong treatment path.

This is why the subjective history has to be extensive. You have to consider several things such as how the tendon responds to load, where the pain is, what other factors might be contributing to that patient's pain. However, one piece of information is particularly relevant: what caused the condition and how the person responds to load on a day-to-day basis.

Table 1: Consensus checklist for researchers

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Item	Description
Sex	Report sex of all participants
Age	Report in years/months (and whether part of inclusion/exclusion into study)
Standing height	Report unit of measure* and whether it was measured or self-reported
Body mass	Report unit of measure* and whether it was measured or self-reported
Symptom duration	Length of current episode, and whether first episode
History of tendon pain	Description of whether any prior history at any tendon site
Level of disability	Report what was used (eg, VISA or other valid scale)
Severity of symptoms	Report what was used as a measure for symptom severity (eg, pain intensity rated on a visual analogue scale or numerical rating scale) Record context and instructions given to participant (eg, recall pain or pain during a load test)
Loading tests	Report whether loading tests were used, and detail appropriate methodology of used tests (eg, joint angles, repetitions)
Location of symptoms	Describe location of patient reported pain, and whether this was a selection criterion and whether the pain was recorded during a pain provocative load test
Imaging	State whether imaging was used
Recruitment	Detail recruitment source and strategy, including number excluded from participation. Consider reporting geographical location, duration and target audience, whether participants were paid and if they were currently seeking care
Comorbidities	Report whether any significant comorbidities for tendon conditions were included/excluded—use comorbidities infographic (online supplementary file 1)
Medication use	Report whether any medication use was included/excluded—use medication guide (online supplementary file 2)
Other characteristics	Consider previous interventions, other pain locations, nicotine use or sleep/well-being factors, psychological factors
Physical activity level	Report physical activity measure appropriate to the population
All items listed apply to both cases and controls	

(Rio, et al., 2019, p. 3)

The four key areas for reporting characteristics are of fundamental importance to clinical practice and researchers. Demographic information is a vital contributing factor to tendinopathy, particularly in areas such as gluteal and Achilles tendinopathy, where factors such as sex and age are often associated with symptoms. Similarly, descriptors of tendinopathy such as symptom duration or severity may be associated with differences in clinical effects or demonstrate different subgroups of patients within a certain type of tendinopathy. This information also helps clinicians appraise

how generalizable and relevant research findings may be to the patient in front of them. Information on general health and comorbidities in relation to tendon symptoms is also often gathered as part of history taking in clinical practice, yet in research is under-reported. It is potentially a major confounder to treatment outcomes and particularly influential on conditions such as Achilles tendinopathy. (Rio et al., 2019, p.)

In the website *Clinical edge* (2015), there are “5 keys to a great subjective assessment” proposed:

- 1- Arguably, the majority of the important information gathered in your treatment session is obtained in the subjective assessment. Have the courage to spend a lot of time on subjective history. You will pick up an enormous amount of potentially valuable information that you would otherwise miss with superficial history taking.
- 2- Listen to your patient for clues on how to treat their pain, and activities to modify. Patients will reveal a lot of valuable information on aggravating factors, movements and positions you can modify to help your patient improve.
- 3- Keep red flags in the back of your mind when taking history and assessing e.g. night pain, unusual symptoms, cauda equina, unexpected weight loss, neurological symptoms in multiple dermatomes.
- 4- Follow up ‘red flag’ symptoms. It is not enough to ask a question and ignore a positive or equivocal response. Clarify the details of the symptoms if you get a positive response, and refer for further investigations.
- 5- Don’t immediately blame your treatment if your patient is not getting better, ask questions to find out more, e.g. if they have been out dancing all weekend and their heel pain is aggravated. (On the other side, we do love to take the credit when patients improve, but remember to find out if your patient has modified their activity when they improve as well!) (5 keys to a great subjective assessment, 2015) <https://www.clinicaledge.co/blog/5-keys-to-a-great-subjective-assessment>

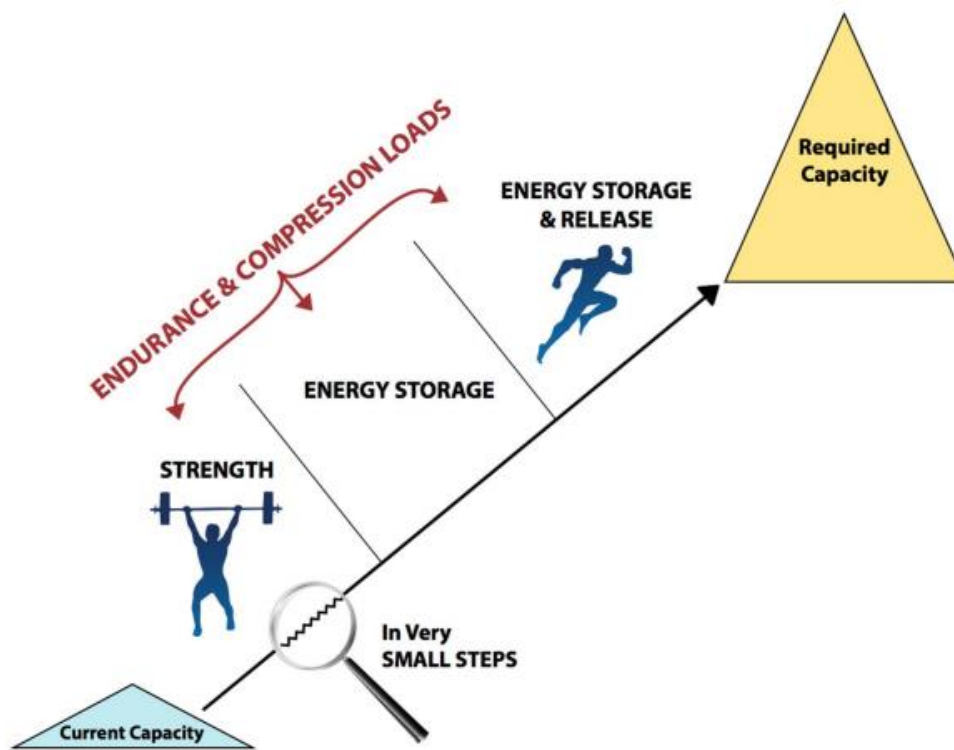
Understanding capacity

According to Cook & Docking, (2015),

A tissue is at full capacity when the individual is able to perform functional movements at the volume and frequency required without exacerbating symptoms or causing tissue injury. A tissue will only ever have the capacity that just exceeds the load that is virtually placed on it. That is, if you decrease your load, then your tissue capacity will decrease. If you increase your load,

your tissue capacity will increase. Now, in tendons, this is particularly true. We know that capacity can change within weeks of unloading and perhaps within weeks of reloading. So sorry. So it doesn't matter what if I were to say so. If you do change your load very abruptly, then a tendon will not have the capacity to deal with that load. That is, we often see people present with tendon pain who have changed what they've done. They've decreased the load on the tendon and then that capacity drops. Then they reload the tendon with a low that exceeds what they're capable of taking. Then they get tendon pain and that's when they present to you as a clinician. (Cook & Docking, 2015)

Figure 1: improving tendon capacity with progressive loads



(Cook & Docking, 2015, p. 1485)

MEASURING CAPACITY IN SPECIFIC TISSUES

Cook and Docking (2015) explain the following:

Bone is the musculoskeletal structure that has the most evidence for its restructure with the application of load (especially up to the end of puberty). We can measure changes relatively easily and structure is clearly linked to capacity (at least at the extremes such as osteoporosis). Muscle bulk can easily be estimated clinically and measured with imaging but may not be an

accurate representation of capacity. Similarly, dynamometry may reflect aspects of capacity (strength and endurance) but cannot easily measure power. There are no easy measures of tendon capacity; even extensively pathological tendons appear to have the capacity to tolerate very high sporting loads. We espouse increasing tendon capacity through rehabilitation with gradual tendon loading that reduces pain and improves function allowing the athlete to return to sport pain-free (figure 1). The mechanisms by which the tendon increases capacity in response to load is unclear as the tissue pathology may not change. Perhaps we are merely increasing the capacity of the normal part of the tendon.⁴ Answers to additional key questions about tendon capacity, such as, 'Do tendons continue to develop after puberty?' and 'How do they adapt to increasing loads?', are unknown. There is evidence that the collagen in the tendon matrix is stable and has limited turnover after puberty.⁵ Loading tendon appears to give different results depending on the age and gender of the participants;⁶ again, younger tendons appear to be able to adapt structurally,⁷ whereas adult tendons may not change in structure but adapt mechanically.

Structural adaptation is easy to understand, with more matrix proteins organized into viable matrix structure, while mechanical adaptation is less clear as the changes in the tendon that make it stiffer and more tolerant to higher loads are unknown.(Cook & Docking, 2015, p.)

THE OPPOSITE OF CAPACITY—TISSUE WEAKNESS

Cook and Docking (2015) also inform us about the opposite of capacity:

The opposite of loading is unloading, and the opposite of capacity is tissue weakness. The 'use-it or lose-it' phenomenon of tissue capacity has been extensively described in bone. While injury may be limited to one tissue, pain and reduced loading must reduce capacity in associated tissues within the kinetic chain. Failure to return capacity in all these tissues may result in reinjury or subsequent injury. Understanding the mechanobiology of musculoskeletal tissues, and how loading and unloading affects the capacity of these tissues is critical to clinicians if we are to rehabilitate resilient athletes. (Cook, & Docking, 2015)

What questions to ask and subjective examination

There are two key questions that you need to ask when you are talking to somebody who has tendon pain: 'Where is the pain?' and 'What aggravates the pain?'

As regards the first question, it might seem very obvious, but it is an essential question. Tendon pain remains isolated to the tendon. That is, it does not spread around the tendon, neither does it shift with loads. So you must ask the person to describe both their resting pain and the load pain, because if you see a difference between resting pain and load pain, then you know that it is likely not to be a tendon. The key information that we can get from this question is whether the pain is localized in one or two fingers and whether it stays in that place regardless of what activity the patient does.

As for the second question, -what aggravates the pain?- the patient is asked to describe the activities that give them tendon pain. As we have talked about in the loading section, there are three types of load that increase the potential for pain in a tendon. That is the energy storage and release, or the spring-like loads, the compressive loads, and the friction loads. We will expect the patient to describe any of those three loads. Athletes, in particular, will describe the spring-like loads. So, for the Achilles tendon, the Patel attended, it will be by changing direction, jumping or sprinting that their tendon pain will maximize. If they describe pain without these loads, for example, when they ride a bike, you can be relatively sure with this question that you are not talking about other energy storage or compressive loads. You might be talking about friction loads because riding causes movement in the tendon against the surrounding tissues.

If people describe pain at night, then you can be again relatively sure they are not talking about tendon pain, but morning pain, particularly in the Achilles or first movement pain. So first load on a tendon in the morning will very often give you tendon symptoms. The classic example is the Achilles tendon: people have pain in the morning for a variable period of time, or sometimes they have stiffness in the morning. That sort of history is very important because it allows us to be really sure that the pain is coming from the tendon. As you can see, these two questions can be really important to discriminate between a load that is likely to induce tendon pain and one that is not.

Still, there is another question that we can ask: 'Were you injured when the symptoms first came up?' Remember we talked about loading capacity, about pain being a result of putting a load on a tendon that exceeds the tissues capacity or attendance capacity. Patients will present to you with symptoms of tendon pain after they have done a load exercise that is not usual for them. For a recreational athlete, it might be as simple as deciding to train for a marathon, it might be about changing the way they run. They always run five kilometers, but an elite athlete one day can decide to run on the hills rather than on a flat surface. It will be much more suitable than that. So, it can be as simple as a change in shoes, particularly changing heel. On the other hand, in athletics sometimes we see a change in the track surface. Sometimes they normally train on a track that is quite soft and then compete on a very hard track; this can actually cause symptoms of Achilles tendon problems. Another similar case is that of the coach that is very upset with the team and makes the training session last three hours instead of two: these changes also commonly cause certain symptoms. Therefore, we should be very careful about pinning down with the athlete or a

person with tendon pain. Exactly what changed when their symptoms came on is highly important.

Here comes another question that is really helpful for: 'If you do more exercise, do you feel worse the next day?' Pain classically at 10 am may hurt a little bit. When you start an activity, the tissue often warms up, so you actually feel less pain as you are active. But it is the next day that you see the patient. Now, this is actually quite unique to tendon pain: Very often musculoskeletal conditions, such as joint pain, will hurt more and more as you do an activity and then settle overnight and be better the next day. By contrast, tendon pain will get better and better as you do an activity -though it can sometimes get a little bit sore at the end of activity-, but the next day it will be substantially worse. As clinicians, we assess this by looking at things like morning stiffness or pain with stairs in the morning or pain when bending forward, depending on which tendon we are examining. But the patient must have this classic history of feeling worse the day after the activity or after a day in which they did more activity than what they normally do. All this can be explained with an example: If I run 5 kilometers, I have morning stiffness that lasts 10 minutes; if I run 10 kilometers, I have morning stiffness that lasts 15 minutes. That is the relationship between pain and activity that we are looking for.

Another key question is to ask the patient is 'What have you previously done to release your tendon pain?' It is important to know whether they have seen a medical person, a physiologist or someone who has given them exercises, as well as what they have tried doing? It commonly occurs that the longer a person has had the pain, the more interventions they have tried (very often, interventions that are medically driven or directed at a pathology.)

As we mentioned previously on Module 2, the tendon pathology actually is not that relevant. And it is interesting to note that many of the interventions that people receive are actually not helpful either for the pathology or for their pain. The other interesting thing about interventions that are medically driven, particularly to the pathology, is that they often come with a period of rest. We have also said that if you rest, the actual capacity of your tendon decreases. Therefore, not only does that sort of intervention not help, but the rest period after the intervention event actually makes the tendon worse. So, actually documenting whether they have undergone any injection therapies and which they were, how much rest they had, what the response was, is very important.

We should also document on how many active and passive interventions they have received. Many people have tried passive interventions, such as machine or electro therapy, driven elements, lots of massage, lots of tended frictions. As far as we know, most of those will give a very short-term relief of pain, but they will not give long-term relief of pain. It is important to know whether their interventions have actually been mainly passive, or whether they have also had active interventions, (exercise based on a loading-based approach.) Again, documenting exactly what they have done is important. What we know from tendon load and capacity is that you have to load a tendon highly to change capacity. And very often patients have tried active interventions, but at a much lower level than anything that changes

the tendon. It is very common that patients try exercises with a band, or that they do unloaded exercises, or just bodyweight-load exercises. All of these are helpful, but none of them are actually going to change the capacity of the tendon, which is what we want in the long run. In brief, you must document exactly what that patient has done in terms of exercises, the sort of sets and reps they have done, whether such exercises had an effect on their tendon or not, etcetera. In this way, you can really analyze whether their previous interventions have been helpful or not and what different ways you are going to take to reduce or relieve the patient's tendon pain.

Modifiable risk factors

Clearly, the most important modifiable risk factor is load. We have previously seen how changes in load, particularly an increasing load from a base capacity, can actually induce tendon pain. That should be quite covered off in your history of what has changed and what your loading history is like. There are a couple of other modifiable risk factors that are linked to this.

The first one has to do with injuries, and the question that you need to ask is 'Have you had any other injuries within the last three months or even within the last 12 months?' This is so because it is very common what to see patients presenting with tendon pain after a period of unloading, and they not having linked that to a changing load. The two classic examples are the following: A person is having time off for an ankle sprain or a knee sprain, which lasts three or four weeks. The person's tendon capacity has decreased either in the Achilles or Patellar tendon. The person then returns to training because the ankle is feeling fine. What happens is that, since the tendon capacity is decreased, the return to training actually implies an increased load and, even though the ankle seems to be fine, the tendon pain returns.

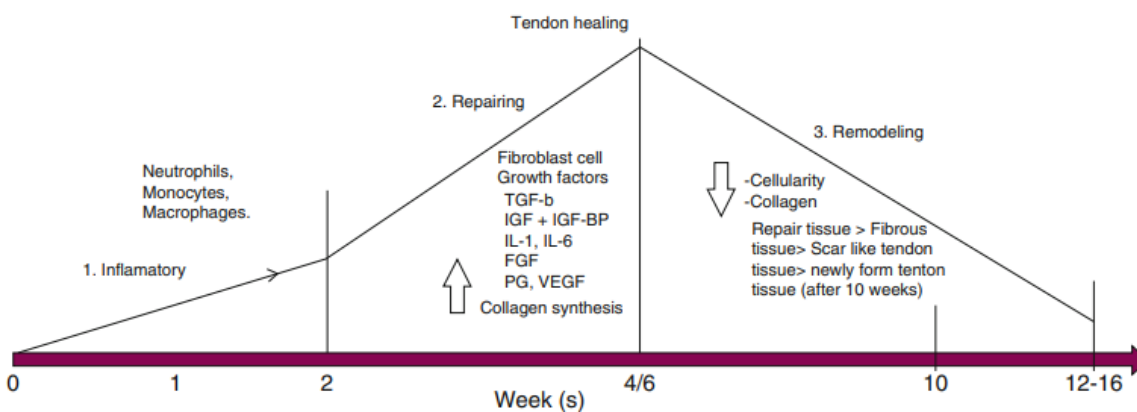
The second modifiable risk factor, as we have talked about in the load section, is related to activities, like changing orthotics, shoes or surfaces, that change the interaction between the tendon and the surface that the patient interacts with. And that, again, is a load change that is really important.

The third modifiable risk factor is dorsiflexion load. This is particularly the case in people who have had an ankle sprain. On the contrary, people who have a naturally stiff ankle need to address the fact that they do not see that flexion range is limited. It is known that with mobilizations or stretching we can allow more dorsiflexion range. Particularly for the Achilles and the patella tendon, that allows us to absorb more energy at our foot ankle complex and, in fact, that will take the load off the tendon.

The last model for modifiable risk factor I want to talk about is body weight. What we know, of course, is that when you increase your body weight, more mechanical stress is created on your tendon. Therefore, if somebody has had a period of time off, it is alright. But if somebody

has put on weight during an unloading period of time because of an injury, the effect of that will be that load on the tendons will actually increase. But more importantly, from a perspective of increased weight, we know that visceral fat is very pro inflammatory and that it drives a lot of systemic changes and inflammatory changes in the body. In turn, this increases a person's chances of developing both diabetes and insulin resistance, which can lead to high cholesterol. As regards the implications in tendons, as we know, cholesterol can deposit in tendons, and very high systemic cholesterol can directly affect tendons. But more importantly, probably, diabetes has a profound effect on tendons. What it does is to stiffen the tendon, move it, get more cross linking in the tendon, and that changes the response of the tendon to the load. So, if somebody presents to you with an increase in weight, it is really important to consider whether they have had a recent blood test to check their levels of cholesterol, whether they have had any sort of insulin resistance in insulin sensitivity tests, and whether they have type 2 diabetes; both of these things are critical in modifying. In this way, we can have a lower risk of tendon pain.

Figure 2: Period for tendon healing and remodeling



(Ahmed, 2016,)

Ranger, et al. (2015) provide compelling evidence for an association between tendinopathy and diabetes:

People with diabetes had more than three times the odds of tendinopathy compared to controls (OR 3.67, 95% CI 2.71 to 4.97, I² =81%, figure 2). People with tendinopathy had 1.3 times increased odds of diabetes compared to controls (OR 1.30, 95% CI 1.12 to 1.50, I² =0%, figure 3). People with both diabetes and tendinopathy had a longer duration of diabetes than people with diabetes but not tendinopathy (MD 5.26, 95% CI 4.15 to 6.36, I² =46%, figure 4). People with diabetes had thicker tendons than people without diabetes (SMD 0.79 95% CI 0.47, 1.12). A biological pathway exists whereby hyperglycaemia associated with diabetes affects collagen cross-linking in tendons. (Ranger, et al. (2015,)

Van der Vlist, et al. (2019) determined the following four elements as modifiable patient characteristics that worked as risk factors for Achilles tendinopathy:

- **Body mass index (BMI) and body weight.** There is limited evidence that BMI or body weight do not affect the risk for AT. No association was found in five cohort studies for BMI and in three cohort studies for body weight. One cohort study found that being overweight (BMI $\rightarrow$ 25.0) and obesity (BMI $\rightarrow$ 30.0) are associated with Achilles tendinopathy (AT). Another cohort study found that body weight is increased in people who develop AT. (Van der Vlist, et al., 2019, p. 6)

In relation to this, Malliaras, et al. (2007) have something to add:

waist girth seems to have a stronger association with abnormal patellar-tendon injury than other anthropometric factors among men. As waist girth is an accurate anthropometric surrogate for abdominal adipose tissue, free fatty acids and cytokines released by abdominal adipose tissue may have a role in tendon pathology. (Malliaras, et al. 2007,)

- **Alcohol use.** There is limited evidence that moderate alcohol use increases the risk for AT. Moderate alcohol use was defined as 7–13 units per week for men and 4–6 units per week for women. One cohort study reported that moderate alcohol use is associated with AT compared with no alcohol use. No association was found for light alcohol use or heavy alcohol use compared with no alcohol use.¹⁷
- **Smoking.** There is limited evidence that smoking is not associated with AT based on one cohort study.
- **Physical activity level, physical activity performance and hours of sports participation.** There is limited evidence that physical activity level, physical activity performance or hours of sports participation do not affect the risk for AT. Two cohort studies found no association between the physical activity level measured with the Baecke questionnaire and AT.^{16 19} One cohort study found no association between the physical activity performance (2km run and maximum number of chin-ups and sit-ups) and AT.²² Another cohort study found no differences in hours of sports participation between patients with AT and unaffected controls. (Van der Vlist, et al., 2019, p. 6)

On the same subject, Sprague et al. (2018) suggest the following:

At the moment the study was done, there were some well none facts, where Patellar tendinopathy is extremely common in athletes who participate in jumping sports, and many will experience recurrent symptoms after treatment. In addition, the symptoms of patellar tendinopathy are load-

dependent, with increased loads resulting in a greater degree of pain. However, the study showed that there was limited or conflicting evidence that decreased ankle dorsiflexion range of motion, decreased posterior thigh and quadriceps flexibility, greater volume of jump training, more volleyball sets played per week, greater CMJ (counter movement jump) height, and greater activity volume are potential modifiable risk factors for patellar tendinopathy. Also, moderate to conflicting evidence supported greater volume of strength training, better jumping performance, less normalized hip extension torque, decreased knee extension fatigue ratio and altered corticospinal excitability to the quadriceps as associated modifiable factors. (Sprague et al., 2018,)

Visnes & Bahr (2012) analyzed total training and competition load and body composition as potential risk factors for developing jumper's knee, and the finding in young, elite volleyball players was that previous and current training and match exposure were significant risk factors, as was gender. In contrast, they detected no association between body composition and injury risk.

Holmes and Lin (2006) also found out the following:

Hypertension has been cited as a common and important risk factor for nearly all vascular disorders. An animal study showed that the presence of hypertension results in alterations in the composition and biomechanical properties of arterial vessels causing them to be less distensible. The cumulative result of hypertension affects all three limbs of Virchow's triad which includes 1) endothelial damage, 2) stasis or turbulence of flow, and 3) hypercoagulability. This promotes a prothrombotic state in which small thrombi, occlusive arterial disease, and decreased blood flow may alter circulation to the Achilles tendon. Obesity has been found to be an independent epidemiologic risk factor for atherosclerotic disease. Obesity also has been shown to cause an insulin-resistant state that results in decreased endothelial cell nitric oxide levels. A similar decrease in nitric oxide levels also is caused by hypertension. These factors both lead to decreased blood flow, which would compromise the normal metabolism and reparative maintenance of the Achilles tendon.

Female hormones have been shown to play a significant role in the composition and biomechanical properties of connective tissues. Approximately 70% of the dry weight of a tendon is collagen, with about 95% of this being type I collagen. Type I pro-collagen and cellular proliferation have been shown to decrease in a dose-dependent manner with increasing levels of estrogen in human in-vitro anterior cruciate ligament fibroblasts.

Steroid use has been linked to rupture of the Achilles tendon through studies using animal models and human case reports. Some human studies have suggested that weakening of the Achilles tendon can occur with both local and systemic steroid exposure. (Holmes and Lin, 2006,)

Also Cook, et al. (2000) concluded the following:

Patellar tendon sonographic lesions occur in asymptomatic, elite 14 to 18 year old basketball players, which suggest that patellar tendinosis can begin in young adulthood. Clinicians need to include this condition in the differential diagnosis of anterior knee pain in the young jumping athlete. This may suggest that certain sporting activities may predispose athletes to develop tendinopathy. (Cook, et al., 2000,)

Non-modifiable risk factors

There are several factors that can affect tendon pathology and tendon pain, which are not modifiable by the person or by the clinician. The first one is genetics. There is quite a considerable ratio. Research shows that your **COL5A1** gene is implicated in your risk of having both tendon pathology and tendon pain. But we know that any collagen-based genetic polymorphisms actually are implicated in intending disease. It can be type 5 that has direct implications, particularly for Achilles tendinopathy, but type 1 collagen diseases, such as Marfan syndrome, can also affect tendon disease, tendon conditions.

September, et al. (2008) determined that the BstUI restriction fragment length polymorphisms is associated with chronic Achilles tendinopathy in a second population and that “a region within the COL5A1 39 untranslated region may predispose individuals to an increased risk of developing chronic Achilles tendinopathy.” (September, et al., 2008)

The second non-modifiable fact factor is any of our zero negative diseases. These are conditions like psoriatic arthritis, writer syndrome, ulcerative colitis, which are autoimmune diseases. Many of these affect the bone tendon junctions. We often see these patients as an Achilles insertion problem, and the ability of the clinician to affect this condition is really constrained by the disease process itself. If you are suspicious of implications of zero negative disease, you must ask all of the key questions that indicate that this is a problem. We talk about morning stiffness, about response to anti inflammatory about skin manifestations, eye manifestations, gut manifestations or any factor critical in evaluating if a zero negative disease is part of the presentation. If the patient looks like they may have an autoimmune disease of some description, it is really important that that condition is managed first before you try and manage the tendon condition. If you try and manage the tendon condition first, while the autoimmune disease is still active, you will not get a good result. So these people must be referred to a rheumatologist so as to modify their disease

process first. Then you can actually modify their loading environment, their strength and all of the things that we will talk about in management.

The third factor that is important in women is menopause. What we know is that menopause is implicated in the presentation of both tendon pathology and tendon pain. So, once you are in a pre menopausal or post menopausal state, you are at risk of tendon pathology and tendon pain. What we see with women who are usually active or have changed their loading environment, is that they can get an onset of tendon pain that can be actually quite hard to control. The ability to modify the menopausal status is unknown with hormone replacement therapy (HRT). Whether HRT is effective or not has not been studied, but it is quite possible that short-term interventions that modify the hormonal status of women could be effective in helping managing the tendon pain. You must be very sensitive to your load changes and to the way you manage them because they can be quite difficult to settle down. We have talked about diabetes and cholesterol as systemic diseases that are implicated in tendon pathology and tendon pain, and we have talked about these muscles, the status of these must be known in somebody with persistent tendon pain because, again, you need to modify the disease status to be effective in managing the tendon pain. That is, if somebody is type 2 diabetic, then they need to take in medications to alter their glucose status. That, then, allows you to affect their tendon pain.

The last semi-modifiable risk factor is Fluoroquinolones antibiotics. These are antibiotics that are usually taken in by people who are very ill, but can be taken in by people in general to treat urinary tract infections and sometimes respiratory tract infections. What we know from Fluoroquinolones antibiotic prescriptions is, it has profound effect on all sorts of college and but it seems to manifest as tendon pain and pathology. And it happens very quickly. So you do not have to finish a course on Fluoroquinolones antibiotics to present with tendon pain. And in fact, there is evidence that the tendon can rupture within six days of taking the medications. Stephenson, et al. (2013) suggested in a review that there is a significant association between fluoroquinolone use and tendon injury, in particular Achilles tendon rupture and tendinopathy. This was shown using different study designs and in different populations. Fluoroquinolone-associated tendinopathy is relatively uncommon, but when it occurs, it tends to happen soon after exposure (within the first month).

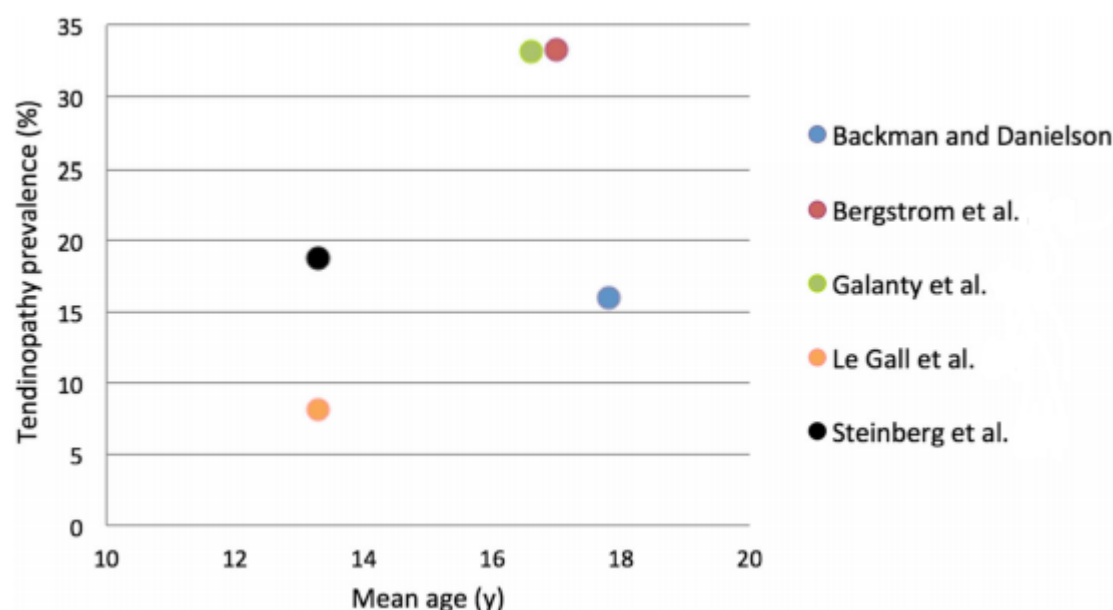
So, one of the key questions you must ask your patients is 'Have you had antibiotics recently? And if you have, what type were they?' If you suspect that the person has a Fluoroquinolones induced tendon, obviously your whole strategy must change because they are very unresponsive. These tendons are very unresponsive to many of our loading interventions and many of the things that working in people with tendon pain from other sources.

Van der Vlist (2019) determined the following non-modifiable risk factors for Achilles tendinopathy according to a systematic review: age, sex, ethnicity, height, and prior lower limb tendinopathy or fracture:

Age: “There is conflicting evidence that age affects the risk for AT. One cohort study reported in 2013 that a birth year of 1980 or later is associated with a decreased risk for AT. Two cohort studies showed no association.” (Van der Vlist, 2019, p. 6)

On this subject, Simpson, et al. (2015) found that lower limb tendon pathology and tendinopathy are present in children and adolescents, and coincide with the ages of puberty and biological maturation. In addition, in relation with the following non-modifiable risk factor (sex), he found that male sex is significantly associated with tendinopathy in studies that report tendinopathy by tendon.

Figure 3: Relationship between age and tendinopathy prevalence as diagnosed by physical examination.



(Simpson et al., 2015, p. 554)

Sex There is conflicting evidence that sex affects the risk for AT. One cohort study reported that being female is associated with AT. No association was demonstrated in two cohort studies.

Ethnicity There is limited evidence that ethnicity does not affect the risk for AT. One cohort study reported no increased risk for white (non-Hispanic), black (non-Hispanic) or other ethnicity.

Height There is limited evidence that height does not affect the risk for AT. No association was found in three cohort studies. One cohort study reported an increased height in patients with AT.

Prior lower limb tendinopathy or fracture There is limited evidence that a prior lower limb tendinopathy or fracture increases the risk for AT. One cohort study reported that a prior lower limb tendinopathy (plantar fascia, Achilles or patellar) or fracture (regardless side of injury) is associated with AT.¹⁷ (Van der Vlist, 2019, p. 6)

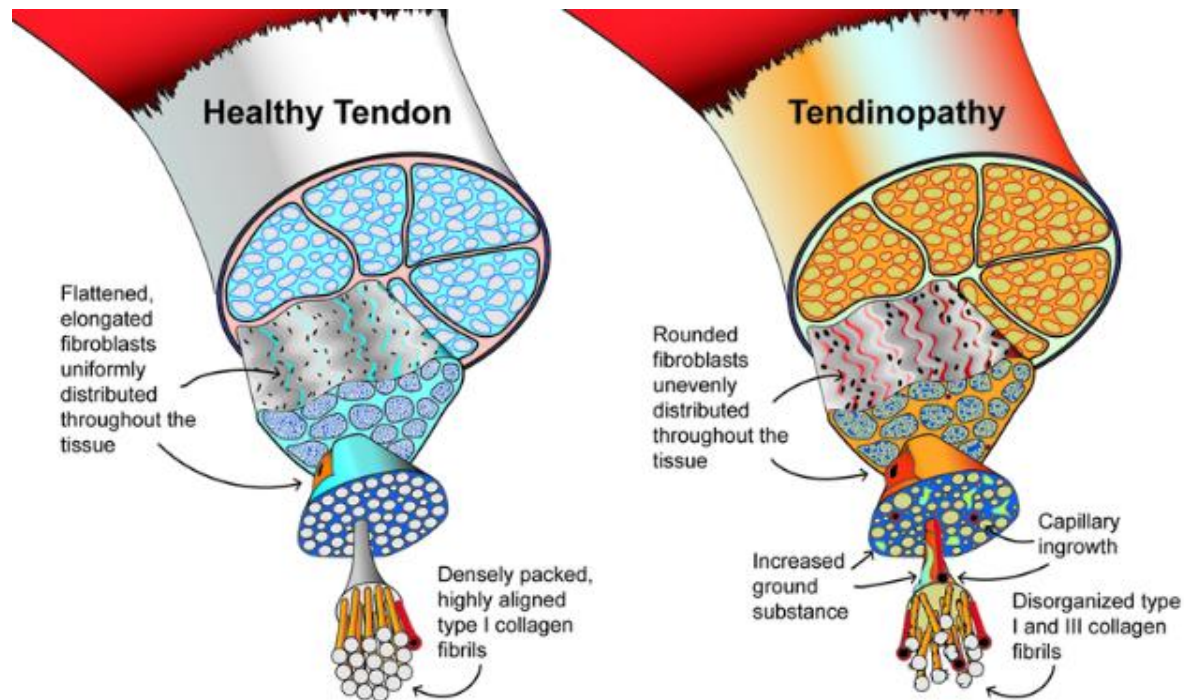
According to Fearon et al., (2012) "greater trochanteric pain syndrome (GTPS) is thought to be a degenerative condition of the gluteal tendons and bursa". And they add:

The development of GTPS is likely to be multifactorial in nature. Women with GTPS appear wider at the hips, than women without GTPS due to increased levels of peripheral adiposity and, in some cases, lower femoral neck shaft angles, rather than differences in bony width of the pelvis or hips. We found that women with gluteal tendon tears and with a lower femoral neck shaft angle are more at risk of failure of conservative management than those with a normal neck shaft angle. We also found that increased gynoid adiposity and pelvic width at the level of the superior aspect of the greater trochanter was associated with GTPS, supporting previous findings that gynoid adiposity is associated with tendinopathy. (Fearon et al. 2012).

Hallmark features

To start, it is important to graphically see the differences between a healthy tendon and a tendinopathy (figure 4).

Figure 4: morphological features in healthy tendon and tendinopathy



(Mead, et. al., 2018)

Each tendon has some classic signs and symptoms associated with presentation, and what we will do is discuss each tendon specifically.

People with Achilles tendon pain other than having localized pain and load dependent pain have morning stiffness and pain. This will be the case for a varying amount of time. And as we have talked about, the more activities you do the day before, the stiffer your tendon tends to be. We tend to look at these tendons being stiff for up to 30 minutes. If it is longer than 30 minutes, then we start to consider zero negative diseases. But this is such a classic sign of Achilles tendon problems. If somebody presents with Achilles tendon type pain and does not have morning stiffness and pain, then you can be relatively sure the problem is not the Achilles tendon.

Here is a summary of Graded Evidence in Achilles tendinopathy treatment, according to Chimenti et al., (2017):

No operative Treatment Recommendations:

- Eccentric exercise through a limited range of motion (ie, avoiding loading the tendon during ankle dorsiflexion) has been demonstrated to reduce pain and have a high level of patient satisfaction (Grade B recommendation).
- Extracorporeal shock wave therapy can reduce pain at 4-month to 18-month follow-up (Grade B recommendation). However, there is currently no evidencebased standardized ESWT protocol for IAT, some patients may not be satisfied with the treatment due to pain during the treatment, and effectiveness may be limited in patients with enthesophytes.
- There is currently insufficient evidence to make a recommendation about the use of night splints as one component of a home exercise program (Grade I recommendation).
- Injections may be used to target inflammation in tissues surrounding the tendon or neovascularization within the tendon. However, there is insufficient evidence in patients with IAT to support long-term efficacy (Grade I recommendation).
- Although only supported by theoretical and anecdotal evidence (Grade I recommendation), there are additional pain-relieving options that can be used in the clinic. Changes in footwear, such as using a heel lift, are often recommended to alleviate pain in patients with IAT. Anti-inflammatories, iontophoresis, and/or ice can also be used to alleviate pain due to inflammation associated with IAT. However, there is insufficient evidence to support more than a Grade I recommendation.

Operative Treatment Recommendations

- After failure of conservative care, open debridement and decompression for IAT is supported by many studies that report high postoperative function and patient satisfaction (Grade B recommendation).
- Patients who are candidates for endoscopic/minimally invasive procedures may have fewer wound-healing complications than open procedures, but there is currently insufficient evidence on postoperative outcomes to make a recommendation about the routine use of this procedure (Grade I recommendation), especially in patients with insertional calcification.
- Although an FHL transfer can be used to augment the Achilles tendon after debridement, there is currently insufficient evidence to support the routine

use of this procedure when initially operating on patients with IAT (Grade I recommendation).

- There is conflicting, low-level evidence on the effectiveness of isolated gastrocnemius recession to reduce pain and improve function in patients with IAT (Grade I recommendation). More research is needed to understand the efficacy of a gastrocnemius recession as an adjunct to other operative procedures.
- There is currently no literature on the efficacy of percutaneous procedures in patients with IAT (Grade I recommendation). (Chimenti et al., 2017, pp. 1165-1166)

And we have to sort out differential diagnosis issues for the patella tendon. The hallmark sign is sit pain, particularly in the car. These people, passengers, either particularly in the back seat, or drivers, often cannot sit for more than 10 or 20 minutes and they have to get out and straighten their knee. This can be a sign of patella femoral pain. So you have to have other differential diagnosis strategies in place. But patella tendon pain cannot be modified without getting out of the car. These people also hate stairs, particularly in the morning or the day after they have done some activity. Those are the hallmark signs of Patellar tendon.

For the hamstring tendon, these people dislike sitting on a hard chair and they cannot be sitting during any period. They will complain that this type of seat has a negative effect on the period of time they can be sitting. It is classic to ask them to sit for a long period on a hard chair: they will not be able to do it, but they can modify their position or modify their chair and be able to sit for longer periods. That is like a classic hamstring tendon sign.

For the glut med tendons or the gluteus tendons. This occurs in older postmenopausal women. They have again, classic signs, the most important being having trouble to sleep at night, sleeping on either side. If they sleep on their affected side, they feel pain; if they sleep on the unaffected side, they have pain because of the compressive loads of the tendon. These women will describe the trouble that implies getting to sleep or waking up several times at night, being more comfortable on their back, but often unable to sleep in that position. These women also describe disliking going up and down stairs and climbing up and down sloping ramps. Those are the hallmark signs of good heel tendon problems.

Malliaras, et al. (2015) proposed that the hallmark features of patellar tendinopathy consist of the following:

- (1) pain localized to the inferior pole of the patella and (2) load-related pain that increases with the demand on the knee extensors, notably in activities that store and release energy in the patellar tendon. While imaging may assist in differential diagnosis, the diagnosis of patellar tendinopathy remains clinical, as asymptomatic tendon pathology may exist in people

who have pain from other anterior knee sources. A thorough examination is required to diagnose patellar tendinopathy and contributing factors. Management of patellar tendinopathy should focus on progressively developing load tolerance of the tendon, the musculoskeletal unit, and the kinetic chain, as well as addressing key biomechanical and other risk factors. Rehabilitation can be slow and sometimes frustrating. This review aims to assist clinicians with key concepts related to examination, diagnosis, and management of patellar tendinopathy. Difficult clinical presentations (eg, highly irritable tendon, systemic comorbidities) as well as common pitfalls, such as unrealistic rehabilitation periods and overreliance on passive treatments, are also discussed. (Malliaras et al., 2015)

According to Rudavsky & Cook “while pathology in a patellar tendon may not ever completely resolve, symptoms of patellar tendinopathy can generally be managed conservatively” (2014, p. 126).

These two authors also present information about active and passive interventions:

Active interventions: Intervention is aimed at initially addressing pain reduction, followed by a progressive resistive exercise program to target strength deficits, power exercises to improve the capacity in the stretch-shorten cycle, and finally functional return-to-sport training. Daily pain monitoring using the single-leg decline squat, provides the best information about tendon response to load; consistent or improving scores suggest that the tendon is coping with the loading environment. (Rudavsky & Cook, 2014, p. 126)

Table 1: Suggested rehabilitation progression for patellar tendinopathy.

Table 2
Suggested rehabilitation progression for patellar tendinopathy.

Phase of rehabilitation	Aim of treatment	Intervention	Example exercises
Pain management	Reduce pain	Isometric exercises in mid-range as tolerated. Reduce loading and activity modification	Sustained holds on leg extension; 45 s, 4 repetitions, 2 times/day.
Strength progression	Improve strength	Heavy slow resistance as tolerated (isotonic)	Leg extension/press, 4 sets of 6-8 repetitions, 3-5 times/wk
	Functional strengthening	Progressive resistance exercise program, functional tasks, address movement patterns, kinetic chain and endurance training as required	Walk lunge with body weight or extra weight, stair walking
	Increase power	Increase speed of muscle contraction, lower the number of repetitions	Split squats, faster stairs, skipping exercises
Energy-storage/ stretch-shorten cycle	Develop stretch-shorten cycle	Plyometric exercises, graded gradually	Jumping, deceleration and change of direction tasks
Maintenance	Training sport-specific	Drills specific to sport including endurance training	Sports specific drills at set intensity and duration
	Management of symptoms and prevention of flare ups	Education, continue strength training and manage loading as tolerated	Continue leg extension strength or Spanish squat exercise while training and playing

(Rudavsky & Cook, 2014, p.126)

Passive Interventions: Manual therapy techniques, including myofascial manipulation of the knee extensor muscle group, have had a positive effect

on reducing pain in patellar tendinopathy patients in short-term and long-term follow-up.⁵¹ Other passive therapies, including braces and taping techniques, are often used clinically to help unload the patellar tendon, however, no evidence supports their efficacy. Passive therapies are best used to reduce symptoms in season so the athlete can continue to participate in rehabilitation and sport (Rudavsky & Cook 2014,)

Differential diagnosis

The history and examination are crucial to distinguish patellar tendinopathy from other diagnoses including: patellofemoral pain; pathology of the plica or fat pad; patellar subluxation or a patellar tracking problem; and Osgood-Schlatter disease. (Rudavsky & Cook, 2014).

The key points you need to take from a history are: where someone's pain is, when they load, and what loads provoked their pain. This is so because tendon pain has two critical features that are really important in the lower limb: Tendon pain remains incredibly localized, it does not move or spread, regardless of the length of time of symptoms, and it is associated with high load on that tendon. So, it is important to understand what high load for tendon is, when you ask for tendon to store and release energy like a spring, or to do something fast. So by definition, something that is slow or static is not difficult to deal for a tendon; it can be very heavy, it can be hard for the muscle, but it is not hard for the tendon.

We are interested in knowing where the pain is (it is very specifically localized), and what type of loads provoke their pain. For the Achilles, these are loads like a change of direction. Now our running loads, our faster activities, like hopping when we consider the fatality end, our jumping loads are of fast change direction, like when you are lunging at a tennis net. It does not correspond to activities like running.

So, clinically, you need to be very suspicious of someone with a knee pain. We should ask 'where is the pain located, and what are the loads we should be suspicious about?' It is really important that you ask them what changed in the last time that could affect their tendons, which not necessarily is what you expect. Under loading. So, for example, if someone has a capacity that is quite good and they overload the tendon in some way, that is, they increase their load above their capacity, that can produce some symptoms. A common symptom is unloading, resulting from pain, which, in turn, makes their capacity drop. Now, when they go to load again, they may not think they are overloading because they might have previously completed that sort of activity, but now it corresponds to overload on the basis of their new capacity.

This is why we need to find out what changed in patients' history: to better understand their situation and help them recover their debt load loading capacity relationship. Being aware

of changes is also critical because if you cannot identify a change in load or some sort of change in their history, then you might be dealing with some sort of systemic driver in relation to their tenant and apathy. You might have a non-load related tendon. You might have a differential diagnosis, but you might have a non-work related tenant apathy. Understanding changes is important because the patient might need you not only during their rehabilitation of load capacity, but also (and more importantly) they might need you to get their disease process managed first. And a lot of patients with non-load related tenant openings actually have their situation aggravated by load. This is why they are important to recognize.

According to Rio, et al., differential diagnosis is complex:

as there may be pain from more than one structure. The site of pain may be a guide to the structures involved (Table 1) and there are several key clinical history questions that will guide the clinician to the likely source of pain (Table 2). The most important questions to ask are 'Where is your pain? When did it start and what were you doing when it started?' Mechanical causes of heel pain are often brought on by a change in activity or a change in shoes. A specific incident or trauma to the region will guide further questioning around the forces and potential sources of pain. (Rio et al., 2015)

Table 2: Possible structures and sites of pain

Site/type of pain	Common sources of pain	Less common sources of pain
Posterior	• Achilles tendon insertion • Superficial calcaneal bursa • Posterior impingement of soft-tissues/os trigonum in active people • Calcaneal apophysis in adolescents	• Sural nerve
Inferior	• Plantar fascia • Calcaneal fat pad	• Medial or lateral calcaneal nerve, especially as they split from the tibial branch
Medial	• Tibialis posterior tendon and sheath • Tibialis posterior insertion and apophysis in adolescents	• FHL and sheath • Abductor hallucis • Deltoid and spring ligaments • Posterior tibial nerve in tarsal tunnel (is associated with neural symptoms such as tingling) • Bone: medial malleolus
Lateral	• Lateral ligaments of the ankle • Sinus tarsi	• Peroneal tendinopathy or tenosynovitis associated with subluxation • Cubometatarsal joint • Peroneus brevis insertion/apophysis of base of 5th metatarsal in adolescents or after ankle sprain
Deep, vague pain	• Subtalar joint	• Bone pain: calcaneus, talus, navicular

FHL, flexor hallucis longus

(Rio, et al., 2015)

Table 3. Subjective assessment questions to direct clinical reasoning

Question	Common responses	Diagnoses to consider
Age	Young	Common: Sever's disease (calcaneal apophysitis) Uncommon: calcaneal stress or tumour
	Middle-aged	Common: Achilles insertion tendinopathy, plantar fascia pain
	Older	Common: tibialis posterior tendinopathy and lengthening
What aggravates the pain?	Mechanical causes (eg walking, running)	Insertional Achilles tendinopathy (note, a warm-up phenomenon with activity is reported) Pain that increases with activity may indicate: • involvement of sheath (paratendinitis) • bone (stress reaction or stress fracture) • sinus tarsi or neural sources (including tarsal tunnel); prolonged standing can irritate tarsal tunnel.
	Non-mechanical	Pain at rest should be questioned further in terms of positioning and neural symptoms Tendon pain at rest is uncommon but pain on rising after sitting is a hallmark sign of tendinopathy
Was the onset of pain associated with an incident?	Yes: change to weight-bearing load (eg running or footwear)	Achilles tendinopathy Calcaneal bone stress
	No	Consider arthritic causes
Pain behaviour	Morning pain and stiffness	Achilles tendinopathy, FHL tenosynovitis, plantar fascia pain Long time to warm up (>60 minutes): consider rheumatological cause
	Night pain	Bone stress (eg calcaneal stress or more sinister causes)
General health questions and red flags	Night pain Other flags such as loss of weight, night sweats, joint pain and swelling	Tumour Consider non-musculoskeletal cause and include rheumatoid arthritis, gout, spondyarthropathies, infection
	Past injury	Repeated ankle sprains can commonly cause posterior impingement and sinus tarsi syndromes
Other	Cramping (calf and feet)	Vascular claudication Can be an early indicator of bone stress
	Neural symptoms	Sharp pain, burning, 'pins and needles' or numbness indicate neural involvement (eg tarsal tunnel syndrome (posterior tibial nerve and branches) present with symptoms in posteromedial ankle and heel and may extend to distal sole and toes)

(Rio, et al., 2015)

As for differential diagnosis for High Hamstrings Tendinopathy, when a running athlete presents with buttock or posterior thigh pain, the differential diagnosis can be very challenging, even for the most astute clinician. A thorough history and examination, complemented by the appropriate diagnostic studies, can help guide the clinician to an accurate diagnosis.

The first step is to determine whether the pain is local or referred. Diffuse pain that varies in location is indicative of referred pain. Buttock pain associated with low-back pain suggests referral from the lumbar spine; however, as in our illustrative case, sometimes the cause of pain can be both local and referred. Pain in the lumbar spine can refer from the disk, zygapophyseal joints, muscles, or ligaments. More localized, fairly constant pain is more likely to stem from pathology in the buttock itself. Pain localized to the ischial tuberosity is consistent with proximal hamstring tendinopathy, but it could also represent ischiogluteal bursitis or, in children, an apophysitis or avulsion. If the pain is more proximal (in the upper gluteal region and medial to the greater trochanter), the piriformis muscle may be the pain generator. Pain from the piriformis may be myofascial or from muscle strain. Local and referred pain from pressure on the sciatic nerve caused by its aberrant course through the piriformis muscle is commonly called piriformis syndrome, although piriformis impingement may be a more accurate term. Pain directly over the sacrum or along the sacroiliac joint may derive from sacroiliac joint mal alignment, a pelvic stress fracture (typically the sacrum or, less commonly, the ilium), or inflammation of the sacroiliac joint, often associated with spondyloarthropathy. Less common causes of diffuse exercise-induced buttock or posterior thigh pain include chronic compartment syndrome of the posterior thigh, endofibrosis of the external iliac artery, or a pseudo aneurysm of the inferior gluteal artery resulting from iatrogenic or accidental pelvis trauma (Fredericson et al., 2015).

Remember that some of our tendons have specific features that are unique to that tendon. So we say that night pain is not common on tendinopathy, being glute med the exception. We say that tendon pain remains very localized. Again, glute med being the exception. And that is because a lot of the big bursa around that greater trochanteric area can refer down into the leg, usually not past the knee, but they can certainly have some referral.

So the reason why people with gluteal tendinopathy experience night pain, is because of the compression. Either the direct compression lying on that side or the top leg drops down into adduction. It is important that we recognize there are always exceptions to the rule. Patients that we see with gluteal tendinopathy are often our postmenopausal women. This tendinopathy is a diagnosis that can coexist with other symptoms; so, you can have it as well as some low back issues and some hip joint issues.

A fantastic question for differentiating hip issues from gluteal tendinopathy is: 'Do you have problems putting or taking your shoes on and off?' Someone with a hip joint restriction will have an issue taking their shoes on and off, but someone with gluteal tendinopathy will not.



24 hours response

When you have a patient who suffers from tendon pain, you should educate them on when to listen to their tendon, since it will 'talk' to them. Moreover, it is important to know when to listen and what we are listening to.

The most important time to listen to a tendon is 24 hours after you have put a load on it, because that is when it will tell you whether it is 'happy'. Therefore, our 24-hour load response is a critical assessment tool to use right from our subjective assessment all the way through our rehabilitation.

In our subjective assessment, it is a vital question to establish tendon pain. If you do more today, are you worse the next stakes? That is a hallmark feature. However, when we are going through rehabilitation, it is also one of our best monitoring tools.

We have tools that monitor people for over weeks or months, such as our patient reported outcome measures. Nevertheless, we need something that is more sensitive to change and sensitive to load. Therefore, what we do is to ask people to listen to their tendon after they have placed a load on it. We teach people that if their tendon is happy, pain will be low and stable. Therefore, what we are looking for is actually stability of symptoms, once we have settled someone from the initial peak.

We are after pain reducing in that initial phase. We have removed the compression and we have started some isometric and some tonic loadings, we have applied loading that is very safe for the tendon, which is nice and slow or static. We have removed anything that is fast and anything that is in compression. So we are looking for symptoms to come down and we would assess this on their 24 hour response once their symptoms have come down. Our intention is to increase the load that we place on the tendon. And the reason why I say low and stable rather than zero is that some people can have a low level of symptoms for years. But if they are doing everything that they would like to do and the symptoms are only for a few minutes each morning, then this is actually a happy tendon.

So, how do we know when tendons are not happy? 24 hours after you put the load on the tendon, you will see a spike in their load in their provocative low test. So our low test for the killings might be morning pain and stiffness in terms of a time-based measure, or if it is the case of an athlete, it could be a hop test. We need to make sure it is appropriate for their level of function. Our best 24 hour load response tests for the patella tendon is the single leg daikon squat. If the tendon is happy, we will see symptoms are stable or even better, they are improving. And it is important that you educate patients on the fact that they actually have a happy tendon. And we are really looking to slowly increase the load. If our tendons are not happy, we will see it 24 hours later.

Now, why do we listen to tendons 24 hours after a load? Well, you might recall that tendons have a warm-up phenomenon, which means that, as people start to exercise, they can have



some discomfort, but after the warm-up, they will feel quite good. So, what we would like to avoid is that people listen to their tendon during exercise and that they base how much they are doing on how they feel at the time, because that actually can be really misleading. What we want to do is progress people's load in response to their 24 hour load test. And we do that.

Typically, throughout rehabilitation, our pain will be reducing in the initial phases because our stage 1 and 2 are not provocative loads; they are slow and they are static loads, they are very safe. This puts a lot of load on our muscle when we start to add speed in stages 3 and 4; that is when we really have to make sure that we are listening to our tendon in that 24 hour response. We keep our 'eye sensitive stage' going throughout this phase. And what we do is to keep our faster loads on alternate days, and the reason is it gives us the opportunity to listen to our tendon and the opportunity to realize if we have put too much load. If we did our faster loads each day, we may have a cumulative effect and we might have a large increase in pain, and we can really get into trouble with our rehabilitation. This is why when we are changing things in rehabilitation we change one thing at a time. In that way we know exactly what the change was and whether there is an increase in pain. We know the provocative change that we made and we can address it really simply. If we change multiple things and 24 hours later we are much worse, we will not know which particular aspect it was that caused something or whether it was because of a combination. So, it is really important that we change one thing at a time.

One of the questions that people commonly ask is "what do we do if we see a change in our 24-hour load response?" "What happens if we provoked someone's pain?"

It is important here to educate your athlete. We need to make sure we continue applying our safe loads. Therefore, you should remember that our safe loads are, analgesic, static, and slow (isometric and isotonic).

All in all, what we teach people to do is that, if they see an increase in pain or an increase in their morning stiffness, we do not want them to rest. What we want them to do is add back in the isometric load if they have seen this and continue with their gym loads, and then, the next time, they try that fast, start energy storage. So, in stage 3 or 4 exercises, what we would do is to reduce the load, reduce the number that we always ask someone to do or make some sort of change in response to their provocation. Whilst decreasing pain is a nice indication that we should continue to increase our stage 3 and 4 exercises in terms of our number, when we see a spike in pain, we also need to know how to manage it and how to educate our athlete. And we need to make sure that we avoid complete rest. The patient should never completely rest. We just need to recognize that high tendon load was perhaps too much for what someone was ready for at a certain time.

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