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INJURIES AND TEAM SPORTS

TENDINOPATHY IN SPORTS

Tendinopathy in Sports

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1. Definition

For years the word *tendinitis* was used to encompass anything pathological located in the tendon, assuming that there was an inflammatory basis that justified the use of this term. Subsequently, histological and biochemical studies ruled out the existence of inflammatory markers significant enough to cause symptoms, while, on the contrary, they did reveal a marked disorganization and degeneration of the collagen fibers of the yellowish tendon (this macroscopic appearance is called *myxoid* or *mucoid degeneration*), variable fibrosis and the presence of new blood vessels and nerve fibers that could explain the painful symptoms, which we prefer to call *tendinosis* (Fu, SC, Rolf, C., Cheuk, Y. C., Lui P.P., Chan KM, 2010; Maffulli, Khan, and Puddu, 1998). According to the anatomical location of the discomfort, they can be classified as *insertional tendinopathies or enthesopathies* when the injury is in the insertion where the tendon joins with the bone, and as *tendinopathies*, as a general term, when the discomfort is located in the tendon.

This clinical framework can be complicated by inflammation, on this occasion, of the outer sheath of the tendon, the paratendon. The inflammation of this conjunctive sheath can occur in isolation and is called *peritendinitis* or associated with a tendinosis: *tendinosis with peritendinitis* (Brukner and Khan, 2007). In a generic way, today there is unanimous consensus that the term *tendinopathy* defines any clinical issue that affects a tendon.

2. Epidemiology

The growing number of recreational athletes and the high demands in professional sport mean that there is a high prevalence of tendinopathy among all sports groups. In professional soccer, for example, patellar tendinopathy represents 1.5% of all injuries, with an incidence of 0.12 injuries per 1000 hours and up to 20% relapse (Hägglund, Zwerver, and Ekstrand, 2011). Achilles tendinopathy represents 2.5% of injuries, with an incidence of 0.18/1000 hours and up to 27% recurrence (Gajhede-Knudsen, Ekstrand, Magnusson, and Maffulli, 2013). These two tendinopathies have a much higher incidence and prevalence in sports such as basketball and volleyball, hence the patellar tendinopathy is known as *jumper's knee* (de Vries, van der Worp, Diercks, van den Akker-Scheek, and Zwerver, 2015; van der Worp, van Ark, Zwerver, and van den Akker-Scheek, 2012). Distal tendinopathy of the iliotibial band is common in long-distance runners (*runner's knee*). Rotator cuff tendinopathy is more common in *over-head* throwing sports such as handball, water polo, baseball, among others (Lewis, 2009), thus, the vast majority of tendinopathies relate to a specific athletic motor pattern and some are more common than others according to the sport that is practiced (technique-based pathologies).

3. Pathophysiology

Although different theories have been proposed to explain the pathophysiology of tendinopathies, in our group we accept the validity of the *tendinopathy continuum* model described by Cook and Purdam (2009), which includes three pathological stages in the tendon: reactive tendinopathy, tendinopathy in which repair mechanisms fail and degenerative tendinopathy (Figure 1). Although three different stages are described, there is a continuity and an overlap between them.

a) Reactive tendinopathy: is characterized by a cellular proliferative and non-inflammatory extracellular matrix response, due to mechanical compression and distraction forces on the tendon, which causes a relative thickening of a part of the tendon. This phase is reversible if the mechanical load on the tendon disappears.

b) Failed reparative tendinopathy: increase in the proliferation and number of cells (chondrocytes and myofibroblasts) together with an increase in the production of proteins (proteoglycans and collagen) that leads to the disorganization of collagen fibers, with an increase in type III collagen fibers and increase of the extracellular matrix together with a neoformation of blood vessels and nerve endings. This neovascularization seems to be implicated in the generation of pain, but this is still a matter of debate in the medical community (Dean, Gwilym, and Carr, 2013). This stage is reversible if the physical loads are controlled and the appropriate exercises are performed (which will be discussed later).

C) Degenerative tendinopathy: the disorganization of the collagen fibers and restructuring of the extracellular matrix and cellular changes progress: apoptosis in cellular islets, degeneration of the tenocytes and areas of acellularity and neovessels. The reversibility of this stage is very difficult and chronic pain is common and caused by cytokines, pain mediators, hypoxia phenomena and pH changes.

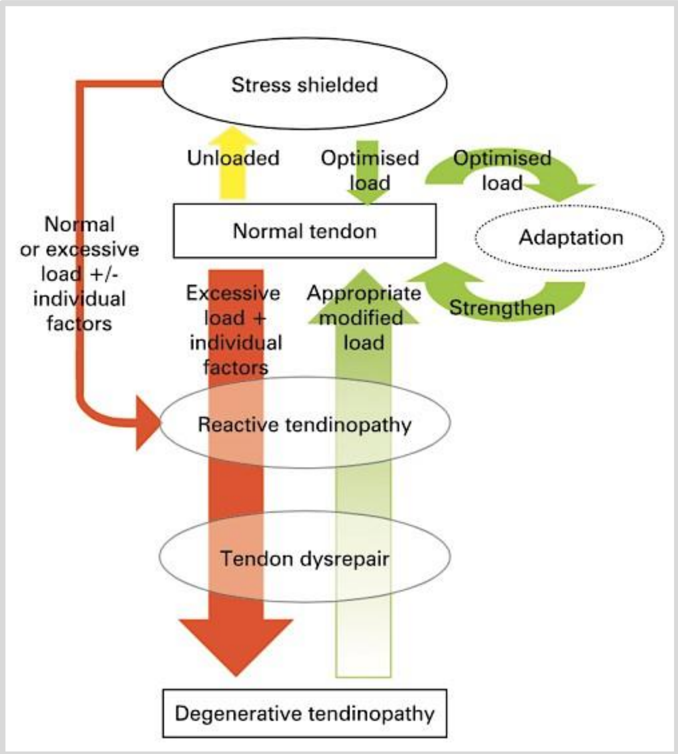


Figure 1: "Tendinopathy continuum" model described by Cook and Purdam

Source: Cook & Purdam (2009)

Model of "tendinopathy continuum" described by Cook and Purdam (2009) that includes three stages: reactive tendinopathy, tendinopathy in which repair mechanisms fail and degenerative tendinopathy.

4. Etiology and risk factors

Tendinopathy has a multifactorial etiology and risk factors are often divided into intrinsic factors, those that act from within the body, and extrinsic factors, those that act on the body (Cook and Purdam, 2014; Malliaras and O'Neill, 2017).

Among the *extrinsic factors*, it is important to bear in mind errors in training programming. An increase in the intensity of work sessions or an increase in the total training volume can cause the tendon to adapt poorly to the imposed workloads and can lead to an injury (tendinopathy). Repeated intense workloads (understood as compressive and traction loads) without sufficient recovery time (e.g., skipping the necessary recovery between sessions) may be a risk factor in tendon pathology. In insertional tendinopathies it has been suggested that reducing the pressure on the enthesis is important for prevention and treatment. For example, in the case of the

Achilles tendon, this can be achieved by using a supine heel elevator (Malliaras and O'Neill, 2017).

Multiple *intrinsic factors* have been proposed. Systemic factors for Achilles tendinopathy include being overweight, insulin resistance, type 2 diabetes and Hypercholesterolemia. Genetic predisposition and existence of previous tendon injuries have also been suggested.

In any case, it does seem to show a fairly direct relationship with mechanical factors. The horizontal jump landing is associated with greater impact on the patellar tendon than the vertical landing. Foot posture and function (dynamic pronation) have been proposed as risk factors for tendinopathy of the lower limbs. Increased and decreased range of dorsiflexion movement of the ankle have been associated with the development of Achilles tendinopathy in prospective studies. Patellar tendinopathy has been associated with both an increase and decrease in hamstring muscle flexibility (Malliaras and O'Neill, 2017).

5. Clinical and diagnosis

The symptomatology of tendinopathies can be directly related to one of the stages proposed by Blazina, Kerlan, Jobe, Carter and Carlson in 1973. Depending on the degree of involvement and the accompanying clinical symptoms, the tendinopathy will be classified into:

- Grade 1: pain appears only after physical exercise, as a painful response to the workload, to wane later with rest.
- Grade 2: pain appears at the beginning of physical exercise to disappear during the athletic activity and reappear after having completed physical work.
- Grade 3a: the pain is now continuous, interfering with the development of the exercise and a clear decrease in performance.
- Grade 3b: pain is constant, including during everyday activities.

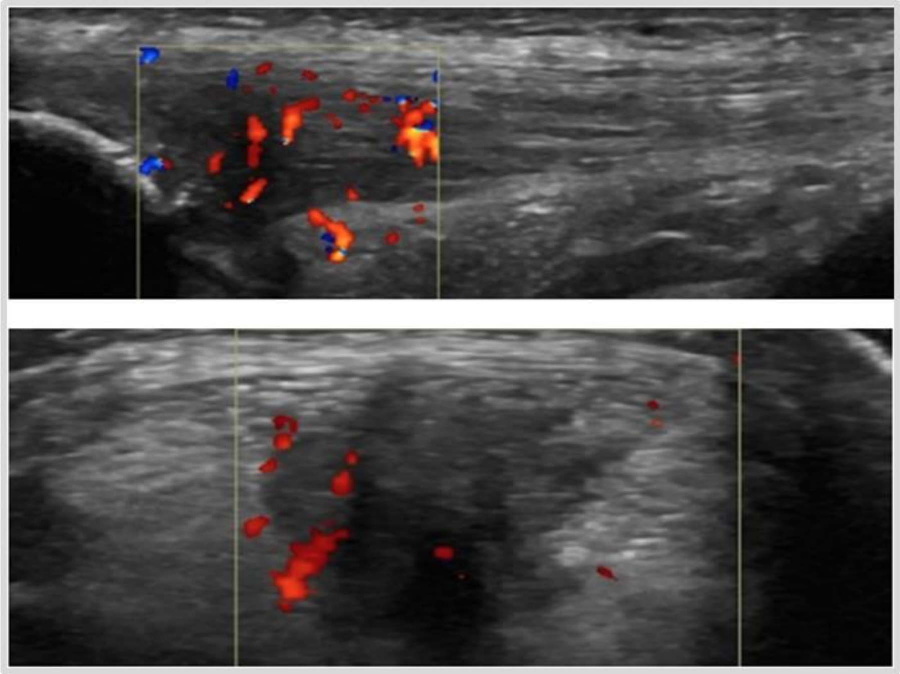
The athlete's *physical examination* will include assessing whether the range of mobility of the knee is painful, if the palpation and mobilization of the tendon arouses pain, if the tendon presents a thickening, if there is pain associated with stretching or if there is pain with active contraction or against resistance. In addition, in highly chronic cases, quadriceps may present a greater or lesser degree of atrophy.

There are two main complementary imaging tests: ultrasound and magnetic resonance imaging.

Ultrasound (Balius, Sala, Álvarez, and Jiménez, 2007) is an innocuous, non-invasive method that does not emit ionizing radiation, is economical (taking into account its low cost compared to other diagnostic tests) and allows the injured structure to be assessed both statically and dynamically, with the collaboration of the athlete who is asked to contract or relax their muscles. However, it is observer-dependent and its interpretation requires previous training in this diagnostic technique. The ultrasound can be accompanied by color Doppler to expose the presence or absence of neovascularization.

Under normal conditions, a tendon presents a clear fibrillar structure that represents the bundles of successively packed collagen fibers. There are tendons that are covered by a sheath of elastic connective tissue and between the sheath (paratendon) and the body of the tendon itself can be located, under normal conditions, some fluid. An ultrasound gives us images on the long axis of the transducer (longitudinal cuts) and on the short axis (cross sections). Figure 2 shows the characteristics of a pathological tendon ultrasound.

Figure 2: Longitudinal (top) and transverse (bottom) ultrasound of a pathological patellar tendon.

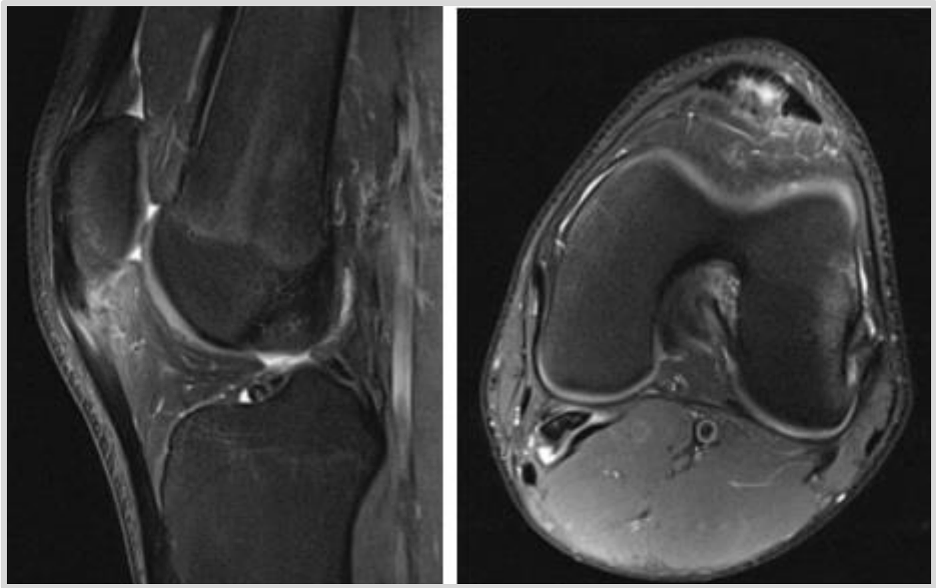


Source: Prepared by the author

Longitudinal (top) and transverse (bottom) ultrasound of a pathological patellar tendon. The image above shows a thickening of the tendon in its most proximal part (lower pole of the patella), with disorganization of the fibrillar pattern of its fibers and positive color Doppler activity that indicates the presence of new blood vessels that, accompanied by new nerve endings, have been postulated as being responsible for pain in tendinopathies. In the image below, the same fibrillar disorganization and color Doppler activity can be seen in the transverse view.

Magnetic resonance imaging (MIR) is a more economically costly diagnostic test, but provides more details regarding soft tissues. An MRI takes a long time to complete. The images are static and are shown on three planes: axial, coronal and sagittal. This test is not dependent on the observer and is useful for assessing injuries associated with the tendon process. Figure 3 shows the characteristics of an MRI for examining tendinopathy.

Figure 3: T2 fat sat image of a sagittal (left) and axial (right) section in a knee with patellar tendinopathy.



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In both images, an intratendinous signal change (white hypersignal) can be seen, which reveals the disorganization of the collagen fibers in the patellar tendon.

6. Management and treatment

In a tendinopathy it will be important to differentiate the treatment (medical or physiotherapy) from the acute treatment phase (based primarily on physiotherapy and gym) of a chronic tendinopathy.

Finally, in cases of persistent tendinopathies that do not respond to any conservative measures, surgical treatment is the last option.

6.1 Acute tendinopathy

Non-steroidal anti-inflammatory drugs (NSAIDs): although there is very little evidence regarding the existence of an inflammatory response in tendinopathies, a short course (7-14 days) of NSAIDs in reactive tendinopathy may be effective as the first line of treatment in terms of controlling the initial pain. NSAIDs have shown to be less useful in cases of chronic tendinopathies.

Analgesics and local anesthetics: few analgesics are powerful enough to control pain and the use of local anesthetics injected into the affected tendon is not an advised practice.

Corticosteroids: local injection of corticosteroids seems to show a certain effectiveness in the short term when it comes to reducing pain, but its effectiveness in chronic tendinopathies is much more doubtful. In addition, the poor reputation of local infiltrations is due to their use in inappropriate phases (degenerative stage) as well as the intratendinous injection with its associated risk of rupture within the tendon.

Sclerosing agents (polidocanol) and injection of large volumes of saline: have been used for the purpose of reducing pain through neurovascular disruption (veins that appear in chronic tendinopathies), but there is still very little evidence to justify their use within the first line of therapy.

Platelet-rich plasma (PRP) is a biological therapy with very promising experimental results, there are also biological therapies with a regenerative purpose such as *stem-cell therapy.*

Local injections of *aprotinin*, an inhibitor of extracellular matrix metalloprotease activity, or *prolotherapy*, injections of various irritating substances (phenol), osmotic agents (dextrose) or sclerosing agents (sodium morrhuate) have shown minor and inconclusive results.

Extracorporeal shock wave therapy: not indicated as an option in the first line of treatment – its use is reserved for chronic tendinopathies that are more impervious to conventional treatments; good results have been reported in up to 74% of patients without having to interrupt their sporting activity (van Leeuwen, Zwerver, & van den Akker-Scheek, 2009). The action mechanism disrupts the new nerves and blood vessels existing in chronic tendinopathies, as well as inducing the proliferation of tenocytes.

6.2 Chronic tendinopathy

Tendinopathy is understood not as a transient inflammatory process of the tendon, as previously indicated, but as a chronic pathology that evolves according to the previously proposed stages and that requires constant management of painful exacerbations using the therapeutic tools outlined in the previous paragraph – above all through proper management of the various physical training loads and with the introduction of a series of exercises to perform in the gym. From a biochemical perspective, the objective would be to reduce the activation or sensitization of tenocytes.

Storing elastic energy increases cellular signals and very high physical loads generate cell death. Reducing cellular activity can cause a decrease in the release of cytokines and neuropeptides and the deposit of proteoglycans in the extracellular matrix, so that future disruption of the matrix would be prevented and the progressive tolerance to workloads would be increased.

The reduction of both compressive and tensile physical loads is especially important since not doing so would trigger a reactive response (stage one of the three-stage model) (Cook, & Purdam, 2014). Eccentric work, which has been so strongly advocated as key in the prevention and treatment of tendinopathies, becomes a dangerous weapon if it is added to training without first reducing the general physical workload in the training. In addition, a direct contusion on the body of the tendon also induces a reactive response within the tendon. Also, stretching seems to be counterproductive in the insertional tendinopathies of adductors, Achilles and hamstrings (Cook, and Purdam, 2012).

It is clear then that the high workloads that cause tendon pain must be eliminated, but these, at a lower intensity, should be introduced as soon as possible and a certain workload stimulus must be maintained, because if not, a tendon that does not receive any physical workload enters into a process of catabolism and degeneration (Arnoczky, Lavagnino, and Egerbacher, 2007; Kubo et al., 2004). Therefore, the most appropriate type of work for this purpose has been studied and there is scientific evidence that confirms that isometric exercise, when there is pain, manages to generate an analgesic situation. In patellar tendinopathy, researchers have studied the effect of five sets of quadriceps isometric work (60º of knee flexion to 70% of the maximum voluntary contraction) of 45 seconds duration, and it was found that, in the 45 minutes immediately after this intervention, analgesia is produced by inhibition mechanisms (Rio et al., 2015). It is advisable to do this work every day throughout the season with athletes with known patellar tendinopathy, along with the individualization of workloads, which will allow the athlete to train and compete normally. Although initially this exercise was described using a *knee extension machine*, a valid alternative could be a double-leg *squat* at 70-90º knee flexion with a muscle brace (Figure 5). A useful, quick and objective method for the athlete to monitor the clinical evolution of her tendinopathies is through physical pain provocation tests, as proposed by Cook and Purdam (2014), as shown in Figure 4.

Figure 4: Pain provocation test for the clinical monitoring of a tendinopathy, taken from Cook and Purdam (2014).

Table 2 Provocative clinical tests useful to monitor tendon pain

Tendon	Low-load clinical test	High-load clinical test
Achilles	Single leg heel raise	Hop
Patellar tendon	Decline squat	High single leg jump, landing from a height
Hamstring tendon	Single leg bent knee bridge	Single leg dead lift
Gluteal tendon	Single leg stance	Hop

Source: Cook & Purdam (2014).

According to Malliaras, Cook, Purdam and Rio (2015), in the case of an athlete who has been removed from training and competition due to a disabling tendinopathy and once the "protocol" of isometric work is done, when reporting a maximum pain of 3/10, it is advisable to progress towards the performance of isotonic exercises that seek to increase muscle mass and strength thanks to the work of the entire range of joint movement. Ideally, the range of motion should be initially limited to between 10º and 60º of flexion and then increased up to 90º (Figure 5). Three to four sets are recommended with loads that allow approximately 15 reps until maximum fatigue, progressing by working every two days, increasing the load until it is possible to perform six reps before reaching maximum fatigue (Figure 5). Finally, and moving on to a phase where the aim will be the storage of elastic energy in the tendon, exercises will be carried out, such as a one leg squat (four sets of eight reps with 150% of body weight), always with a pain perception of less than or equal to 3/10 (Figure 5).

Figure 5: Workload progression model in patellar tendinopathy, extracted from Malliaras

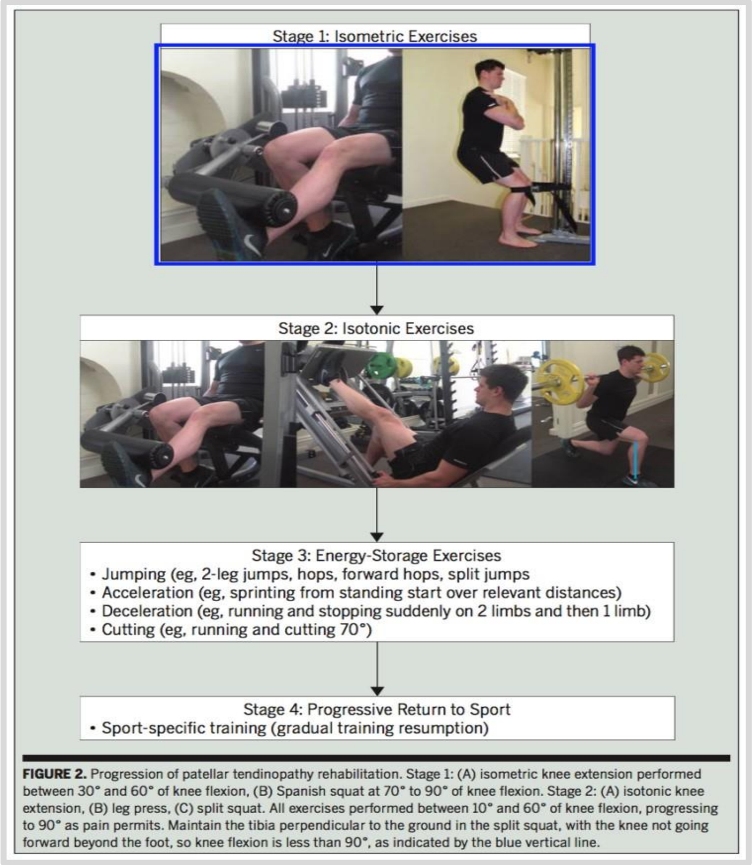

FIGURE 2. Progression of patellar tendinopathy rehabilitation. Stage 1: (A) isometric knee extension performed between 30° and 60° of knee flexion, (B) Spanish squat at 70° to 90° of knee flexion. Stage 2: (A) isotonic knee extension, (B) leg press, (C) split squat. All exercises performed between 10° and 60° of knee flexion, progressing to 90° as pain permits. Maintain the tibia perpendicular to the ground in the split squat, with the knee not going forward beyond the foot, so knee flexion is less than 90°, as indicated by the blue vertical line.

et al (2015).

Source: Malliaras et al (2015)

Figure 6: Workload progression model in patellar tendinopathy, extracted from Malliaras et al (2015).

TABLE — REHABILITATION STAGES AND PROGRESSION CRITERIA

Stage	Indication to Initiate	Dosage
1. Isometric loading	More than minimal pain during isotonic exercise*	5 repetitions of 45 seconds, 2 to 3 times per day; progress to 70% maximal voluntary contraction as pain allows
2. Isotonic loading	Minimal pain during isotonic exercise*	3 to 4 sets at a load of 15RM, progressing to a load of 6RM, every second day; fatiguing load
3. Energy-storage loading	A. Adequate strength† and consistent with other side B. Load tolerance with initial-level energy-storage exercise (ie, minimal pain during exercise and pain on load tests returning to baseline within 24 h)*	Progressively develop volume and then intensity of relevant energy-storage exercise to replicate demands of sport
4. Return to sport	Load tolerance to energy-storage exercise progression that replicates demands of training	Progressively add training drills, then competition, when tolerant to full training

Abbreviation: RM, repetition maximum.
**Minimal pain defined as 3/10 or less.*
†For example, around 150% body weight (4 × 8) for most jumping athletes.

Source: Malliaras et al (2015).

6.3 Surgical treatment

Surgery in cases of chronic tendinopathies (basically in Achilles and patellar tendons) is recommended only as a last therapeutic option when there has been no satisfactory result from conservative measures after a minimum of six months. In any case, it is unpredictable whether a patient/athlete's clinical problems will be solved even after the intervention; thus, it is advisable to insist that surgery is the last of the therapeutic resources that should be used.

The main objective of tendon surgery is to free the tendon of fibrous adhesions and remove dendritic intratendinous nodules, restoring vascularization and stimulating immature tenocytes to initiate a synthesis of new tendinous material and thus regenerate the injured tendon. Traditional surgeries require post-surgical rehabilitation of about six to nine months.

Also, in recent years, less invasive surgical approaches have been proposed (percutaneous tenotomy, some are even ultrasound guided), with the aim of reducing surgical aggression, a less painful postoperative outcome and reincorporating the athlete into training and competition in less time than after conventional surgeries.

In cases of insertional tendinopathies, there is no clear consensus about the best surgical option to treat them; there is also a debate about whether to use surgical techniques similar to those used for the tendinopathies of the tendon body or if it is appropriate to involve the bone where the tendinous fibers (osteotomies) are inserted (Marcheggiani et al., 2013).

References

Arnoczky, S. P., Lavagnino, M., & Egerbacher, M. (2007). The mechanobiological aetiopathogenesis of tendinopathy: is it the over-stimulation or the under-stimulation of tendon cells? *International Journal of Clinical and Experimental Pathology*, *88*, 217-226.

Balius, R., Sala, X., Álvarez, G., & Jiménez, F. (2007). *Ecografía musculoesquelética*. Barcelona, Spain: Paidotribo.

Blazina, M. E., Kerlan, R. K., Jobe, F. W., Carter, V. S., & Carlson, G. J. (1973). Jumper's knee. *Orthopedic Clinics of North America*, *4*, 665-678.

Brukner, P., & Khan, K. (2007). Sports Injuries, *Clinical Sports Medicine* (3. ed.). Sydney, AU: Mc Graw Hill.

Cook, J. L., & Purdam, C. (2012). Is compressive load a factor in the development of tendinopathy? *The British Journal of Sports Medicine*, *46*, 163-168.

Cook, J. L., & Purdam, C. R. (2009). Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. *The British Journal of Sports Medicine*, *43*, 409-416.

Cook, J. L., & Purdam, C. R. (2014). The challenge of managing tendinopathy in competing athletes. *The British Journal of Sports Medicine*, *48*, 506-509.

Dean, B. J., Gwilym, S. E., & Carr, A. J. (2013). Why does my shoulder hurt? A review of the neuroanatomical and biochemical basis of shoulder pain. *The British Journal of Sports* Medicine, *47*, 1095-1104.

De Vries, A. J., van der Worp, H., Diercks, R. L., van den Akker-Scheek, I., & Zwerver, J. (2015). Risk factors for patellar tendinopathy in volleyball and basketball players: A survey-based prospective cohort study. *Scandinavian Journal of Medicine & Science in Sports*, *25*, 678-684.

Fu, S. C., Rolf, C., Cheuk, Y. C., Lui P.P., Chan K.M. (2010) Deciphering the pathogenesis of tendinopathy: a three-stages process. *Sports Med Arthrosc Rehabil Ther Technol*, *2*, 30.

Gajhede-Knudsen, M., Ekstrand, J., Magnusson, H., & Maffulli, N. (2013). Recurrence of Achilles tendon injuries in elite male football players is more common after early return to play: an 11-year follow-up of the UEFA Champions League injury study. *The British Journal of Sports Medicine*, *47*, 763-768.

Hägglund, M., Zwerver, J., & Ekstrand, J. (2011). Epidemiology of Patellar Tendinopathy in Elite Male Soccer Players. *The American Journal of Sports Medicine*, *39*, 1906-1911.

Kubo, K., Akima, H., Ushiyama, J., Tabata, I., Fukuoka, H., Kanehisa, H., & Fukunaga, T. (2004). Effects of 20 days of bed rest on the viscoelastic properties of tendon structures in lower limb muscles. *The British Journal of Sports Medicine*, *38*, 324-330.

Lewis, J. S. (2009). Rotator cuff tendinopathy. *The British Journal of Sports Medicine*, *43*, 236-241.

Maffulli, N., Khan, K. M., & Puddu, G. (1998). Overuse tendon conditions: time to change a confusing terminology. *Arthroscopy*, *14*, 840-843.

Malliaras, P., Cook, J., Purdam, C., & Rio, E. (2015). Patellar Tendinopathy: Clinical Diagnosis, Load Management, and Advice for Challenging Case Presentations. *Journal of Orthopaedic and Sports Physical Therapy*, *45*, 887-898.

Malliaras, P., & O'Neill, S. (2017). Factores de riesgo potenciales que conducen a la tendinopatía. *Apunts Medicina de l'Esport*, *52*, 71-77.

Marcheggiani Muccioli, G. M., Zaffagnini, S., Tsapralis, K., Alessandrini, E., Bonanzinga, T., Grassi, A., Marcacci, M. (2013). Open versus arthroscopic surgical treatment of chronic proximal patellar tendinopathy. A systematic review. *Knee Surgery, Sports Traumatology, Arthroscopy*, *21*, 351-357.

Rio, E., Kidgell, D., Purdam, C., Gaida, J., Moseley, G. L., Pearce, A. J., & Cook, J. (2015). Isometric exercise induces analgesia and reduces inhibition in patellar tendinopathy. *The British Journal of Sports Medicine*, *49*, 1277-1283.

Van der Worp, H., van Ark, M., Zwerver, J., & van den Akker-Scheek, I. (2012). Risk factors for patellar tendinopathy in basketball and volleyball players: a cross-sectional study. *Scandinavian Journal of Medicine & Science in Sports*, *22,* 783-790.

Van Leeuwen, M. T., Zwerver, J., & van den Akker-Scheek, I. (2009). Extracorporeal shockwave therapy for patellar tendinopathy: a review of the literature. *The British Journal of Sports Medicine*, *43*, 163-168.