

Heavy Eccentric Loading for a Recalcitrant Case of Lateral Epicondylitis in a Hockey Player: A Case Report

Ian Macintyre, BSc, DC, CSCS
Graduate Education and Research, Canadian Memorial Chiropractic
College
6100 Leslie St, Toronto, ON
M2H 3J1
T: (647) 221-0853
E: imacintyre@cmcc.ca

Heavy Eccentric Loading for a Recalcitrant Case of Lateral Epicondylitis in a Hockey Player: A Case Report

Ian G MacIntyre BSc CSCS DC

ABSTRACT:

In the past, the observed positive effects of eccentric loading for chronic tendon pain have been attributed to increased tensile strength in the muscle or tendon or decreased muscular strain during joint movements due to a muscular lengthening effect. Alfredson et al.⁴ suggested that eccentric training might aid in correcting a neuromuscular disturbance caused by the initial injury, resulting in increased muscular strength.^{1,2,6} The recent findings of a local vasculo-neural in-growth in the chronic painful tendon has led to ideas around new treatment models, and pilot studies where sclerosing injections target the area with neovessels and nerves outside the tendon have shown promising results.^{4,5,6} Painful eccentric calf muscle training has scientifically shown to give good short-to-mid term clinical results in patients with chronic painful mid-portion achilles tendinosis, and a good clinical result seems to be associated with decreased tendon thickness and more normal tendon structure.^{1,11,26} Indications show that the eccentric training regimen might work by interference with the vasculo-neural in-growth, similar to what is accomplished with sclerosing injections using polidocanol. If this vasculo-neural in-growth is of major importance in the pathogenesis of the degenerated tendon, maybe destruction via eccentric training could start a remodeling process in the tendon. Non-operative treatment with eccentric exercises has been utilized successfully in chronic achilles, supraspinatus and patellar tendinosis. Since these different types of tendinopathies show similarities in clinical behavior and in their histopathologic appearance, eccentric training for lateral epicondylitis was designed to provide pain relief for a patient where many other treatment regimens have been unsuccessful. The findings in this case study indicate that our conservative regimen of eccentric training may reduce the symptoms in lateral epicondylar pain regardless of duration of symptoms and previous trials of therapy.

INTRODUCTION:

In the world of athletics and soft tissue overuse injuries, tendon pain and subsequent dysfunction is among the most common conditions which patient present. Unfortunately, due to a lack of scientific knowledge about the pain mechanisms associated with these conditions and confusion surrounding their pathogenesis, they remain to be one of the most poorly managed conditions. There is no doubt the nomenclature around the chronic painful tendon has been puzzling. At one time, tendon pain was considered to include an inflammatory component. Thereby

necessitating the use of the suffix “itis” for these conditions.^{1,11, 13,18,22,25} The implementation of methods such as biopsies, intratendinous micro dialysis and gene technology analyses of biopsies, have shown that there are no signs of prostaglandin-mediated inflammation in chronic painful tendons.⁶ Therefore, describing this injury as inflammatory by using such terms as tendonitis cannot be justified. The term tendinosis is used instead because it more accurately defines the histopathological presentation of the degenerative process. The characteristic appearance of biopsied tendon reveals

tissue, which has been invaded by immature fibroblasts and disorganized, nonfunctional vascular elements.^{3,4,5,6,18}

Electron microscopy has demonstrated that these vascular buds do not possess a lumen. This granulation-like tissue has been termed angiofibroblastic hyperplasia by Nirschl.¹⁷

Angiofibroblastic hyperplasia is intrinsically abnormal, and insinuates itself through the adjacent normal-appearing tendon fibers, disrupting them. Many studies have discussed the development of tendinosis in areas of poor or no blood supply.^{1,6,7,17,18,22,25}

However, these recent findings using Doppler ultrasound and biopsies, demonstrating vascular elements within the effected tissues, are in contrast to the hypothesis of hypovascularization and have lead to new innovations with regard to proper treatment protocols.^{4,5,6}

In 1929, John North prophetically stated that “the etiology of tendinosis is various, its pathology is obscure and its cure is uncertain.”¹⁵ Numerous treatment regimens have been purported to effectively manage chronic painful conditions, but it is important to state that there is sparse scientific evidence for most of the conservative and surgical treatments proposed and used. Even with this ambiguity, a non-operative treatment regimen is recommended as the initial strategy by most authors.^{1,7,10,11,13,15}

Nirschl et al.¹⁷ lists the primary goals of treatment of tendonosis as being pain control, preservation of motion, flexibility and strength, and the development of endurance over time. Since the exact cause of pain in tendonosis is not known, many theories

attempt to explain the mechanism of pain in order to improve treatment.

The original theory of pain is linked to the concept of tendonitis where inflammation is the source of pain. Many clinicians’ have, and still use, corticosteroid injections in attempt to alleviate the burden of tendinosis. The efficacy and risks of such treatments are unclear. Local steroid injection has been associated with numerous adverse effects, including tendon and fascial degeneration and possible rupture.^{12,16,17}

A critical review by Nichols et al.¹⁶ in 2005 revealed that the existing medical literature does not provide precise estimates for complication rates following such injections, and it should be emphasized that the literature does not provide clear evidence as to what constitutes a safe maximum number of corticosteroid injections. The common usage of such treatments calls for more precise estimates of complication rates following the therapeutic use of injected or systemic corticosteroids in the treatment of athletic injuries.^{12,16}

However, corticosteroid injections have been shown to be effective in short term pain reduction for tendinosis.¹⁶ The mechanism is unclear, but it is thought that bathing the area may alter or interfere with the local chemicals that cause pain stimulus in the area of the degeneration. Lewis et al.¹² states that steroid injections must be set in the context that they do not influence long-term outcomes, and the value of other treatments, such as physical therapy, should not be ignored. Since there are no inflammatory cells present in tendinosis, this theory should no longer apply to its proper management of chronic tendon-derived pain.

The literature has yet to fully elucidate the cause of pain in the absence of an inflammatory mechanism of pain production. Some have hypothesized that certain byproducts of increased cellular activity or tendon degeneration, such as lactic acid, glutamate and chondroitin sulphate act as biomechanical irritants that activate peritendinous nociceptors.^{2,7,22} Findings from several recent studies suggest a potential neurogenic pain mechanism rather than a chemical inflammatory condition.¹⁸

The next theory to evolve stated that it is biomechanical changes that result in pain production. Since a disruption of collagen in a ligament sprain is painful, it can be assumed that the disruption of collagen in a tendinosis can also be the cause of pain. Maffulli et al.¹³ argues this notion by comparing post surgical patients with patellar tendinopathy who experience pain against those who do not. He mentions that the area, when visualized, is indistinguishable between painful and pain-free subjects, which he believes negates a relationship between pain and collagen status. However, based on such theories, surgical and arthroscopic procedures have been developed in an attempt to release, repair or denervate the painful area. The arthroscopic findings of hyaline degeneration, subsynovial fibrosis and a leathery noncompliant tissue further illustrate a lack of an inflammatory condition. Much like a majority of treatment regimens for a tendinopathy, it is not uncommon that these procedures fail to alleviate symptoms with success outcomes seen in only 65%-90% of patients treated operatively, and up to 15% of these patients will go on to have refractory symptoms.¹⁴

It is clear that the successful treatment of tendinosis continues to depend on a thorough understanding of the structural injury, and on identifying the presence of additional co-morbidities that could contribute to a continuance or exacerbation of symptoms. Although the signs and symptoms of a classic tendinopathy are clear and its diagnosis easy, no ideal treatment has emerged in medical literature to date. There have been several conventional therapies suggested for treatment, including transcutaneous electrical nerve stimulation, braces, conventional physiotherapy, corticosteroid injections and surgery.^{19,20} These interventions are believed to relieve pain, promote tissue healing and improve joint mechanics. However, there is conflicting information regarding the effectiveness of these therapies. Three systematic reviews on these conventional therapies for tendon pathologies have been published in the Chochrane review series. All three reviews concluded that there was little evidence supporting the effectiveness of any of the conservative or surgical treatments.²³ Despite this enigmatic process of recovery, a non-operative treatment regimen is recommended as the initial strategy by most authors.

The majority of sport physicians and physical therapists agree that tendon overuse injuries are difficult to treat and they continue to explore new treatment protocols for their athletes. Curwin and Stanish stressed the importance of eccentric training as part of the rehabilitation of tendon injuries.¹ However, the background to the good clinical results is unknown, although there have been several explanations in the past. It has been suggested that it is

the effects of loading-induced hypertrophy and subsequent increased tensile strength that eventually lead to a decrease of demand on the tendon. It may be an effect of stretching, with a “lengthening” effect of the muscle-tendon unit and consequently less strain during motion. Another theory is based on the fact that eccentric training is, and should be, painful to perform, and possibly this type of painful loading is associated with an alteration of the pain perception from the tendon.¹

Recently, Alfredson et al.⁴, in a study using ultrasonography (US) and color Doppler (CD), demonstrated that in achilles tendons with chronic painful tendinosis, but not in normal pain-free tendons, there was a neovascularisation outside and inside the ventral part of the area demonstrating tendon changes. Also, studies on tendon biopsies have shown nerve structures in close relation to the vessels.¹⁸ By using the US and CD, Alfredson et al.⁵ demonstrated during eccentric training of the achilles tendon that the flow in the neovessels disappeared when the ankle was in dorsiflexion and returned when the ankle was brought back to neutral. These observations raised the question whether the good clinical results demonstrated with eccentric training could be because of the action on the neovessels and accompanying nerves. Theoretically, the vessels and nerves could be traumatically damaged during the eccentric training regimen when traveling from outside the tendon into the dense tendinous tissue.⁶ Very recent results from Alfredson et al.^{4,6} have shown that in the majority of the patients that were satisfied with the result of the eccentric training regimen, there were no remaining neovessels, and in all patients

with poor clinical results, the neovessels remained.

In a follow-up experiment, Alfredson et al.⁵ evaluated the possible importance of the neovessels and accompanying nerves by injecting local anesthesia in the area, resulting in a pain-free tendon. These findings again raised the hypothesis that the neovessels and accompanying nerves were responsible for the pain in the area of a tendinosis. Alfredson et al.⁵ then injected a sclerosing agent (Polidocanol) in the area with neovessels and the short-term results were successful at 6 months post-injection. There were no neovessels outside or inside the tendon, but in the two non-successfully treated patients, the neovessels remained.

This case report was developed upon the fact that non-operative treatment with eccentric exercise has successfully been applied to achilles tendinosis, patellar tendinosis and recently supraspinatus tendinosis.^{1,26} Since these different types of tendinopathies show similarities in clinical behavior and in their histological appearance, eccentric training for a recalcitrant case of lateral epicondylitis was designed to attempt some pain relief for a patient where many other treatment protocols were unsuccessful.

CASE:

A 26 year old male amateur hockey player presented with right lateral elbow pain. When asked to point to the pain, the patient pointed directly to the common extensor tendon on the lateral epicondyle of the humerus. The pain began three years ago while attempting a wrist shot. The patient is a right handed shot, therefore the complaint involves the lower hand on the stick, where majority of wrist power is generated.

The pain gradually progressed and the patient began to notice weakness in the forearm due to the pain. Twenty weeks of physiotherapy was undertaken, which consisted of electric modalities and stretching at a frequency of twice per week, with limited success. The patient then received a cortisone injection, which completely resolved the symptoms. Approximately nine months after the injection, the arm was re-injured via the same mechanism. The patient received another injection, which relieved the symptoms for three months, only to return insidiously. Since that time, the patient had not received any therapy or medication for the complaint. The current complaint is described as a constant dull ache over the lateral epicondyle. The pain becomes sharp with activities involving wrist extension, ulnar deviation and picking up objects with the wrist in a pronated position. No radiating symptoms were reported. The pain was rated as a 7/10 on a visual analog scale. The patient claims the arm is getting weaker and the condition is becoming easier to aggravate.

Upon examination, the screening evaluation of the cervical spine and shoulder were non-contributory. Visual examination was negative for the cardinal signs of inflammation. Passive range of motion of the wrist revealed a reproduction of the patient's complaint with full wrist flexion and pronation when the elbow was extended. Resisted range of motion of the wrist elicited pain with extension and ulnar deviation. However, the most intense pain was reported with resisted radial deviation of the wrist. Pain was reproduced with resisted extension of the middle phalanx, as well as resisted wrist extension with the elbow flexed beyond 90°. Both

Mill's and Cozen's reproduced the pain at the lateral elbow, while Butler's Upper Limb Tension Test for the radial nerve was negative. Static palpation revealed tenderness over the common extensor origin, with the most intense pain located in the proximal attachment of the extensor carpi radialis brevis. Objectively, an average of three trials of grip strength testing using a dynamometer was obtained. The values from the grip strength test were 83 lbs on the left and 61 lbs on the right. The amount of pain during activity was evaluated on a 100-mm visual analog scale. The amount of pain was recorded from 0 -10, where 0 was no pain and 10 was severe pain. The evaluation was done before (week 0) and after treatment (week six). No signs of radioulnar joint dysfunction, radial tunnel syndrome or posterior interosseus nerve syndrome were observed.

The patient was subsequently diagnosed with lateral epicondylitis and a trial of heavy eccentric loading was implemented. The loading regimen consisted of two sessions daily, seven days a week for a total of six weeks. The author chose this time frame, which differs slightly from that proposed by Alfredson, due to the fact that the patient's competitive season was only seven weeks away. During the trial, the patient was allowed to undergo all normal daily activities with no restrictions. The common extensor origin was eccentrically loaded with both the elbow straight and, to maximize tension in extensor carpi radialis brevis, also with the elbow bent. Each of the two exercises included fifteen repetitions completed in three sets. The patient was instructed to continue with the program despite pain. The only indication to

discontinue the protocol was in the event of unbearable pain. When the repetitions were able to be completed with decreasing amounts of pain, weight was increased by two pounds until the upper limit of tolerance was once again achieved. The patient was instructed to rest the forearm on a weight bench with the wrist and hand hanging over one end of the bench. With a 15 lbs dumbbell in the right hand, the patient was instructed to use the uninjured hand to lift the injured wrist into full extension while the forearm remained in full contact with the bench (fig. 1). Once in full extension, the patient slowly lowered the weight over an eight second count until full wrist flexion was achieved (fig. 2). Then the uninjured hand lifted the weight back to the starting position to eliminate any concentric contraction in the injured structures. After three sets, the exercise was then repeated with the elbow flexed to 90 degrees for another three sets (fig. 3).

At the end of the six week session, the patient reported significantly less pain. No aggravation of the complaint was noted during activities of daily living or during competition. The only remaining complaint was minor discomfort over the lateral epicondyle while performing weighted shoulder flys during weekly workout sessions. The patient subjectively claimed to be back to full strength in the forearm. Objectively the visual analog scale pain rating dropped from 7/10 (week 0) to 1/10 (week 6). Average grip strength testing from three trials changed to 85lbs on the left and 79lbs on the right with no pain reproduction reported. Mill's and Cozen's orthopaedic tests were no longer positive. Resisted middle phalanx extension was unremarkable.



Figure 1. Using uninjured hand, lift weight into starting position to bring wrist into full extension.



Figure 2. Over eight seconds, lower weight until wrist is in full flexion.



Figure 3. Repeat above sets with elbow flexed to 90 degrees.

The only remaining signs and symptoms of the original complaint were minor pain with direct manual palpation of the lateral epicondyle. The patient subsequently returned to full competition with no reoccurrence of the condition

reported upon re-examination 16 weeks after initiation of the eccentric loading regimen.

DISCUSSION:

Although the majority of research on tendinosis has focused on the achilles tendon, there are numerous sites, which this degenerative process may effect. One of the most common areas of complaint is the lateral epicondyle. Lateral elbow tendinopathy (LET) is one of the most common lesions of the arm²². It is a degenerative process characterized by the increased presence of fibroblasts, vascular hyperplasia and disorganized collagen in the origin of the extensor carpi radialis brevis (ECRB).¹⁵ It is generally a work-related or sport-related pain disorder usually caused by excessive quick, repetitive eccentric contractions and gripping activities of the wrist, and has been associated with weakness in the external rotators of the shoulder in athletes. The restricted shoulder range of motion is interlinked in a dysfunctional pathokinetic chain predisposing to LET. Excessive wrist movement is substituted in an effort to compensate for an already restricted arc of shoulder motion¹⁵. The dominant arm is commonly affected with a prevalence of 1-3% in the general population.²⁰ Although LET is not influenced by the gender of the patient, it does appear to be of longer duration and severity in females.²⁰

The most tender area on physical examination is usually on the anterior portion of the lateral epicondyle or slightly distal to it. Often tenderness on palpation can be elicited along several areas of the ECRB insertion or on associated muscle/fascia.²⁷ The onset can be either acute or insidious. The

symptoms tend to improve with warm up and worsen with movement requiring wrist extension or gripping.²⁷ Differential diagnoses include, radial tunnel syndrome, posterior interosseus nerve syndrome, radiocapitellar joint degeneration, posterolateral rotary instability or cervical spine pathologies.

Functional evaluation will usually demonstrate pain with resisted wrist extension with the elbow extended and the wrist in pronation. When the ECRB is the main culprit, this pain will be exemplified with the elbow flexed.²⁷ There may be pain and limited wrist flexion when the wrist is pronated with the elbow extended.²⁷ Many authors also describe painful resisted middle finger extension due to stress placed on the extensor digitorum with this maneuver.

Painful tendonopathies affecting the achilles, patellar, common extensor origin, supraspinatus or posterior tibial tendons are common in athletic populations. The etiology and pathogenesis to these conditions is largely unknown, making treatment difficult.¹⁸ By using methods like microdialysis, cDNA arrays, ultrasonography combined with color Doppler and immunohistochemical analyses of biopsies, there is now new and potentially important information about chronic painful tendons.¹⁸

Previous studies have demonstrated obvious structural changes within chronic painful tendons. Many authors have noted structural deformations, such as tendon thickening in the degenerative tendons.⁶ In recent studies, neovascularisation in close relation to the tendon changes have been shown to be associated with pain in patients with

chronic painful achilles tendinosis. The hypothesis that the area with neovascularisation was responsible for the pain was tested by injecting a sclerosing agent (Polidocanol) towards the neovessels outside the tendon under US control.³ In eight out of ten patients, this treatment cured the pain and allowed for full achilles tendon loading activity. A two-year follow up of these patients showed that the same eight patients were satisfied with the results and had no remaining neovascularisation. Interestingly, the tendon thickness had decreased significantly, and the ultrasound appearance was that the tendon structure looked “normalized” compared to baseline findings. The results of these studies suggest that the area of neovascularization, which is in close relation to the area of structural change, may be the source of pain. Previous studies have provided evidence using immunohistochemical techniques that there are nerves traveling with these vessels.⁸ Therefore, it is not known whether the neovessels, associated nerves or a combination of both are the chief pain generators in the chronic painful tendon.

Nevertheless, Alfredson et al.⁴ used the microdialysis method and demonstrated that concentrations of glutamate (a known chemical mediator of pain in the CNS) were significantly higher in tendons with tendinosis compared to those that were pain-free. These findings, together with diagnostic injections, indicate that the area with neovascularisation is of major importance for the pain in tendinosis. Alfredson et al.⁵ also found high levels of intratendinous lactate in painful achilles tendons compared to normal tendons. These high levels may indicate ischemic conditions within the tendon

that triggered the neovascularization to occur in response to the anaerobic condition. These findings help explain the high prevalence of degenerative changes in hypovascular areas and why mechanical overload and hypoxia play such a large role in degenerative tendon disorders. However, whether ischemia precedes the start of tendinosis, or the tendinotic tendon changes itself is causing the ischemia, has not yet been investigated.¹⁸

Many sports medicine and physical therapy journals agree eccentric training has been shown to be a good treatment model for patients with chronic mid-portion achilles tendinopathy.^{1,2,21} However, the background of the positive clinical results remain elusive to those who implement this training tool. Ohberg et al.⁵ demonstrated that passive dorsiflexion of the ankle eliminated blood flow in the vessels in the areas containing neovascularisation. These authors also demonstrated in follow-up studies that in the majority of tendons after eccentric training with a positive result, the neovessels no longer appeared in the tendons upon examination. In the tendons with a poor result, the neovessels remained.⁵

There is no consensus on exactly how eccentric training influences the area of neovascularisation. One hypothesis is that the stoppage in blood flow might directly damage the neovessels and accompanying neural structures.⁵ Another explanation might be that the intratendinous hydrostatic pressure under load might increase the production of antiangiogenic factors.¹⁸ Whatever the mechanism, it is difficult to argue with the results that eccentric training has obtained in recent literature. It has been

shown to have excellent short-to-mid term clinical results in patients with chronic painful tendinosis. These results are associated with a decrease in neovascularisation, decreased tendon thickness and normalization of tendon architecture.

To date the pilot work on sclerosing injections for treatment of chronic tendon pain seem positive. After treatment of 150 achilles tendons and 70 patellar tendons, Alfredson's group have only reported two complications that may have been related to treatment. Both patients suffered a rupture of the achilles tendon, however it was in the area of previous corticosteroid injections. There have been no other reported complications, however, the technique is in its infancy.⁶ Although no negative side effects have been discovered, the long term prognosis of such treatments is largely unknown. Therefore, with the positive results eccentric training has demonstrated, it seems a viable, low risk option that perhaps should be implemented prior to injection of sclerosing agents.

CONCLUSION:

Lateral epicondylitis and tendon disorders in general are a complex condition and it may be years before we

fully understand the etiology of, and best treatment for, these conditions.

However, we cannot ignore the current literature, which provides consistent evidence that there is an absence of an inflammatory component.²⁵ It is time to acknowledge that the traditional model of an inflammatory condition has been disproved. This is more than just accuracy of nomenclature because it directly relates to appropriate management and realistic treatment goals, and prognosis is dependant upon correct comprehension of pathoeitiology.²⁵ Likewise, cortisone injections, and the comfort modalities of traditional physical therapy, do not directly or specifically induce proliferation or improve the quality of the tendon collagen, or bring in new vascularity to promote tissue healing. Therefore, they must be used in the perspective of a broader treatment plan.¹⁷ The findings of this case report suggest conservative treatment consisting of eccentric training may reduce the symptoms involved with lateral epicondylar pain. The eccentric training regimen appeared to considerably reduce the patient's symptoms of epicondylitis, despite the long duration of symptoms and countless failed treatment attempts.

REFERENCES:

1. Alfredson H, Pietila T, Jonsson P, Lorentzon. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *The American Journal of Sports Medicine* 1998; 29(3): 360-366
2. Alfredson H, Lorentzon R. Chronic Achilles tendinosis: recommendations for treatment and prevention. *Sports Med* 2000; 29(2): 135-146
3. Alfredson H. Eccentric training in patients with chronic Achilles tendinosis: normalized tendon structure and decreased thickness at follow up. *British Journal of Sports Medicine* 2004; 38(4)
4. Alfredson H, Ohberg L. Neovascularisation in chronic painful patellar tendinosis – promising results after sclerosing neovessels outside the tendon challenge the need for surgery. *Knee Surg Sports Traumatol Arthrosc* 2005; 13: 74-80
5. Alfredson H, Ohberh L. Sclerosing injections to areas of neovascularisation reduce pain in chronic Achilles tendinopathy: a double-blind randomised controlled trial. *Knee Surg Sports Traumatol Arthrosc* 2005; 13: 338-344
6. Alfredson H. The chronic Achilles and patellar tendon: research on basic biology and treatment. *Scand J Med Sci Sports* 2005; 15: 252-259
7. Bisset L, Paungmali A, Vincenzino B, Bellar E. A systematic review and meta-analysis of clinical trails on physical interventions for lateral epicondylalgia. *British Journal of Sports Medicine* 2005; 39: 411-422
8. Bjur D, Alfredson H, Forsgren S. The innervation pattern of the human Achilles tendon: studies of the normal and tendinosis tendon with markers for general and sensory innervation. *Cell Tissue Res* 2005; 320: 201-206
9. Erak S, Day R, Wang A. The role of supinator in the pathogenesis of chronic lateral elbow pain: a biomechanical study. *The Journal of Hand Surgery* 2004; 29B(5): 461-464
10. Ekstrom R, Holden K. Examination of and intervention for a patient with chronic lateral elbow pain with signs of nerve entrapment. *Physical Therapy* 2002; 82(11): 1079-1088
11. Hamilton B, Purdam C. Patellar tendinosis as an adaptive process: a new hypothesis. *British Journal of Sports Medicine* 2004; 38: 758-761
12. Lewis M, Hay M, Paterson S, Croft P. Local steroid injections for tennis elbow: does the pain get worse before it gets better? *Clinical Journal of Pain* 2005; 21(4): 330-334.
13. Maffulli N, Wong J, Almekinders L. Types and epidemiology of tendinopathy. *Clinics in Sports Medicine* 2003; 22: 675-692
14. Mullett H, Sprauge M, Brown G, Hausman M. Arthroscopic treatment of lateral epicondylitis. *Clinical Orthopaedics and Related Research* 2005; 439: 123-128
15. LaBan M, Iyer R, Tamler M. Occult periarthrosis of the shoulder: a possible progenitor of tennis elbow. *Am. J. Phys. Med. Rehabil* 2005; 84(11): 895-898
16. Nichols A. Complications associated with the use of corticosteroids in the treatment of athletic injuries. *Clinical Journal of Sports Medicine* 2005; 15(5)
17. Nirschl R, Ashman E. Elbow tendinopathy: tennis elbow. *Clinics in Sports Medicine* 2003; 22: 813-836
18. Pufe T, Petersen W, Mentlein R, Tillmann B. The role of vasculature and angiogenesis for the pathogenesis of degenerative tendons disease. *Scand J Med Sci Sports* 2005; 15: 211-222

19. Stasinopoulos D, Johnson M. Cyriax physiotherapy for tennis elbow/lateral epicondylitis. *British Journal of Sports Medicine* 2004; 38: 675-677
20. Stasinopoulos D, Stasinopoulos K, Johnson M. An exercise programme for the management of lateral elbow tendinopathy. *British Journal of Sports Medicine* 2005; 39: 944-947
21. Svernlöv B, Adolfsson L. Non-operative treatment regime including eccentric training for lateral humeral epicondylalgia. *Scand J Med Sci Sports* 2001; 11: 328-334
22. Sevier T, Wilson J. Treating lateral epicondylitis. *Sports Med* 1999; 28(5): 375-380
23. Trinh K, Phillips S, Ho E, Damsma K. Acupuncture for the alleviation of lateral epicondyle pain: a systematic review. *Rheumatology* 2004; 43: 1085-1090
24. Vincenzino B. Lateral epicondylalgia: a musculoskeletal physiotherapy perspective. *Manual Therapy* 2003; 8(2): 66-79
25. Waugh E. Lateral epicondylalgia or epicondylitis: what's in a name? *Journal of Orthopaedic and Sports Physical Therapy* 2005; 35(4): 200-203
26. Young M, Cook J, Purdam C, Kiss Z, Alfredson H. Eccentric decline squat offers superior results at 12 months compared with traditional eccentric protocol for patellar tendinopathy in volleyball players. *British Journal of Sports Medicine* 2005; 39: 102-105
27. Hammer, WI. *Functional soft tissue examination and treatment by manual methods: new perspectives*. 2nd ed. Maryland: Aspen Publishers; 1999