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Etiology and pathophysiology of tendon ruptures in sports

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Of all spontaneous tendon ruptures, complete Achilles tendon tears are most closely associated with sports activities (1–3). Schönbauer (3) reported that 75% of all ruptures of the Achilles tendon are related to sports. In Plecko & Passl (2) the number was 60%. In our material of 430 cases, the number of sports-related Achilles ruptures was very similar (62%), while only 2% of ruptures of other tendons were sports-related ($P<0.001$) (1). Also, the majority of Achilles reruptures occurred in sports. The ruptures occurred most often in soccer (34%), track and field (16%) and basketball (14%). The distribution of Achilles ruptures according to different sports varies considerably from country to country, according to the national sport traditions. For example, in northern and middle Europe, soccer, tennis, track and field, indoor ball games, downhill skiing, and gymnastics are the most common; and in North America, football, basketball, baseball, tennis and downhill skiing dominate the statistics (1, 2, 4). In sports, some Achilles ruptures are not spontaneous or degeneration-induced but may occur as a consequence of the remarkably high forces that are involved in the performance (2). Ruptures in the high jump or triple jump are good examples. In such cases, failure in the neuromuscular protective mechanisms due to fatigue or disturbed co-ordination can frequently be found. The spontaneous complete rupture of the supraspinatus tendon of the rotator cuff does not occur very frequently in sports. Those sports that include high-energy throwing movements, such as American and Finnish baseball, American football, rugby and discus and javelin throwing, may, however, produce this injury. Partial tears and inflammations of the rotator cuff complex are much more frequent in throwing sports. The complete rupture of the proximal long head of the biceps brachii tendon is rare among competitive and recreational athletes. In our material, under 2% of these ruptures were associated with sports activities (5). The rupture (avulsion) of the distal tendon of the biceps muscle is rare. In sports, gymnastics, body building and weight lifting have been said to be able to produce this injury (6). In general, complete ruptures of the quadriceps tendon and the patellar tendon occur most often in older individuals. In our study, the mean age of these patients was 65 years (5). However, these injuries do also occur in younger age groups, especially in athletes. In athletes, the rupture most frequently occurs in high-power sports events, such as high jump, basketball and weight lifting, at the age of 15–30 years. A chronic patellar apicitis (jumper's knee) may predispose rupture of the tendon (7). As is the case with the rotator cuff complex, overuse inflammation and partial tears of the quadriceps and patellar tendons are one of the most characteristic athletic injuries. Complete spontaneous ruptures of other tendons in sports are rare, although the literature does provide case studies from almost every tendon the human body possesses (8–18).

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Basic etiology of a spontaneous tendon rupture

In many patients, the exact etiology and pathogenesis of the spontaneous tendon rupture remain unknown, and this concerns athletes as well. The literature provides a wide spectrum of factors (diseases) that may cause or predispose a tendon to rupture spontaneously. We feel that in most cases tendon degeneration and following rupture is a multi-etiological disorder. In other words, many etiological factors and pathways may lead to tendon degeneration, thereby reducing the tensile strength of the tendon (5). In many ruptured tendons various forms of degenerative tendinopathy can be detected, and the 'syndrome' is usually accompanied with a mixture of predisposing hereditary, structural, professional and lifestyle factors.

Pre-existing distinct diseases within the tendon

Pre-existing distinct intratendinous diseases are quite uncommon reasons for a spontaneous rupture. We examined histological specimens obtained from biopsy of spontaneously ruptured tendons of 891 patients (5). Among them, only 26 patients (3%) had a distinct pre-existing intratendinous disease (and none of them was an athlete), while in the majority of patients the degenerative changes remained without etiological explanation. The 26 patients showed the following findings: an intratendinous foreign body (nine patients), rheumatoid tendinitis (two), tuberculous tendinitis (one), actinomycotic focus (one), granulomatous tendinitis (one), non-specific tendinitis (two), gout affection (one), xanthoma (two), and a tendon tumor (seven). The rupture had occurred through these various lesions, and it seemed obvious that the lesions were the predominant cause of the rupture.

Local factors around the tendon

Some of the spontaneous tendon ruptures in sports may occur as a result of joint or bone abnormalities around the tendon.

The exact mechanisms of tendon ruptures following fractures and joint dislocations are not well understood. McMaster (19) suggested two possible mechanisms. In the first mechanism, the acute tendon tear occurs when the tendon glides over the rough fracture fragment. This mechanism occurs when the rupture of the flexor pollicis longus tendon follows a Bennett-type thumb fracture treated with a Kirschner wire.

In the second mechanism, the tendon rupture occurs as a late phenomenon. A typical example is a late spontaneous rupture of the extensor pollicis longus (EPL) tendon as a long-term consequence of a distal radius fracture. The pathogenesis of this is unknown but two theories have been given: (a) friction-induced mechanical wearing, and (b) vascular impairment and hypoxia followed by tendon degeneration and weakening (20).

Distally pointing osteophytes of the acromioclavicular joint (hooked acromion) may be found in half of the subjects with a rupture of the supraspinatus tendon. Neer (21) pointed out that proliferative spurs and excrecenses on the undersurface of the acromion process cause repeated impingement of the rotator cuff tendons, and, in this way, may cause the tear.

In 10–20% of patients with a rupture of the quadriceps or patellar tendon, a local predisposing factor for the rupture can be observed. The literature provides such factors as patellar hypermobility, subluxation, high or low riding patella, Osgood–Schlatter or Sinding–Larsen–Johansson disease, and elongation of the lower pole of the patella (22, 23).

The local factors associated with Achillies tendon ruptures are static as well as dynamic by nature. The Achilles tendon is permanently stretched in ordinary activities, not to mention the loadings occurring in sports. The biomechanical fault positions of the ankle and foot, such as forefoot vagus or varus, or calcaneal valgus or varus, causing horizontal, axial and rotational alterations in the course of running of the Achilles tendon fibers are important predisposing factors to the tendon rupture (24). The local peritendinous pathologies such as Haglund's syndrome, retrocalcaneal bursitis, calcaneal bone spurs, tumors around the tendon, and chronic skin ulcerations with adhesions in the lower limb may also be predisposing factors (3). In addition, some anatomical variations in the attachment of the gastrocnemius muscles to the Achilles tendon may have role in the pathogenesis of the rupture.

The majority of lesions in the proximal long head of the biceps brachii (BB) tendon are secondary to other pathological conditions of the shoulder (25). These conditions can be divided into those occurring in the intertubercular sulcus of the humerus and those occurring elsewhere in the shoulder joint. Abnormal bony anatomy (spurs) of the bicipital groove, impingement syndrome of the supraspinatus tendon with or without rotator cuff pathology, and shoulder instability are the most frequent local predisposing alterations related to the rupture of the long head of the biceps brachii tendon.

Generalized concurrent diseases without specific tendon pathology

Older literature frequently mentions that a tendon rupture may occur with chronic systemic diseases such as syphilis, tuberculosis, obesity, systemic malignancies, gonorrhea, trichinosis and typhoid fever

(26). More recently, ankylosing spondylitis, rheumatoid arthritis, acute rheumatic fever, gout, systemic lupus erythematosus, chronic uremia (hemodialysis) and hyperparathyreoidism have been mentioned (7). These diseases are, however, seldom found in athletes.

Drugs and spontaneous tendon ruptures

The issue of corticosteroids and risk of tendon rupture is very controversial in sports medicine literature, and in this context it must be emphasized that current literature provides no convincing evidence either for or against corticosteroids in increasing risk of tendon rupture (27). However, recent literature provides somewhat stronger evidence of the connection of some other drugs and tendon rupture. Anabolic hormone abuse among athletes clearly increases the risk for tendon rupture (28). Kucera & Slezak (29) reported 23 cases of Achilles tears following the use of weight-reducing drugs. More recently, Bottomley & Cunliffe (30) reported three cases of Achilles tendinitis following oral administration of isotretinoin for acne vulgaris. The drug caused other side-effects as well, including myalgia and arthralgia.

Ribard et al. (31) described seven cases of Achilles tendinitis following fluoroquinolone antibiotic treatment. In three of them, the course was complicated by rupture of the tendon. The authors discussed that the quinolones may well have a toxic effect on tendon tissue. More recently, new cases have been reported (32). Finally, tendinitis, and in rare cases tendon rupture, as a result of the antibiotic pefloxacin is well recognized (33).

Genetic and constitutional factors

Genetic factors

The role of the genetic background of the patient in tendon diseases and ruptures is mostly unknown. However, recently a relationship between the ABO blood groups and spontaneous tendon ruptures, as well as the association between the HLA blood groups and tendon diseases, has been recognized. Bauer (34) described bilateral quadriceps rupture in identical or monozygotic twins and suspected an undetermined genetic defect as the predisposing factor. Singer & Jones (35) noted that Achilles tendon rupture is uncommon in children and can be only found in children whose parents have experienced tendon rupture.

The subtle gene defects in collagen metabolism may explain some of the spontaneous tendon ruptures in seemingly healthy adults, such as athletes. The recent advancements in gene technology and the huge world-wide efforts to map the human genome may give interesting information about tendon diseases and ruptures in the near future.

Constitutional factors

Literature provides some evidence that large body weight and size may predispose to tendon injuries (36). However, to our knowledge no study has been published trying to clarify specifically the role of body constitution in tendon ruptures.

Leg length inequality over 1 cm can be detected in 15% of patients with Achilles tendon overuse injury (36). Frequently, a combination of muscle weakness, ligamentous laxity (sometimes joint tightness) and overflexibility can be found in subjects with tendon injuries (36). Bone, tendon and muscle anomalies may also be important constitutional factors that predispose to tendon ruptures. According to Smart et al. (24), pes cavus or planus, and increased passive talocrural or subtalar joint mobility, also predispose the Achilles tendon to injuries. Constitutional laxity of the joints or so-called hyperlaxity syndrome may be a key predisposing factor for patellar tendinitis and apicitis. Knowledge about connections between ligamentous laxity and ruptures in other tendons is sparse, although inherited shoulder laxity has been connected with rotator cuff tears and biceps brachii problems (25).

Blood groups and HBA groups

Blood groups

In recent years, reports of the association between the blood groups and diseases have been published with increasing frequency. We retrospectively analysed the frequencies of ABO and Rh blood groups in 832 patients with a tendon rupture (37). We have continued our study and today have 1138 primary ruptures, 74 multiple ruptures and 53 reruptures of tendons in our file. The ABO and Rh blood groups of the patients are determined with conventional methods and the control data from 1.2 million Hungarians are provided by Rex-Kiss and Szabo from the Hungarian Laboratory Center.

In this material of 1275 tendon ruptures, the overall number of patients with a blood group O is 687 (54%), group A 337 (26%), group B 172 (13%), and group AB 79 (7%). In the control population, blood group O is found in 31% of the subjects ($P<0.005$), group A 42% ($P<0.005$) group B 18% (NS), and group AB 9% (NS). Within the group of multiple or reruptures, the relative number of patients with the blood group O is even higher, 69% and 70%. The Rh groups are not associated with tendon ruptures.

Our original findings have been confirmed in Finland by Kujala et al. (38). They observed, too, that there is an association between the ABO blood groups and injuries of the Achilles tendon.

The exact reason for the association between the blood group O and tendon rupture is unknown. The

ABO blood group gene is located on chromosome 9 but none of the collagen genes is known to locate in this chromosome (39). Nevertheless, since the association is so clear further research is definitely needed to determine the pathogenetic link between blood group O and tendon rupture.

HLA group

Hydroxyapatite periarthritis and tendinitis as well as calcifying tendinopathy is associated with A2 and BW 35 HLA antigens. The frequency of A2 patients with one tendon calcification is 50%, while in patients with two or more calcifications the number is 70%. The relative risk for the disease in individuals with A2 BW 35 is 2.42 (40).

Patients with juvenile rheumatoid arthritis have a high frequency of B7 (66%) provided that they also have tenosynovitis, while in the whole patient population the B7 frequency is only 19% (41). Many other diseases with tendon affections also show clear associations with HLA markers. Polymorphism in the HLA markers is thus associated with a wide variety of diseases, both with immune and non-immune etiology.

Future studies will show whether spontaneous tendon ruptures are in association with specific HLA antigens.

Vascular impairment and hypoxia of tendons

Two major theories about the pathogenesis of spontaneous tendon ruptures have been given in the literature: a mechanical theory (42) and a vascular theory (20, 43), and they both may occur in athletes. The former claims that tendon injury is the primary occurrence that may lead to permanent tendon weakening and incomplete tendon regeneration, possibly by vascular damage and decreased blood flow. Advocates of the vascular theory claim, in turn, that the impaired vascular supply and blood flow to the tendon tissue via aging, disease process or trauma is the primary factor that may result in a process of tendon degeneration so that finally a slight-to-moderate strain ruptures the tendon.

Both of the above-mentioned theories are probably true. The anatomy of the tendon, its vascularization and its surrounding structures may predispose a tendon to mechanical wearing, microtrauma or macrotrauma followed by incomplete regeneration. Traumatic bleeding into the synovial sheath of a tendon without rupture of the fibrous sheath of the same may increase the intrasynovial pressure and obliterate the vessels of the sheath itself, the vincula and intratendinous channels, and thus begin a vicious circle of decreased blood flow, tissue hypoxia, and fiber degeneration. If this occurs in areas of poor basic vascularity, as is the case in some areas of supraspinatus, extensor pollicis longus, posterior tibial and Achilles tendons, the effect of pressure increase may well be dramatic. On the other hand, it has been shown that without any trauma the blood flow of the tendons decreases with age, in human Achilles tendons after the third decade of life (43), and this may begin the process of tendon degeneration. A sedentary adulthood lifestyle has, in turn, been proposed as the main reason for poor circulation to the tendon (1).

Thus, it seems likely that many etiological factors and pathways may lead to tendon degeneration, thereby reducing the tensile strength of the tendon. The available evidence suggests that vascular changes and decreased arterial blood flow with resulting local hypoxia and impaired metabolic activity and nutrition may be the key factors (5, 44). In addition, exercise-induced hyperthermia of the tendon has been suggested to be detrimental to tendon cells (45).

In our study (5) of 290 control tendons that were completely normal structurally, pathological vascular changes were seen in only 5%, while two-thirds of the 149 control tendons that had evidence of tendon degeneration had vascular changes similar to those seen in the 891 ruptured tendons. Pathological vascular changes of tendons are thus very closely related to tendon fiber degeneration in human population.

The histopathology of the vascular changes of the ruptured tendons is described by Józsa and Kannus in the following article of this issue.

Tendon inflammation

Acute and chronic tendinous, peritendinous and insertional inflammations of tendons as a result of overuse (overuse injuries) are a major problem in sports traumatology. For a long time there has been a suspicion that chronic inflammatory processes of tendons may cause tendon degeneration, the degeneration resulting from an inflammation-induced decrease in blood flow and following ischemia, direct toxic effects of the inflammatory catabolites on tenoblasts, or both. However, direct evidence is still lacking.

On the other hand, Arner et al. (46) found no inflammatory cellular infiltrations in their patients who had a rupture of the Achilles tendons, providing evidence that clear tendinous inflammation was not present at the time of the rupture. Our findings were similar; there were no signs of infiltration of inflammatory cells in the ruptured tendons that we studied (5).

It seems logical that rupture of a tendon usually occurs in a very late stage of the degenerative process. At that time, the tendon is weak enough to be prone to rupture, but unfortunately the pain and discomfort, which usually are prominent in the earlier

(inflammatory) phase of degeneration, have subsided, so that the patient may engage in more strenous physical activities despite the attendant risk of rupture (1).

As noted above, there is popular theory that degeneration of tendon goes through acute, recurrent, subacute, subchronic and chronic phases of tendon inflammation before actual degeneration develops (47), and it is true that leukocytes have collagenase enzymes capable of collagenolysis, and other catabolic enzymes for digestion of the glycosaminoglycans and other extracellular matrix components of a tendon, but it is not clear why only one-third of our patients had had symptoms, such as tenderness, stiffness, pain or discomfort, in the region of the tendon that subsequently ruptured, while two-thirds of the patients (almost 600 individuals) had been completely asymptomatic and could not recall any warning signal before the rupture (5).

In addition, it may be possible that tendon tissue degeneration can create or predispose the tissue to inflammatory reaction so that the relationship between these two factors is not from inflammation to tendon degeneration but vice versa. Also, an interactive vicious circle between inflammation and degeneration is one explanation, or alternatively these two factors have an entirely non-interactive or independent co-existence during tendinosis development. Finally, the previously described vascular impairment and tissue hypoxia are a third contributing factor, and it is likely that this parameter interacts closely with both inflammatory reaction and tendon tissue degeneration.

Thus, the true cause–consequence relationships between inflammation, hypoxia and tendon tissue degeneration are largely unknown but it seems likely that many etiological factors and pathways, including inflammation and hypoxia, may lead to fiber degeneration, thereby reducing the tensile strength of the tendon.

Lifestyle factors

The general lifestyle of the population may have become more sedentary, which, it has been proposed, has resulted in decreased circulation and nutrition of the tendons, followed by tendon degeneration and increased predisposition to traumatic tendon lesions during acute (sports) exertion (1). The occupational and social background of patients with spontaneous tendon ruptures has changed during recent decades from manual laborers and farmers to white collar workers, professionals and pensioners, and this may reflect the decreasing physical demands on the tendons of people of our time. If so, along with other musculoskeletal tissues, human tendons have adapted to lowered physical demands by structural weakening, and so are at increased risk of injury on acute exertion. Today, such an exertion is likely to occur in leisure time sporting activities.

Conclusions

In athletes as well as non-athletes, tendon degeneration and a following rupture seems to be a multi-etiological disorder. In other words, many etiological factors and pathways may lead to tendon degeneration, thereby reducing the tensile strength of the tendon. The ruptured tendons show various forms of degenerative tendinopathy, and the 'syndrome' is usually accompanied with a mixture of predisposing hereditary, structural, professional and lifestyle factors.

References

1. Józsa L, Kvist M, Bálint BJ, Réffy A, Järvinen M, Lehto M, Barzò M. The role of recreational sports activity in Achilles tendon rupture. A clinical, pathoanatomical and sociological study of 292 cases. Am J Sports Med 1989: 47: 338–43.
2. Plecko M, Passl R. Ruptures of the Achilles tendon: causes and treatment. J Finn Orthop Traumat 1991: 14: 201–4.
3. Schönbauer HR. Erkrankungen der Achillessehne. Wien Klin Wschr 1986: 98 (Suppl 168): 1–47
4. Kellam JF, Hunter GA, McElwain JP. Review of the operative treatment of Achilles tendon rupture. Clin Orthop 1985: 201: 80–3.
5. Kannus P, Józsa L. Histopathological changes preceding spontaneous rupture of tendon. A controlled study of 891 patients. J Bone Joint Surg (Am) 1991: 73: 1507–25.
6. D'Alessandro DF, Shields CL, Tibone JE, Chandler RW. Repair of distal biceps tendon ruptures in athletes. Am J Sports Med 1993: 21: 114–19.
7. Dejong CHC, Van de Luytgaarden WGAC, Vroemen JPAM. Bilateral simultaneous rupture of the patellar tendon. Arch Orthop Trauma Surg 1991: 110: 222–26.
8. Alho A. Ruptured pectoralis major tendon. Acta Orthop Scand 1994: 65: 652–3.
9. Bach BR Jr, Warren RF, Wickiewicz TL. Triceps rupture, a case report and literature review. Am J Sports Med 1987: 15: 285–9.
10. Coghlan BA, Clarke NMP. Traumatic rupture of the flexor hallucis longus tendon in a marathon runner. Am J Sports Med 1993: 21: 617–8.
11. Evans JD. Subcutaneous rupture of the tendon of peroneus longus. J Bone Joint Surg (Am) 1966: 48: 507–9.
12. Jensen IH, Kramhoft M. Distal rupture of the biceps femoris muscle. Scand J Med Sci Sports 1994: 4: 259–60.
13. Orava S, Kujela U. Rupture of the ischial origin of the hamstring muscles. Am J Sports Med 1995: 23: 702–5.
14. Postacchini F. Subcutaneous ruptures. In: Perucia L, Postacchini F, Ippolito E, ed. The tendons: biology-pathology-clinical aspects. Milano: Editrice Kurtis 1986: 237–362.
15. Sim FH, Deweerd JH. Rupture of the extensor hallucis longus tendon while skiing. Minn Med 1977: 60: 789.
16. Stannard JP, Bucknell AL. Rupture of the triceps tendon associated with steroid injections. Am J Sports Med 1993: 21: 482–5.
17. Wenger DR. Avulsion of the profundus tendon insertion in football players. Arch Surg 1973: 106: 145–8.
18. Woods L, Leach RE. Posterior tibial tendon rupture in athletic population. Am J Sports Med 1991: 19: 495–8.

19. McMaster PE. Late ruptures of extensor and flexor pollicis longus tendon following Colle's fractures. J Bone Joint Surg 1932: 14: 93–7.
20. Engkvist O, Lundborg G. Rupture of the extensor pollicis longus tendon after fracture of the lower end of the radius. A clinical and microangiographic study. Hand 1979: 11: 76–86.
21. Neer CS. Anterior acromioplasty for the chronic impingement syndrome in the shoulder. J Bone Joint Surg (Am) 1972: 54: 41–5.
22. Kelly DW, Carter VS, Jobe FV, Kerlan RK. Patellar and quadriceps tendon ruptures – Jumper's knee. Am J Sports Med 1984: 12: 375–80.
23. Vainionpää S, Böstman O, Pätiälä H, Rokkanen P. Megapatella following a rupture of patellar tendon. A case report. Am J Sports Med 1985: 13: 204–5.
24. Smart GW, Taunton JE, Clement OB. Achilles tendon disorders in runners – a review. Med Sci Sports Exerc 1980: 12: 231–43.
25. Dines D, Warren RI, Inglis AE. Surgical treatment of lesions of the long head of the biceps. Clin Orthop 1982: 164: 165–70.
26. Detzel H. Beitrag zur Ätiologie der Achillessehnenruptur. Arch Orthop Unfallchir 1950: 44: 306–12.
27. Read MT, Motto SG. Tendo Achilles pain: steroids and outcome. Br J Sports Med 1992: 26: 16–21.
28. Laseter JT, Russell JA. Anabolic steroid-induced tendon pathology: a review of the literature. Med Sci Sports Exerc 1991: 23: 1–3.
29. Kucera M, Slezak Z. Die Verletzungen der Achillessehnen bei den pharmacologisch behandelten Sportlern. Sportärztliches Achillessehnen – Symposium, Prag 1966.
30. Bottomley WW, Cunliffe WJ. Acute achilles tendonitis following oral isotretinoin therapy for acne vulganis. Clin Exp Dermatol 1992: 17: 250–1.
31. Ribard P, Audisio F, Kahn MF, De-Bandt M, Jorgensen C, Hayem G, Meyer O, Palazzo E. Seven achilles tendinitis including 3 complicated by rupture during fluoroquinolone therapy. J Rheumatol 1992: 19: 1479–81.
32. Huston KA. Achilles tendinitis and tendon rupture due to fluoroquinolone antibiotics. N Engl J Med 1994: 331: 748.
33. Perrot S, Kaplan G, Ziza JM. Three cases of achilles tendinitis caused by pefloxacin, 2 of them with tendon rupture. Rev Rhum Mal Osteoartic 1992: 59: 162.
34. Bauer U. Spontane subcutane Rupturen der Quadricepssehne insbesondere bei eineiigen Zwillingen. Unfallheilk 1980: 83: 520–4.
35. Singer KM, Jones DC. Soft tissue conditions of the ankle and foot. In: Nicholas JA, Hershmann EB, ed. The lower extremity and spine in sports medicine. St Louis: Mosby 1986: 148.
36. Kvist M. Achilles tendon injuries in athletes. Sports Med 1994: 18: 173–201.
37. Jòzsa L, Bàlint BJ, Kannus P, Réffy A, Barzò M. Distribution of blood groups in patients with tendon rupture. An analysis of 832 cases. J Bone Joint Surg (Br) 1989: 71: 272–4.
38. Kujala UM, Järvinen M, Natri A, Lehto M, Nelimarkka O, Hurme M, Virta L, Finne J. ABO blood groups and musculoskeletal injuries. Injury 1992: 23: 131–3.
39. Sandberg M. Expression of collagen, TFG-β, and c-fos genes in developing human skeletal tissues. University of Turka, Finland: Academic Thesis, 1990.
40. Amor B, Cherot A, Delbarre F, Roldan AN, Hors J. Hydroxyapatite rheumatism and HLA markers. J Rheumatol 1977: 4: 101–4.
41. Gibson D, Schur PH, Carpenter CB, Stillman IS. Distribution of HLA antigens in patients with juvenile rheumatoid arthritis (JRA). Arthritis Rheum 1975: 18: 285–8.
42. Knörzer E, Folkhard W, Greeken W, Boschert C, Koch MHJ, Hilbert B, Krahl H, Mosler E, Nemetschek-Ganzler H, Nemetschek T. New aspects of the etiology of tendon rupture. An analysis of time-resolved dynamic-mechanical measurements using synchroton radiation. Arch Orthop Trauma Surg 1986: 105: 113–20.
43. Håstad K, Larsson LG, Lindholm A. Clearence of radiosodium after local deposit in the achilles tendon. Acta Chir Scand 1958/59: 116: 251–5.
44. Archambault JM, Wiley JP, Bray RC. Exercise loading of tendons and development of overuse injuries. Sports Med 1995: 20: 77–89.
45. Wilson AM, Goodship AE. Exercise-induced hyperthermia as a possible mechanism for tendon degeneration. J Biomech 1994: 27: 899–905.
46. Arner O, Lindholm A, Orell SR. Histologic changes in subcutaneous rupture of the achilles tendon. A study of 74 cases. Acta Chir Scand 1958/59: 116: 484–90.
47. Hess GP, Cappiello WC, Poole RM, Hunter SC. Prevention and treatment of overuse tendon injuries. Sports Med 1989: 8: 371–84.