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ARTICLE

C. Edwin Martin
Mark E. Schweitzer

MR imaging of epicondylitis

Abstract *Objective.* To systematically evaluate the MR findings in patients with epicondylitis compared with asymptomatic volunteers.
Design and patients. We imaged 43 elbows: 24 with epicondylitis (22 lateral, 2 medial) diagnosed by clinical examination, and 19 in 16 normal volunteers. MRI was performed at 1.5 T using axial T1-weighted, axial fat-saturated FSE, and coronal or sagittal Fast STIR sequences. Two independent observers evaluated the images for intratendon signal, tendon thickening, periosteal reaction, fluid in the radial head bursa, and anconeus edema.
Results. All 24 patients with epicondylitis had increased signal on fat-saturated FSE and Fast STIR images. Twenty-two of these patients had increased intratendon T1 signal, and 19 had tendon thickening. No patient demonstrated fluid in the radial head bursa or periosteal reaction. Only two patients had subtle anconeus edema, while three patients unexpectedly had increased T2 signal within the involved epicondyle. One asymptomatic volunteer (high-performance athlete) had increased T1 and T2 signal with tendon thickening. An additional two asymptomatic volunteers had increased T1 signal only.
Conclusion. MRI of epicondylitis demonstrates tendon thickening with increased T1 and T2 signal, but these findings may be seen in a small minority of asymptomatic individuals. Anconeus edema, previously demonstrated on MRI in epicondylitis, was only rarely found, and distension of the radial head bursa, surgically described, was not seen. Increased marrow T2 signal within the involved epicondyle is occasionally seen.

Key words Elbow injuries · Epicondylitis · Elbow, MRI · Tendons, MRI

C.E. Martin, M.D.
M.E. Schweitzer, M.D. (✉)
Thomas Jefferson University Hospital,
132 S. 10th Street, 1096 Main Building,
Philadelphia, PA 19107, USA

Introduction

Pain at the origin of the wrist flexor or extensor tendon complexes from the medial or lateral epicondyle of the distal humerus is termed epicondylitis, and is the most common cause of discomfort and disability of the elbow [1]. There have been several anecdotal descriptions of the MRI findings in epicondylitis which include increased intratendon or peritendon T2 and/or T1 signal intensity, tendon thickening, small joint effusions, and periostitis [2–6]. In two scientific investigations of MRI in chronic lateral epicondylitis, one described anconeus edema [7], and one correlated intratendon and peritendon signal hyperintensity with both severe degeneration and tears at surgery [8]. Our goal was to systematically compare the MRI findings in patients with epicondylitis with those in asymptomatic volunteers.

Materials and methods

The study population consisted of 24 elbows in 24 consecutive epicondylitis patients (ages 29–62 years, mean 38 years), and 19 el-

bows in 16 asymptomatic volunteers (ages 22–46 years, mean 31 years), giving a total of 43 elbows in 40 subjects during a 19-month time period. The diagnosis of epicondylitis was made by usual clinical criteria on history and physical examination by an orthopedic surgeon within 2 weeks of the MRI examination [1, 9–11].

Imaging was performed on a 1.5-T magnet (GE Signa, Milwaukee, Wis.) using a shoulder coil (Medrad, Pittsburgh, Pa.). Subjects were generally placed prone in the swimmer's position with the elbow extended. Axial T1-weighted conventional spin echo, axial fast spin echo (FSE) with fat saturation, and either coronal or sagittal fast short tau inversion recovery (Fast STIR) sequences were performed in all subjects. Although sequences evolved somewhat during the time period of the study, typical parameters included repetition time (TR) 400–550 ms and echo time (TE) 14 ms for axial T1-weighted images; TR 3000–4000 ms, effective echo time (TEf) 90–95 ms, and echo train length (ETL) 8 for fat-saturated FSE; and TR 2600–5000 ms, TEf 40–48 ms, time to inversion (TI) 150 ms, and ETL 8 for Fast STIR. Other parameters were 2 acquisitions, 4 mm slice thickness, 12–15 cm field of view, and 256×256 matrix.

Two masked independent observers (M.E.S., C.E.M.) evaluated all MRI examinations for intratendon signal intensity, tendon thickening, periosteal reaction, fluid in the radial head bursa, and anconeus edema. Normal tendons were expected to show uniformly low signal intensity on both T1- and T2-weighted sequences [5]. Increased intratendon T1 or T2 signal intensity was reported, but no attempt was made to grade extent or severity on initial review. A tendon was considered thickened when focally enlarged. While mature periosteal reaction may have a low signal intensity on T1- and T2-weighted images, our experience has been that acute periosteal reaction is visualized as circumferential or semicircumferential high T2 signal intensity immediately adjacent to cortical bone – a finding anecdotally reported in lateral epicondylitis [4]. The criterion used for fluid distension of the surgically described adventitial bursa between the radial head and wrist extensor tendon complex [12–15] was well-defined high signal between the radial head and the wrist extensor tendon complex on fat-saturated FSE or Fast STIR images. Anconeus edema was defined as increased signal intensity of the anconeus muscle compared with adjacent muscles on fat-saturated FSE or Fast STIR images [7].

Kappa values were calculated as a measure of interobserver variability for the above findings. The bias-adjusted prevalence-adjusted kappa value was also calculated when observers disagreed over an infrequent positive finding, since kappa can be underestimated. When discrepancies were present between the two observers, a conclusion was reached by mutual agreement after conference and reported results reflect consensus values.

Lastly, the images considered to have increased intratendon T2 signal intensity were retrospectively reviewed to grade the intratendon T2 signal intensity.

Data on age and duration of symptoms were obtained from the medical records in clinical cases. Age and any past or present history of elbow abnormality were obtained through questioning of the normal volunteers at the time of the MRI examination.

Results

Twenty-two patients had a clinical diagnosis of lateral epicondylitis, and two had medial epicondylitis. Review of patient records showed that duration of symptoms in 21 of the 24 epicondylitis patients was from 1 to 24 months with a mean of 5.5 months. Data on duration of symptoms were not available for three patients. All normal volunteers were asymptomatic at the time of imaging, and none had a past history of elbow disorders.

All 24 symptomatic patients had increased T2 signal within the tendon complex at or near its origin from the epicondyle on fat saturated FSE and Fast STIR images (Figs. 1, 2). Of these patients, 22 had increased intratendon signal on T1-weighted images, and 19 also had tendon thickening (Figs. 1, 2). None was felt to have periostitis or visible fluid within a radial head bursa. Two were felt to have subtle anconeus edema by MRI. In addition, three patients unexpectedly had increased T2 signal within the involved epicondyle.

One asymptomatic volunteer, a professional athlete, had increased T1 and T2 signal with tendon thickening at the origin of his wrist extensor tendon complex, similar to that seen in patients with epicondylitis (Fig. 1A;B). The abnormal T2 signal intensity was central, involved more than 50% of the thickness of the tendon, and was less intense than fluid in character. An additional two asymptomatic volunteers had increased intratendon T1 signal intensity at the origin of their wrist extensor or flexor tendon complexes, but normal T2 signal intensity and no tendon thickening.

For increased intratendon T2 signal intensity, periosteal reaction, and fluid distension of the radial head bursa, observers were in 100% agreement (kappa value 1.000). The kappa value was 0.905 for increased intratendon T1 signal intensity, and 0.860 for tendon thickening. For anconeus edema the kappa value was 0.655, and the bias-adjusted prevalence-adjusted kappa value was 0.953.

Retrospective review of the character of abnormal intratendon T2 signal intensity revealed that 13 (54%) of the elbows with epicondylitis had areas of intense T2 signal intensity similar to fluid (Figs. 1D, 2B,D), and in seven this involved most or all of the thickness of the tendon (Fig. 2). The remainder demonstrated increased T2 signal intensity compared with normal tendon, but less intense than fluid.

Discussion

Lateral epicondylitis, commonly called tennis elbow, involves pain at the origin of the wrist extensor tendon complex from the lateral epicondyle [1, 9–11]. At the common extensor origin, a definitive carpi radialis brevis tendon originates from the lateral epicondyle with aponeurotic fibers from the adjacent extensor digitorum communis [9]. Pathologic change in lateral epicondylitis is almost always seen involving the extensor carpi radialis brevis tendon, with involvement of extensor digitorum communis in approximately one-third of these patients [16]. The less frequent medial epicondylitis involves pain and pathologic alteration at the origin of the forearm flexors and pronator teres [10, 11].

While the name “epicondylitis” suggests an inflammatory process, some investigators have suggested that active inflammation does not play a significant role in

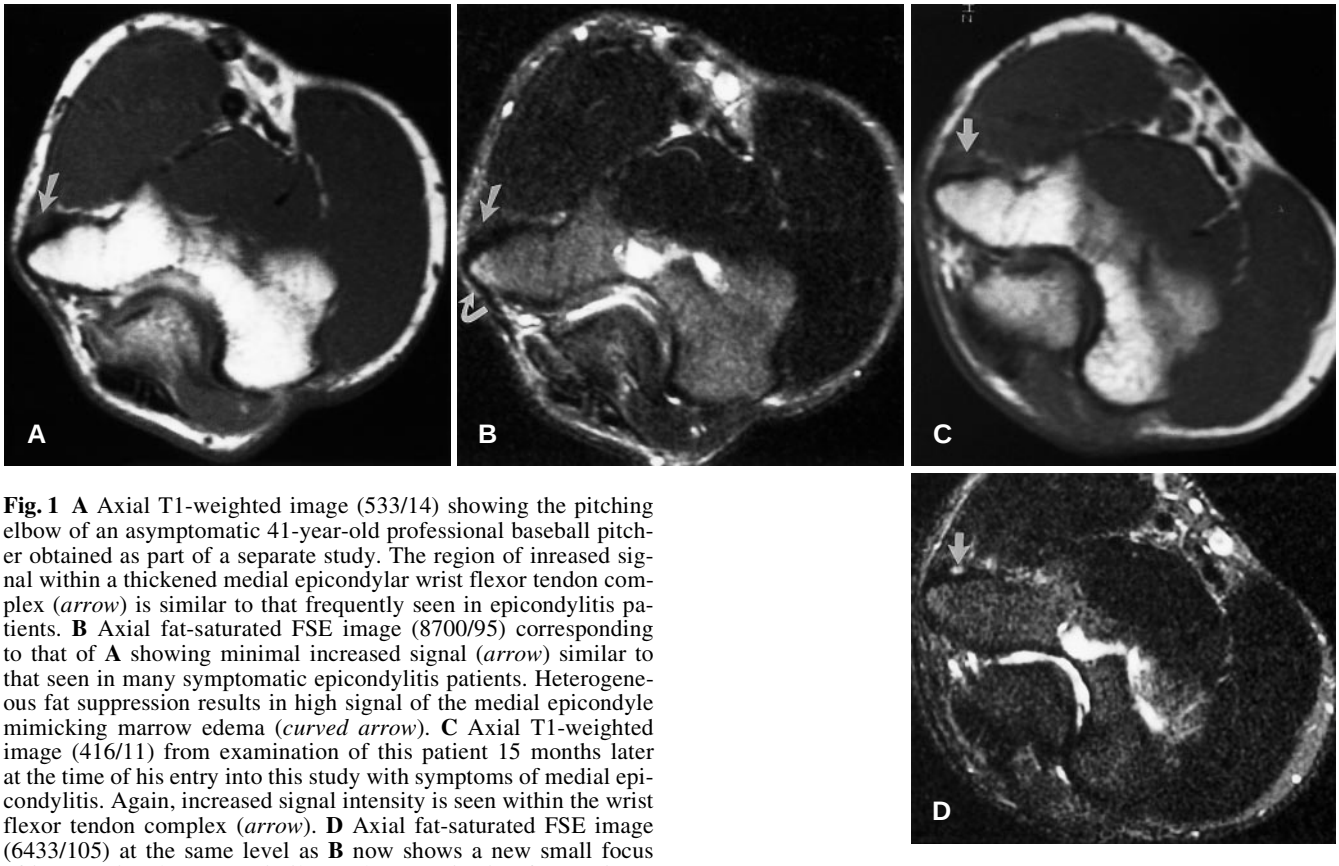

Fig. 1 **A** Axial T1-weighted image (533/14) showing the pitching elbow of an asymptomatic 41-year-old professional baseball pitcher obtained as part of a separate study. The region of inreased signal within a thickened medial epicondylar wrist flexor tendon complex (*arrow*) is similar to that frequently seen in epicondylitis patients. **B** Axial fat-saturated FSE image (8700/95) corresponding to that of **A** showing minimal increased signal (*arrow*) similar to that seen in many symptomatic epicondylitis patients. Heterogeneous fat suppression results in high signal of the medial epicondyle mimicking marrow edema (*curved arrow*). **C** Axial T1-weighted image (416/11) from examination of this patient 15 months later at the time of his entry into this study with symptoms of medial epicondylitis. Again, increased signal intensity is seen within the wrist flexor tendon complex (*arrow*). **D** Axial fat-saturated FSE image (6433/105) at the same level as **B** now shows a new small focus of intense signal (*arrow*) similar to that seen in six of our epicondylitis patients

the chronic condition since polymorphonuclear leukocytes and lymphocytes are uncommon [8, 10, 16, 17]. Histopathologic samples often reveal angiofibroblastic hyperplasia, a proliferation of fibroblasts, and vascular granulation tissue [8, 16–18], which represents a healing response [19] to chronic microtraumatic [11, 20] and possibly macrotraumatic injury [1]. Degenerative histopathologic changes [8, 16, 17, 20–22] and tendon tears [8, 17, 20] have also been found. Note that histopathology specimens in acute epicondylitis are generally not available, since surgery is rarely performed early in the condition.

While the mechanism for the sensation of pain is uncertain, the etiology of epicondylitis is thought to be repetitive overuse of the wrist and elbow in a way that stresses the wrist flexor and extensor origins secondary to athletic participation or occupation [1, 9–11]. Overuse causes degenerative change or tendinosis [23–28], which then weakens tendons, predisposing to macro- and microtraumatic injury [29].

The diagnosis of epicondylitis is made by a typical history and physical examination [1, 9–11]. While the clinical diagnosis may often be straightforward, there can be difficulty in distinguishing radial or posterior interosseous nerve entrapment syndrome from lateral epicondylitis [9], and medial collateral ligamentous strain and ulnar neuritis from medial epicondylitis [11].

In our study, MR images obtained were evaluated for concominant conditions and underlying osseous changes. One could argue that since the clinical diagnosis of epicondylitis is usually accurate in the hands of an experienced orthopedist, MRI plays no role in the diagnosis and should only be used for surgical planning [8]. There may be a trend in the current practice environment, however, for some primary care physicians to order an MRI examination prior to referral to an orthopedist [30], and the radiologist may be first to suggest the diagnosis of epicondylitis.

The treatment is initially conservative with avoidance of activities that incite the pain, cryotherapy, anti-inflammatory medication, and splinting if necessary for symptomatic relief [10]. Surgery is considered if symptoms persist for a year or more despite a quality rehabilitation program [10].

In our 24 patients with epicondylitis, MRI consistently demonstrated increased intratendon signal intensity on fat-saturated FSE and Fast STIR images, suggesting this is associated with epicondylitis. Almost all of these patients also had increased intratendon T1 signal intensity and most had tendon thickening. These MRI findings

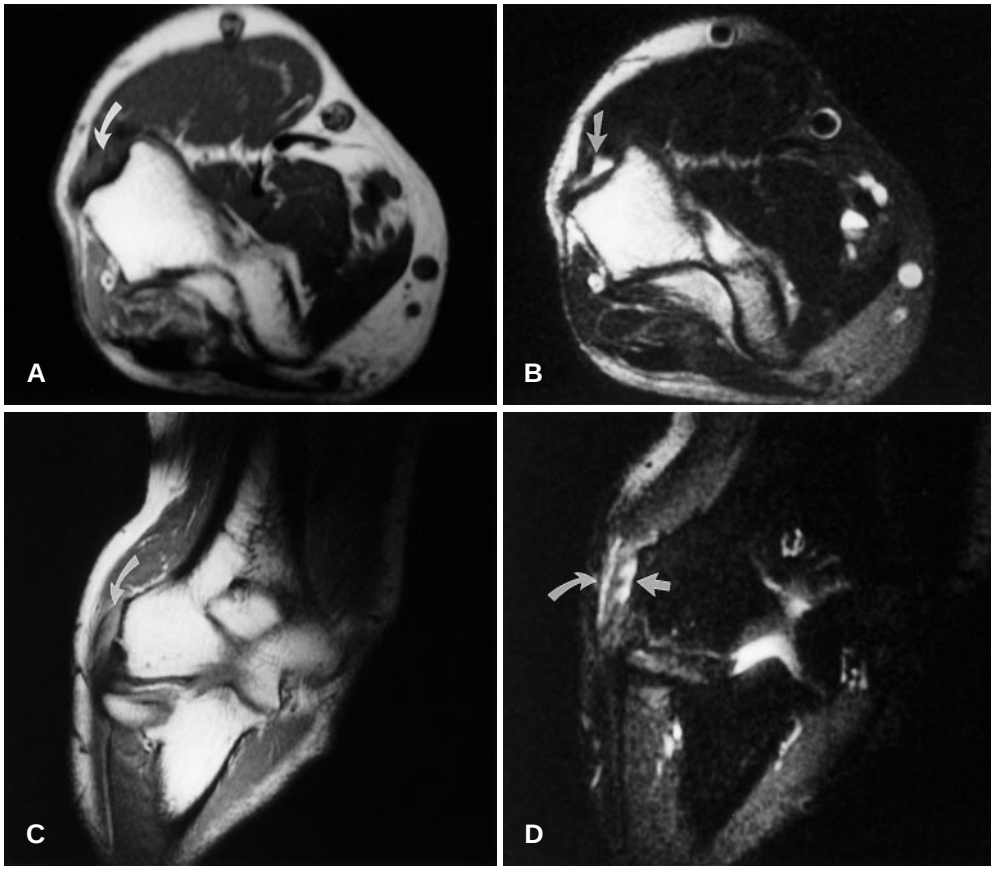

Fig. 2 **A** Axial T1-weighted image (433/14) in a 43-year-old patient with 2 months of discomfort clinically diagnosed as lateral epicondylitis. There is intermediate signal intensity of the wrist extensor tendon complex (*arrow*). **B** Axial fat-saturated FSE image (3717/90) showing corresponding intense increased signal intensity involving the entire tendon (*arrow*) similar to that seen in seven epicondylitis patients. **C** Coronal proton density weighted image (1000/20) again showing intermediate signal at the origin of the wrist extensor tendon complex (*arrow*). **D** Coronal Fast STIR image (5833/48, TI 150) showing corresponding intense signal at the tendon origin (*arrow*), and adjacent soft tissue edema (*curved arrow*). This probably represents a full-thickness tear

are similar to those seen in patellar tendinitis [31–34], a chronic overuse condition associated with degenerative histopathologic changes and angiofibroblastic proliferation similar to epicondylitis [26, 27, 31, 32, 34].

In 13 patients, the character of abnormal T2 signal intensity included areas of more intense signal, similar to fluid. This type of signal has been associated with tendon tears in other locations [34–41]. In the rotator cuff, the high signal intensity of a tear is probably due to fluid acutely [39, 40], but scar and granulation tissue can cause high signal intensity in the subacute stage [40]. In the patellar tendon, a proliferation of fibroblasts or synovial cells and capillaries similar to angiofibroblastic hyperplasia in epicondylitis has been found filling tears in the tendon and accounting for high signal intensity [34]. From this, we postulate that intense fluid-like intratendon T2 signal intensity in epicondylitis could represent tears, tears filled with granulation tissue (angiofibroblastic hyperplasia), or angiofibroblastic hyperplasia without macroscopic tear. This is supported by a study correlating the MR findings in chronic epicondylitis with the histopathologic findings from surgical specimens [8]. While intense T2 signal intensity usually correlated with tears, at least one case in this series represented angiofibroblastic hyperplasia without frank tear [8].

Abnormal intratendon signal intensity was seen not only in epicondylitis but also in some asymptomatic individuals. Within our control population, a 27-year-old asymptomatic professional athlete had increased T1 and T2 signal intensity with tendon thickening of the origin of his wrist extensor tendon complex. Two other asymptomatic volunteers, one aged 39 years and the other a 23-year-old professional athlete, had increased intratendon T1 signal intensity. This abnormal intratendon signal intensity may be due to degeneration, one of the explanations offered [39, 42, 43] for asymptomatic intratendon signal seen in other locations [42–45]. The 27- and 23-year-old subjects seem young to have degenerative change, but both were professional athletes who may have developed early degeneration secondary to rigorous training.

An alternative explanation for increased intratendon signal intensity with short echo times in asymptomatic individuals is the “magic angle” phenomenon seen when tendons are placed at an angle of 45°–65° to the static magnetic field [46]. While there is some angulation of the wrist flexor and extensor tendons just beyond their origins from the epicondyle, all our normal volunteers were able to extend their elbows fully so the angle of these tendons with the static magnetic field was less than 45°. For this reason, the “magic angle” phenomenon is not felt to

be a likely cause of the increased intratendon T1 signal intensity seen in three of our normal volunteers.

Anconeus edema on MRI has been described in lateral epicondylitis [7], but only two of our patients demonstrated increased T2 signal intensity of the anconeus muscle, which was subtle in both cases.

Although periostitis has been described on MRI anecdotally [6], none of our patients demonstrated this. We did find evidence of an avulsive injury to the epicondyle in three patients. Microscopic evidence of avulsive injury to the epicondyle in epicondylitis has been documented in at least one case [18], and MRI evidence of avulsion in an epicondylitis patient has been described anecdotally [5].

A limitation of our study is that pathologic specimens were not available for direct comparison with MR findings since the mean duration of symptoms for our epicondylitis group was 5.5 months. Surgery is usually not performed prior to 1 year of symptoms [9, 10], and consequently there is no pathologic gold standard for our cases. Another limitation is our small number of both patients and volunteers, although we feel the population was adequate to show that MR changes of epicondylitis patients are reasonably specific. There is also the potential for population bias; not all patients with epicondylitis were imaged. A further limitation is that the first observer (M.E.S.) participated in the residency training of the second observer (C.E.M.), possibly causing the excellent interobserver agreement.

We did not attempt to study the potential effects of corticosteroid injections on the MRI appearance of epicondylitis. This could be a limitation, since corticosteroid injections into rabbit Achilles tendons cause tendon necrosis with a secondary fibroblastic healing response [47, 48]. One prior study, however, showed little effect of corticosteroid injection on the MRI appearance of the shoulder [49].

We conclude that increased intratendon T1 and T2 signal intensity with tendon thickening is associated with epicondylitis, but a small minority of asymptomatic volunteers also demonstrate these findings. Increased marrow T2 signal intensity within the involved epicondyle is occasionally seen in epicondylitis patients while anconeus edema is only rarely associated.

References

1. Bennett JB. Lateral and medial epicondylitis. Hand Clin 1994; 10: 157–163.
2. Ho CP. Sports and occupational injuries of the elbow: MR imaging findings. AJR 1995; 164: 1465–1471.
3. Patten RM. Overuse syndromes and injuries involving the elbow: MR imaging findings. AJR 1995; 164: 1205–1211.
4. Fritz RC, Steinbach LS. The elbow. In: Chan WP, Lang P, Genant HK, eds. MRI of the musculoskeletal system. Philadelphia: WB Saunders, 1994: 193–206.
5. Herzog RJ. Magnetic resonance imaging of the elbow. Magn Reson Q 1993; 9: 188–210.
6. Golimbu CN. Elbow. In: Firooznia H, Golimbu CN, Raffi M, Rauschning W, Weinreb JC (eds) MRI and CT of the musculoskeletal system. St Louis: Mosby-Year Book, 1992: 564–593.
7. Coel M, Yamada CY, Ko J. MR imaging of patients with lateral epicondylitis of the elbow (tennis elbow): importance of increased signal of the anconeus muscle. AJR 1993; 161: 1019–1021.
8. Potter HG, Hannafin JA, Morwessel RM, DiCarlo EF, O'Brien SJ, Altchek DW. Lateral epicondylitis: correlation of MR imaging, surgical, histopathologic findings. Radiology 1995; 196: 43–46.
9. Gellman H. Tennis elbow (lateral epicondylitis). Orthop Clin North Am 1992; 23: 75–82.
10. Nirschl RP. Elbow tendinosis/tennis elbow. Clin Sports Med 1992; 11: 851–870.
11. Leach RE, Miller JK. Lateral and medial epicondylitis of the elbow. Clin Sports Med 1987; 6: 259–272.
12. Briggs CA, Elliott BG. Lateral epicondylitis: a review of structures associated with tennis elbow. Anat Clin 1985; 7: 149–153.
13. Boyd HB, McLeod AC. Tennis elbow. J Bone Joint Surg Am 1973; 55: 1183–1187.
14. Hughes ESR. Acute deposition of calcium near the elbow. J Bone Joint Surg Br 1950; 32: 30–34.
15. Osgood RB. Radiohumeral bursitis, epicondylitis, epicondylagia (tennis elbow): a personal experience. Arch Surg 1922; 4: 420–433.
16. Nirschl RP, Pettrone FA. Tennis elbow. J Bone Joint Surg Am 1979; 61: 832–839.
17. Regan W, Wold LE, Coonrad R, Morrey BF. Microscopic histopathology of chronic refractory lateral epicondylitis. Am J Sports Med 1992; 20: 746–749.
18. Goldie I. Epicondylitis lateralis humeri (epicondylagia or tennis elbow): a pathogenetical study. Acta Chirg Scand 1964; [Suppl] 339: 1–119.
19. Cotran RS, Kumar V, Robbins SL. Robbins pathologic basis of disease, 4th edn. Philadelphia: WB Saunders, 1989.
20. Doran A, Gresham GA, Rushton N, Watson C. Tennis elbow: a clinicopathologic study of 22 cases followed for 2 years. Acta Orthop Scand 1990; 61: 535–538.
21. Coonrad RW, Hooper WR. Tennis elbow: its course, natural history, conservative and surgical management. J Bone Joint Surg Am 1973; 55: 1177–1182.
22. Chard MD, Cawston TE, Riley GP, Gresham GA, Hazleman BL. Rotator cuff degeneration and lateral epicondylitis: a comparative histologic study. Ann Rheum Dis 1994; 53: 30–34.
23. Backman C, Boquist L, Friden J, Lorentzon R, Toolanen G. Chronic achilles paratenonitis with tendinosis: an experimental model in the rabbit. J Orthop Res 1990; 8: 541–547.
24. Puddu G, Ippolito E, Postacchini F. A classification of achilles tendon disease. Am J Sports Med 1976; 4:145–150.
25. Clancy WG, Neidhart D, Brand RL. Achilles tendonitis in runners: a report of five cases. Am J Sports Med 1976; 4: 46–57.
26. Martens M, Wouters P, Burssens A, Mulier JC. Patellar tendinitis: pathology and results of treatment. Acta Orthop Scand 1982; 53: 445–450.
27. Roels J, Martens M, Mulier JC, Burssens A. Patellar tendinitis (jumper's knee). Am J Sports Med 1978; 6: 362–368.

28. Leadbetter WB. Cell matrix response in tendon injury. Clin Sports Med 1992; 11: 533–578.
29. Kannus P, Jozsa L. Histopathological changes preceding spontaneous rupture of a tendon. J Bone Joint Surg Am 1991; 73: 1507–1525.
30. Lufkin RB, Scheck A. Fasten your seatbelt: MR in for a wild ride. Diagn Imaging 1996; [May]: 27–33.
31. Khan KM, Bonar F, Desmond PM, et al. Patellar tendinosis (jumper's knee): findings at histopathologic examination, US, and MR imaging. Radiology 1996; 200: 821–827.
32. Yu JS, Popp JE, Kaeding CC, Lucas J. Correlation of MR imaging and pathologic findings in athletes undergoing surgery for chronic patellar tendinitis. AJR 1995; 165: 115–118.
33. El-Khoury GY, Wira RL, Berbaum KS, Pope TL, Monu JUV. MR imaging of patellar tendinitis. Radiology 1992; 184: 849–854.
34. Bodne D, Quinn SF, Murray WT, et al. Magnetic resonance images of chronic patellar tendinitis. Skeletal Radiol 1988; 17: 24–28.
35. Falchook FS, Zlatkin MB, Erbacher GE, Moulton JS, Bisset GS, Murphy BJ. Rupture of the distal biceps tendon: evaluation with MR imaging. Radiology 1994; 190: 659–663.
36. Rosenberg ZS, Cheung Y, Jahss MH, Noto AM, Norman A, Leeds MF. Rupture of the posterior tibial tendon: CT and MR imaging with surgical correlation. Radiology 1988; 169: 229–235.
37. Robertson PL, Schweitzer ME, Mitchell DG, Schlesinger F, Epstein RE, Frieman BG, Fenlin JM. Rotator cuff disorders: interobserver and intraobserver variation in diagnosis with MR imaging. Radiology 1995; 194: 831–835.
38. Iannotti JP, Zlatkin MB, Esterhai JL, Kressel HY, Dalinka MK, Spindler KP. Magnetic resonance imaging of the shoulder: sensitivity, specificity, and predictive value. J Bone Joint Surg Am 1991; 73:17–29.
39. Kjellin I, Ho CP, Cervilla V, Haghighi P, et al. Alterations in the supraspinatus tendon at MR imaging: correlation with histopathologic findings in cadavers. Radiology 1991; 181: 837–841.
40. Rafii M, Firooznia H, Sherman O, et al. Rotator cuff lesions: signal patterns at MR imaging. Radiology 1990; 177: 817–823.
41. Burk DL, Karasick D, Kurtz AB, et al. Rotator cuff tears: prospective comparison of MR imaging with arthrography, sonography, and surgery. AJR 1989; 153: 87–92.
42. Mirowitz S. Normal rotator cuff: MR imaging with conventional and fat-suppression techniques. Radiology 1991; 180: 735–740.
43. Schweitzer ME, Mitchell DG, Ehrlich SM. The patellar tendon: thickening, internal signal buckling, and other MR variants. Skeletal Radiol 1993; 22: 411–416.
44. Liou JTS, Wilson AJ, Totty WG, Brown JJ. The normal shoulder: common variations that simulated pathologic conditions at MR imaging. Radiology 1993; 186: 435–441.
45. Kaplan PA, Bryans KC, Davick JP, Otte M, Stinson WW, Dussault RG. MR imaging of the normal shoulder: variants and pitfalls. Radiology 1992; 184: 519–524.
46. Erickson SJ, Cox IH, Hyde JS, Carrera GF, Strandt JA, Estkowski LD. Effect of tendon orientation on MR imaging signal intensity: a manifestation of the "magic angle" phenomenon. Radiology 1991; 181: 389–392.
47. Unverferth LJ, Olix ML. The effect of local steroid injections on tendon. J Sports Med 1973; 1: 31–37.
48. Balasubramaniam P, Prathap K. The effect of injection of hydrocortisone into rabbit calcaneal tendons. J Bone Joint Surg Br 1972; 54: 729–734.
49. Schweitzer ME, Magbalon MJ, Fenlin JM, Frieman BG, Ehrlich S, Epstein RE. Effusion criteria and clinical importance of glenohumeral joint fluid: MR imaging evaluation. Radiology 1995; 194: 821–824.