A review of current concepts in flexor tendon repair: physiology, biomechanics, surgical technique and rehabilitation

Rohit Singh,[1,2] Ben Rymer,[1]
Peter Theobald,[2] Peter B.M. Thomas[1]

[1]University Hospital North Midlands, Stoke-on-Trent; [2]Cardiff School of Engineering, Cardiff University, UK

Correspondence: Rohit Singh, University Hospital North Midlands, Royal Stoke University Hospital, Newcastle Road, Stoke-on-Trent, Staffordshire, ST4 6QG, UK.
Tel.: +44.1782.715444.
E-mail: rohitamolsingh@hotmail.com

Key words: Flexor; tendon; review.

Contributions: the authors contributed equally.

Conflict of interest: the authors declare no potential conflict of interest.

Received for publication: 27 July 2015.
Accepted for publication: 13 September 2015.

Orthopedic Reviews 2015; 7:6125
doi:10.4081/or.2015.6125

Abstract

Historically, the surgical treatment of flexor tendon injuries has always been associated with controversy. It was not until 1967, when the paper entitled *Primary repair of flexor tendons in no man's land* was presented at the American Society of Hand Surgery, which reported excellent results and catalyzed the implementation of this technique into worldwide practice. We present an up to date literature review using PubMed and Google Scholar where the terms *flexor tendon*, *repair* and *rehabilitation* were used. Topics covered included functional anatomy, nutrition, biomechanics, suture repair, repair site gapping, and rehabilitation. This article aims to provide a comprehensive and complete overview of flexor tendon repairs.

Introduction

Historically, the surgical treatment of flexor tendon injuries has always been associated with controversy.[1] It was not until the mid-1700s that the first experimental studies of tendon healing were carried out by John Hunter.[2,3] Early work by Bunnell recognized the problems of restrictive adhesions around repair sites and coined the term *no man's land* for the region where the flexor tendon passes through the digital sheath.[4] It was not until the 1960s that primary tendon repair was first carried out but initial success rates for were not universally satisfactory.[5,6]

In 1967, a paper entitled *Primary repair of flexor tendons in no man's land* was presented at the American Society of Hand Surgery. This reported excellent results and catalyzed the implementation of this technique into worldwide practice.[7] As a result, numerous surgeons began to report results indicating success following primary repair of flexor tendons. Furthermore, many experimental studies were initiated, focusing on flexor tendon healing, suture techniques, suture materials and post-operative rehabilitation. The evolving interest in primary flexor tendon repair stimulated research, which raised further questions regarding what can be done to improve the healing process. The aim of this review is to provide a concise overview of this diverse field, including the anatomical, physiological, biomechanical and surgical concepts associated with flexor tendon repair. As a result of this article, we aim to improve the reader's understanding of this complicated topic.

Materials and Methods

PubMed and Google Scholar were searched using the terms *flexor tendon*, *repair* and *rehabilitation*. Papers not in English were excluded. One reviewer manually screened appropriate articles by review of titles and abstracts. Further review of full text was conducted by the first author and fifty-four articles were selected for inclusion based on their relevance and recent publication date.

The functional anatomy of flexor tendons of the hand

The word tendon derives from the Latin word *tendere*, meaning *to stretch* and alludes their role as flexible cables, transmitting forces to the fingers from the common flexor origin in order to provide strength and motion at the finger and wrist joints.

The flexor tendons of the hand mainly originate from the common flexor origin at the medial epicondyle of the distal humerus. These muscles are generally referred to as the long flexors, and run in two layers along the volar surface of the forearm. The superficial layer and deep layer are known as flexor digitorum superficialis (FDS) and flexor digitorum profundus (FDP) respectively. The FDS and FDP tendons travel through the carpal tunnel as two layers. The FDP tendon inserts centrally into the base of the distal phalanx, where it functions as a primary flexor at the distal interphalangeal joint (DIPJ) and as a secondary flexor at the proximal interphalangeal joints (PIPJ) and metacarpophalangeal joints (MCPJ). At the level of the MCPJ, the FDS tendon splits into two lateral bands with the FDP tendon running between, forming the *chiasm of Camper*. The two bands of the FDS tendon insert onto the middle phalanx, where they primarily flex the PIPJ, with secondary action at the MCPJ.[8]

The thumb has a separate primary flexor inserted into its distal phalanx; namely flexor pollicus longus (FPL). This originates from the volar surface of the radius and the adjacent interosseous membrane arrives at the thumb through the carpal tunnel.

Distal to the carpal tunnel, the tendons become enclosed within a synovial sheath. The function of the synovial sheath is mainly nutrition and lubrication, which reduces friction and improves gliding motion during excursion.[8]

The traditional classification of flexor tendons involves five zones, illustrated in Figure 1.[5] Zone I represents the region distal to the insertion of the FDS. Zone II describes the region from the distal palmar crease to the insertion of the FDS on the middle phalanx, where FDS and FDP share the fibro-osseous sheath of the digit. It is this zone that is referred to as *no man's land.* Zone III lies between the distal part of the transverse carpal ligament and the distal palmar crease. The tendons of Zone IV are located within the carpal tunnel. The most proximal part of the tendons where they fuse with the muscle belly (the musculo-tendinous junction) is found proximal to the carpal tunnel and represents Zone V.[5]

Within each the digit, the tendons are held in a fibro-osseous canal where the phalanges form the dorsal wall and the flexor sheath provides palmar coverage. The synovial part of the flexor sheath is segmentally thickened to form annular and cruciform pulleys. These keep the tendons in close proximity to the bone and prevent bowstringing.[9] There are five annular pulleys numbered in ascending order from proximal to distal (A1-A5) and three cruciform pulleys (C1-C3). A1, A3 and A5 are located over the MCPJ, PIPJ and DIPJ respectively. A2 and A4 are the longest and are located over the middle part of the proximal and distal phalanx respectively. The A2 and A4 pulleys are considered to be the biomechanically strongest and the most important to prevent tendon bowstringing.[9,10]

Morphologically the flexor tendons are similar to other tendons in the body, being formed predominantly of fibroblasts and extracellular matrix consisting of 70% water, 30% type 1 collagen, proteoglycans and 2% elastin.[11,12] The collagen fibers are arranged longitudinally and are closely packed running in parallel to the line of the tendon. The strength of the tendon prevails from the collagen fibers, whereas the flexibility of the tendons can be accounted for by elastin.[13]

Nutrition and healing

Sources of nutrition for tendons are twofold; intrinsic and extrinsic. The vascular perfusion of the tendon provides intrinsic nutrition. This derives from four main sources; longitudinal vessels form palmar tributaries, vessels form the synovial reflection in the palm, vessels from the osseous tendon insertion and those entering through the vinulae.[14,15] The majority of the vascular supply enters dorsally, leading to areas of hypovascularity.[15,16]

The extrinsic supply of nutrition occurs from synovial fluid by diffusion to hypovascular areas of tendon. The synovial fluid is produced from the synovium that lines the flexor sheath and production of this fluid is increased during finger movement.[16] The tendon healing process is largely dependent on unobstructed nutritional supply from its various sources.

Initial debate existed regarding whether tendon healing resulted from proliferation of intrinsic cell components or migration of extrinsic cells. Current understanding has largely resulted from research done in the early 1980s, where several studies using lacerated flexor tendon segments placed in an extracorporeal culture environment, with complete absence of extrinsic cells, were performed. This demonstrated that healing did still occur, establishing that the primary mechanism of self-repair is intrinsic and proving that tendons can heal intrinsically, without the need for external sources.[17,18] In fact, it is now known that extrinsic healing, through the ingress of inflammatory cells, leads to the formation of peri-tendinous adhesions. This effectively binds the tendon to the sheath and prevents movement. *In vivo* it is impossible to obliterate the action of extrinsic factors and tendon healing occurs from both processes, with the intrinsic system being the most important. Methods have been introduced to minimize adhesions and enhance the response to injury, such as early mobilization and meticulous tissue handling.[17,18]

The biomechanics of healthy tendons

In order to produce smooth motion of a joint there is a critical interaction of muscle and bone through the tendon-pulley system. The mechanical characteristics of tendons that allow transmission of these forces come from the linear orientation and strong cross-linking of collagen fibers.[19]

Tendons have viscoelastic properties exhibiting characteristics such as stress relaxation and creep. The values for the Young's modulus of flexor tendons lie between 1200-1800 Megapascals (MPa), with ultimate strength varying between 50-150 MPa. Ultimate strain ranges from 9-35% of the initial length.[20]

The mechanical properties of tendons are affected by pathological and physiological factors such as exercise, which increases tendon strength by stimulating tendon synthesis.[21] Rest periods in between exercise are also important for morphological adaption.[22] Strict immobilization leads to a time-dependent reduction in tensile stress and Young's modulus.[23] Degenerative changes occur in tendons during ageing, with a reduction in strength and size and an increased risk of rupture.[24]

The stress-strain curve for a typical tendon is not linear. A tendon under tension initially extends at low stress before elastic extension occurs, until deformation.[25] Microscopically, tendon fascicles have a crimped appearance to the fibrils when under zero load. The low stress required for initial extension is a result of straightening of the fibrillar crimps and allows an extension of approximately 3%.[25] However, once the crimping has straightened out, the load required to elongate the tendon increases, as elastic extension of the tendon fibers is required.

The load-displacement curves typically consist of an initial non-linear toe region, a linear slope and then a failure region. The linear slope of the curve represents the stiffness or rigidity of the tendon. The point of loss of linearity of the curve is considered the yield point, at which the material experiences sudden deformation. Beyond the yield point the tendon begins to change its cross-sectional area uniformly. The ultimate tensile stress is reached once the tendon is at its maximum value of stress.

The biomechanics of tendon repairs

The goal of primary repair of flexor tendons following transection is to recreate a strong construct with minimal iatrogenic trauma to the pulley sheath system and the tendon. The conventional method involves a combination of a core suture and a circumferential epi-

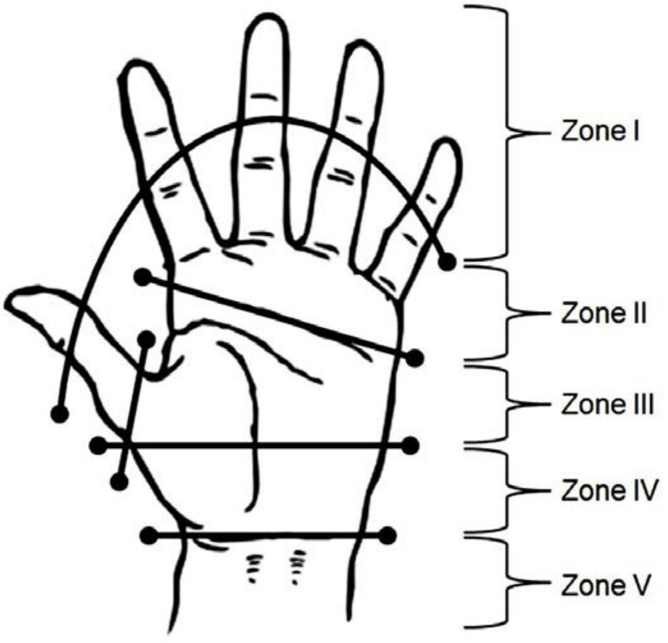

Figure 1. Schematic demonstrating the zones of the hand.

tendinous suture. The result of the suture technique must respect the biological and biomechanical considerations while providing the smoothest possible gliding surface. Good surgical technique is considered to have an impact on outcome, with emphasis placed on atraumatic technique. The ideal surgical scenario would involve a bloodless operative field, asepsis, satisfactory lighting, high-quality instruments and a comfortable position for the surgeon, who should exercise meticulous technique. Minimal sacrifices to the pulley system should be made. Three factors influence the biological and biomechanical properties of the repair, each of which is discussed in turn.

The configuration of the core suture

Initial flexor tendon repairs were performed in the same way as repairing a skin laceration. In order to avoid the inevitable pull through of the sutures, there were long periods of immobilization, with subsequent poor results.[26] In the early twentieth century, this problem was addressed by placing sutures at right angles to the orientation of the fibers.[27] In 1940, there was the addition of a perpendicular weave in either end, resulting in a technique with 4 strands of suture passing through the repair site with only two of them passing within the tendon.[27] In 1973, Kessler modified this by using only two strands passing through the repair site and within the tendon. There were initially two knots securing the sutures in exactly opposite sides of the repair. The Kessler suture has received numerous modifications and has become the most popular repair technique.[27] It is well accepted that techniques with greater number of suture strands crossing the repair site result in higher tensile strengths.[28-30] Studies by Strickland and Boyer suggest that the ideal technique contains at least four strands, although up to eight strands have been used in clinical practice.[31,32]

As the number of strands increases, there is a negative effect as friction and pressure within the synovial sheath increases. A further problem is placement of the knot. Placement within the repair site increases friction and can cause mechanical obstruction during tendon excursion if placed on the surface. Current biomechanical literature regarding tensile strength in relation to knot position shows no significant difference between placements.[33-35]

The epitendinous suture: depth and pattern

An epitendinous suture is used in addition to the core suture and is placed circumferentially around the repair. The aim of this suture is provide a smooth tendon surface and decrease the repair bulk.[36] Biomechanical studies have showed that this suture increases tensile strength at the repair site and decreases early postoperative repair gap formation.[37] More recent studies have looked at ways to improve the strength of this repair, and these suggest that purchase of at least 2 mm on each tendon stump improves strength.[38]

The suture material: size and type

Originally, sutures used in the twentieth century were composed of silk, cotton or catgut. Nylon was introduced in the early 1950s and is still commonly used. The ideal suture material is biologically inert, has a high ultimate tensile strength (UTS) and a high modulus of elasticity, handles and ties easily and holds well when knotted.[39] The UTS of a typical suture used for a core suture in tendon repair is in the order of 35N.[39]

The suture material can be monofilament or multifilament. Monofilaments run through tissues smoothly with no sequestered spaces for bacteria to dwell, but handle less well when compared to multifilament sutures.[40] Traditionally, the most commonly used materials are synthetic polyester for the core suture, usually 3-0 caliber, and a monofilament for the epitendinous suture, usually 6-0 caliber.[41-43]

Formation of repair site gaps

The formation of a gap at the tendon repair site represents a dehiscence of the repair. It was believed that the formation of such a gap would lead to flexor tendon adhesions, decreased tendon glide and consequently digital stiffness.[44,45] Further follow up studies have been conducted to measure gap formation using implanted intra-tendinous metal markers. This concluded that gaps were common and did not affect intra-synovial adhesions or stiffness and did not always result in a poor outcome.[46]

The formation of a gap greater than 3 mm is used to predict subsequent failure of the repair. Gelberman *et al.* studied tendon failure in an *in vivo* canine model and concluded that repaired tendons with a gap of less than 3 mm had increased strength compared to those with a greater than 3 mm gap.[47]

Post-operative rehabilitation

Post-operative management has a significant bearing on the outcome of flexor tendon injuries. Rehabilitation must balance between protection of the repair from excessive forces and prevention of adhesions. Historically, tendon repairs were immobilized for at least three weeks to protect the repair from rupture. This has now been abandoned as early motion of the repair leads to improved tendon excursion, fewer adhesions and improved tensile properties.[48] Numerous rehabilitation protocols are available.[49]

Experiments involving chicken tendons have showed that the intrinsic response of lacerated flexor tendons to cyclical loading produces intensified multiplication and migration of fibroblasts, as well as stimulating collagen synthesis.[50] Kubota *et al*. examined the individual effects of motion and tension on the healing response of injured chicken tendons and found that both motion and tension enhance response to injury. This has encouraged surgeons to prescribe active protocols that further increase the *motion stress* on the repair site in order to stimulate healing.

A recent *in vivo* study of flexor tendon healing demonstrated that during the first three weeks after repair, there is no increase in ultimate tensile strength but there is a significant increase in repair site rigidity.[51] These studies suggest that the mechanical changes of tendon repairs seen in the first few post-operative weeks relate to the effects of early healing. However, it is possible that the process of cyclic loading itself leads to alterations in the tensile properties of the repair site.

It is widely accepted that the tendon should be moved soon after the repair is performed in order to prevent adhesions. There is however considerable discussion over the duration before movement is started, the excursion of the movement required and the loads placed on the tendon. Opinions range from gentle, passive movement to active movement, potentially against increasing resistance. A systematic review of reported results from varying rehabilitation protocols has confirmed that static splinting is likely only to yield 60% of the total active range of motion when compared to dynamic splinting protocols.[52] Dynamic splinting includes both active movement, where the repaired musculo-tendinous unit is used to actively move the finger, and passive movement, where the finger is moved by the therapist or by using the other hand and not by using the repaired structure. Current evidence reports that active rehabilitation protocols give a better range of movement, smaller flexion contractures and greater patient satisfaction when compared to passive rehabilitation protocols.[53]

Clinical results of flexor tendon repair

The outcome of tendon repair in the clinical situation is less predictable than the results of biomechanical studies would suggest for several reasons. Studies in humans are obliged to be done opportunistically, as it is ethically unacceptable to section tendons in humans for the sole purpose of performing a repair. Thus, the repairs performed in humans are done under varying conditions, including quality and size of tendon, zone of injury, quality of repair and the rehabilitation protocol used. Accidental overstress of the repair can occur and compliance with hand therapy can be poor. Patients with a variety of medical conditions affecting tendon quality, such as rheumatoid arthritis, are often included in studies and can confound results. Surgical site infections, which lead to inhibition of movement from pain and swelling and an increase in adhesions, are also included in outcome studies.

Current data suggests good or excellent outcomes in over 75% of flexor tendon repairs.[54] Rupture occurs in 4-10% of finger flexor repairs and 4-17% of long thumb flexor repairs.[54] Modern tissue engineering approaches and focused rehabilitation protocols are the future avenues for further improving recovery.

References

1. Manske PR. History of flexor tendon repair. Hand Clin 2005;21:123-7.
2. Gratz CM. The history of flexor tendon suture. Med J Rec 1928;127:213-5.
3. Mason ML, Shearon CG The process of tendon repair. An experimental study of tendon suture and tendon graft. Arch Surg 1932;25:612-92.
4. Bunnell S. Repair of tendons in the fingers and description of two new instruments. Surg Gynecol Obstet 1919;126:103-10.
5. Verdan CE. Primary repair of flexor tendons. Am J Orthop 1960;42A:647-57.
6. Lindsay WK, McDougall EP. Direct digital flexor tendon repairs. Plast Reconstruct Surg 1960;26:613-21.
7. Kleinert HE, Kutz JE, Ashbell TS, Martinez E. Primary repair of lacerated flexor tendons in No Man's Land. J Bone Joint Surg 1967;49A:577.
8. Ochiai N, Matsui T, Miyaji N, et al. Vascular anatomy of flexor tendons. I. Vincular system and blood supply of the profundus tendon in the digital sheath. J Hand Surg Am 1979;4:321-30.
9. Doyle JR. Palmar and digital flexor tendon pulleys. Clin Orthop 2001:84-96.
10. Peterson WW, Manske PR, Bollinger BA, et al. Effect of pulley excision on flexor tendon biomechanics. J Orthop Res 1986;4:96-101.
11. O'Brien M. Functional anatomy and physiology of tendons. Clin Sports Med 1992;11:505-20.
12. Jozsa L, Kannus P. Histopathological findings in spontaneous tendon ruptures. Scand J Med Sci Sports 1997;7:113-8.
13. Kannus P, Jozsa L, Natri A, Jarvinen M. Effects of training, immobilization and remodelling on tendons. Scand J Med Sci Sports 1997;7:67-71.
14. Lundborg G, Myrhage R. The vascularization and structure of the human digital tendon sheath as related to flexor tendon function. An angiographic and histological study. Scand J Plast Reconstr Surg 1977;11:195-203.
15. Lundborg G, Myrhage R, Rydevik B. The vascularization of human flexor tendons within the digital synovial sheath region: structural and functional aspects. J Hand Surg Am 1977;2:417-27.
16. Harrison RK, Jones NIE, Clayton E, et al. Mapping of vascular endothelium in the human flexor digitorum profundus tendon. J Hand Surg 2003;28A:806-13.
17. Manske PR, Gelberman RH, Lesker PA. Flexor tendon healing. Hand Clin 1985;l:25-34.
18. Manske PR, Gelberman RH, Vande Berg JS, Lesker PA. Intrinsic flexor-tendon repair. A morphological study in vitro. J Bone Joint Surg Am 1984;66:385-96.
19. Goodman HJ, Choueka J. Biomechamcs of the flexor tendons. Hand Clin 2005;21:129-49.
20. Woo S, An KN, Frank C. Anatomy, biology and biomechanics of tendon and ligament. In: Buckwalter JA, Einhorn TA, Simon SR, eds. Orthopaedic basic science.: biology and biomechanics of the musculoskeletal system. Rosemont: American Academy of Orthopaedic Surgeons; 2000.
21. O'Brien M. Functional anatomy and physiology of the tendons. Clin Sports Med 1992;2:505-20.
22. Lin TW, Cardens L, Soslowski LJ. Biomechanics of tendon injury and repair. J Biomech 2004;37:865-77.
23. Yamamoto E, Hayashi K, Yamamoto N. Effects of stress shielding on the transverse mechanical properties of rabbit patellar tendons. J Biomech Eng 2000;122:608-14.
24. Goodman HJ, Choueka J. Biomechamcs of the flexor tendons. Hand Clin 2005;21:129-49.
25. James R, Kesturu G, Balian G, Chhabra AB. Tendon: biology, biomechanics, repair, growth factors, and evolving treatment options. J Hand Surg Am 2008;33:102-12.
26. Ketchum LD Suture materials and suture used in tendon repair. Hand Clin 1985;43:53.
27. Nicoladoni C. Ein vorchlag zur Sehnennaht. Wien Klin Wochenschr 1880;52:1417.
28. Dinopoulos HT, Boyer MI, Burns NE, et al. The resistance of a four- and eight-strand suture technique to gap formation during tensile testing: an experimental study of repaired canine flexor tendons after 10 days of in vivo healing. J Hand Surg 2000;25A:489-98.
29. Thurman RT, Trumble TE, Hanel DP, et al. Two-, four-, and six-strand zone Il flexor tendon repairs: An in situ biomechanical comparison using a cadaver model. J Hand Surg 1998;23A:261-5.
30. Winters SC, Gelberman RH, Woo SL, et al. The effects of multiple-strand suture methods on the strength and excursion of repaired intrasynovial flexor tendon: a biomechemical study in dogs. J Hand Surg 1998;23A:97-104.
31. Strickland JW. Flexor tendon injuries: I. Foundations of treatment. J Am Acad Orthop Surg 1995;3:44-54.
32. Boyer MI. Flexor tendon biology. Hand Clin 2005;21:159-66.
33. Aoki M, Pruitt DL, Kubota H, Manske PR. Effect of suture knots on tensile strength of repaired canine flexor tendons. J Hand Surg 1995;20B:72-5.
34. Momose T, Amadio PC, Zhao C, Zobitz NE, et al. The effect of knot location, suture material, and suture size on the gliding resistance of flexor tendons. J Biomed Mater Res 2000;53:806-11.
35. Pruitt DL, Aoki M, Manske PR. Effect of suture knot location on tensile strength after flexor tendon repair. J Hand Surg 1996;21A:969-73.
36. Lister GD, Kleinert HE, Kutz JE, Atasoy E. Primary flexor tendon repair followed by immediate controlled mobilization. J Hand Surg 1977;2:441-51.
37. Wade PJ, Muir IF, Hutcheon LL. Primary flexor tendon repair: the mechanical limitations of the modified Kessler technique. J Hand Surg Br 1986;11B:71-6.
38. Merrell GA, Wolfe SW, Kacena WJ, et al. The effect of increased peripheral suture purchase on the strength of flexor tendon repairs. J Hand Surg Am 2003;28A:464-8.
39. Marrero-Amadeo IC, Chauhan A, Warden SJ, Merrell GA. Flexor tendon repair with a knotless barbed suture: A comparative biomechanical study. J Hand Surg Am 2011;36:1204-8.
40. Campbell WC, Canale ST, Beaty JH, eds. Campbell's operative orthopaedics. 11 ed. St Louis: Mosby/Elsevier; 2008.
41. Singer G, Ebramzadeh E, Jones NF, Meals R. Use of the Taguchi method for biomechanical comparison of flexor-tendon-repair techniques to allow immediate active flexion. A new method of analysis and optimization of technique to improve the quality of the repair. J Bone Joint Surg Am 1998;80:1498-506.
42. Taras JS, Raphael JS, Marczyk SC, Bauerle WB. Evaluation of suture calibre in flexor tendon repair. J Hand Surg 2001;26A:1100-4.

43. Wade PFJ, Wetherell RG Amis AA. Flexor tendon repair: significant gain in strength from the Halsted peripheral suture technique. J Hand Surg 1989;14B:232-5.
44. Tang JB, Wang B, Chen F, et al. Biomechanical evaluation of flexor tendon repair techniques. Clin Orthop Rel Res 2001;386:252-9.
45. Ketchum LD. Primary tendon healing: a review. J Hand Surg 1977;2:428-35.
46. Silfverskiöld KL, May EJ, Tornvall AH. Gap formation during controlled motion after flexor tendon repair in zone 11: a prospective clinical study. J Hand Surg Am 1992;17:539-46.
47. Gelberman RH, Boyer MI, Brodt MD, et al. The effect of gap formation at the repair site on the strength and excursion of intrasynovial flexor tendons. An experimental study on the early stages of tendon-healing in dogs. J Bone Joint Surg Am 1999;81:975-82.
48. Wada A, Kubota H, Miyanishi K, et al. Comparison of postoperative early active mobilization and immobilization in vivo utilising a four-strand flexor tendon repair. J Hand Surg Br 2001;26:301-6.
49. Woo SL, Gelberman RH, Cobb NG, et al. The importance of controlled passive mobilization on flexor tendon healing. A biomechanical study. Acta Orthop Scand 1981;52:615-22.
50. Tanaka H, Manske PR, Pruitt DL, Larson BJ. Effect of cyclic tension on lacerated flexor tendons in vitro. J Hand Surg Am 1995;20:467-73.
51. Kubota H, Manske PR, Aoki M, et al. Effect of motion and tension on injured flexor tendons in chickens. J Hand Surg Am 1996;21:456-63.
52. Sameem M, Wood T, Ignacy T, et al. A systematic review of rehabilitation protocols after surgical repair of the extensor tendons in zones V-VIII of the hand. J Hand Ther 2011;24:365-72.
53. Trumble TE, Vedder NB, Seiler JG, et al. Zone-II flexor tendon repair: a randomized prospective trial of active place-and-hold therapy compared with passive motion therapy. J Bone Joint Surg Am 2010;92:1381-9.
54. Tang J. Clinical outcomes associated with flexor tendon repair. Hand Clin 2005;21:199-210.