Flexor Tendon Repair
Healing, Biomechanics, and Suture Configurations

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KEYWORDS

• Flexor tendon • Suture configuration • Biomechanics • Growth factors • Biological augmentation

KEY POINTS

- Tendon healing is a complex process that must coordinate healing within the tendon while limiting the amount of fibrosis in the surrounding tissues.
- The ultimate goal of surgical intervention has remained constant: to achieve enough strength to allow early motion, to prevent adhesions within the tendon sheath, and to restore the finger to normal range of motion and function.
- Although certain suture materials may have superior tensile properties, the number of strands crossing a repair site is the most important factor in the overall strength of the repair.
- Recent research has been focused on using pharmacologic agents to modify the healing environment to increase the healing response within the tendon while decreasing the adhesion formation between the tendon and its sheath.

INTRODUCTION

Before the 1960s, tendon repairs in the digits were rarely performed because of the universally poor outcomes, particularly in zone II, lending to the term "no man's land."[1] Sterling Bunnell is often credited as being one of the first to stress the necessity of gentle and precise surgical technique in the treatment of flexor tendon injuries.[2] Additional research has focused on different suture configurations or number of core sutures to maximize the strength of tendon repair and postoperative rehabilitation protocols to maximize function.[3,4] The ultimate goal of surgical intervention has remained constant: to achieve enough strength to allow early motion, to prevent adhesions within the tendon sheath, and to restore the finger to normal range of motion and function. In recent years, basic science research has focused on biological factors that will increase the tendon stability after surgical repair, increase intratendinous healing, and decrease extratendinous fibrosis in order to maximize clinical outcomes.[5,6] It is in this area that there is the potential for great advancement of our understanding of tendon healing.

The purpose of this article is to review the relevant tendon anatomy, biology of tendon healing, biomechanics of tendon healing, biological strategies to augment tendon healing, and suture configurations to maximize strength and motion.

TENDON ANATOMY

Tendons are collagen-based tissues that connect muscle to bone. Tendons are primarily composed of type I collagen, whereas the surrounding endotenon and epitenon are primarily composed of type III collagen. Collagen is synthesized and secreted by tenocytes present within the tendon.

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Once secreted, the collagen fibers arrange into triple helices and undergo cross-linking to increase their strength and stability.[7] The surrounding extracellular matrix (ECM) is thought to help with gliding between collagen fibrils and to provide functional stability to the fibers.

The collagen fiber units are bound together by endotenon fascicles. These fascicles bind together within the epitenon to form the tendon (Fig. 1). Lymphatic, vascular, and neural elements are present within the endotenon to supply the fibroblasts. The epitenon contains the blood vessels and tracts for the lymphatics and nerves. The tendon sheath is covered with synovial cells that provide lubrication to aid in gliding of the tendon within the sheath. Outside of the hand, tendons are not typically enclosed within a sheath and are covered by a continuous paratenon that contains the vascular elements to supply the endotenon and epitenon.

Both the flexor digitorum profundus (FDP) and flexor digitorum superficialis (FDS) tendons in the digits receive dual nutritional supply from vascular perfusion and synovial diffusion.[8,9] The vascular supply is through vincula with each tendon having 2: a longus and a brevis. Proceeding from proximal to distal, the first vinculum encountered is the vinculum longus superficialis (VLS), arising just proximal to the decussation of the FDS and coming off the floor of the digital sheath of the proximal phalanx (Fig. 2). The vinculum brevis superficialis consists of small triangular mesenteries near the insertion of the FDS. The vinculum longus profundus arises from the superficialis at the level of the proximal interphalangeal (PIP) joint. Finally, the vinculum brevis profundus arises near the insertion of the FDP. Each vinculum inserts on the dorsal aspect of the tendon, creating a richer blood supply on the dorsal side of the tendon. The vincula are important in the repair of injured tendons as they may hold the tendons out to length after injury, and one must be careful not to injure any maintained vincula while repairing an injured tendon, thereby decreasing the already tenuous blood supply.

The flexor tendons pass through the carpal canal and then enter a series of pulleys, creating the flexor tendon sheath in the digits. The flexor tendon sheath starts with the first annular pulley, or A1, overlying the metacarpal heads. There are a total of 5 annular pulleys (A1–A5) and 3 cruciate pulleys (C1–C3). The more stout annular pulleys help hold the tendon close to the phalanges, whereas the cruciate pulleys allow for some mobility of the sheath with finger flexion. The tendon sheath needs to be preserved, if at all possible, to maintain the normal function of the repaired tendon. The A1, A3, and A5 pulleys all arise from the volar plates of the metacarpophalangeal, PIP, and distal interphalangeal joints, respectively. These pulleys may be incised and used as windows through which to perform tendon repairs.[10] The A2 and A4 pulleys should be maintained to prevent bowstringing of the tendon after repair.

BIOLOGY OF TENDON HEALING

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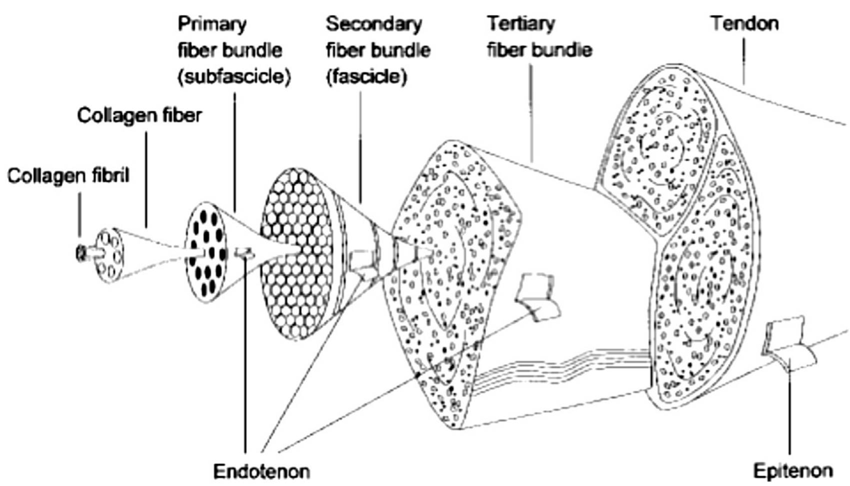

Fig. 1. Basic tendon structure. Collagen fibrils are bound together to form a collagen fiber. Multiple fibers are surrounded by endotenon in multiple stages to form a tertiary fiber bundle. Several tertiary fiber bundles are bound together by the epitenon to form the tendon. (*From* Kannus P. Structure of the tendon connective tissue. Scand J Med Sci Sports 2000;10:313; with permission.)