Pathomechanics and Management of Secondary Complications Associated with Tendon Adhesions Following Flexor Tendon Repair in Zone II

Shrikant J. Chinchalkar^{1,2} Juliana Larocerie-Salgado^{1,2} Nina Suh²

¹Department of Hand Therapy, Roth McFarlane Hand and Upper Limb Centre, St. Joseph's Hospital, London, Ontario, Canada

²Roth McFarlane Hand and Upper Limb Centre, St. Joseph's Hospital, London, Ontario, Canada

Address for correspondence Juliana Larocerie-Salgado, OT, MSc, CHT, Roth McFarlane Hand and Upper Limb Centre, St. Joseph's Hospital, 268 Grosvenor St, London, ON N6A 4V2, Canada (e-mail: jularocerie@hotmail.com).

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Abstract

Keywords

- flexor tendon
- early active mobilization
- complications
- synergistic wrist motion

Despite the number of rehabilitation strategies and guidelines developed to maximize the gliding amplitude of repaired tendons, secondary complications, such as decreased range of motion and stiffness associated with tendon adhesions, commonly arise. If left untreated, these early complications may lead to secondary pathomechanical changes resulting in fixed deformities and decreased function. Therefore, an appropriate treatment regimen must not only include strategies to maintain the integrity of the repaired tendon, but must also avoid secondary complications due to reduced gliding amplitude. This review presents a biomechanical analysis of the dynamics of tendon gliding following repair in zone II and rehabilitation strategies to minimize secondary complications related with tendon adhesions.

Introduction

Complete restoration of proper hand function following flexor tendon injury remains a formidable challenge. Despite strong efforts by the surgeon, therapist, and patient, complications, such as flexion contractures, tenosynovitis, tendon adhesions, and limited active flexion, may arise. A recent systematic review by Dy and colleagues¹ revealed that 4% of all flexor tendon repair cases require surgical intervention to address these common complications; whereas, a review by Tang² demonstrated that only 80% of all repaired tendons achieved a good to excellent recovery of function (>70% return of preinjury motion) based on the “Strickland Criteria of Assessment of Functional Outcomes.”

The anatomic location of a flexor tendon injury has helped to guide treatment options and served as a predictor of outcomes following injury. Although there remains little consensus on a standard approach to tendon repair, hand surgeons agree that an ideal repair should be simple and

atraumatically performed to allow for a smooth gliding repair site with minimal gap formation and provide sufficient strength to withstand the tensile forces experienced during the rehabilitation period.^{3,4}

For years, there was a widespread belief that tendon healing was solely dependent on extrinsic healing from vascular perfusion and cellular ingrowth through adhesions.^{5,6} Consequently, flexor tendons were traditionally immobilized in an orthosis or cast for 3 or more weeks, until the repair site could withstand motion. Unfortunately, these immobilization protocols allowed for uncontrolled formation of adhesions provoking catastrophic consequences, including reduced tendon glide, joint contracture, and loss of functional mobility. Nowadays, prolonged immobilization is solely reserved for patients who are unable to cooperate, such as young children and people with cognitive impairments.^{7–9}

Advances in repair techniques in the late 1970's using 4- and 6-strand core sutures has made early mobilization of tendons possible, providing better outcomes and low rupture

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rates. Initially, most studies advocated some form of passive postoperative mobilization protocol, including Kleinert, Duran, modified Kleinert, and Chow's Washington Regime.^{10–14} However, complications inherent to tendon adhesions, such as decreased active flexion, were still present as repaired tendons in passive protocols would bunch up or fold upon themselves as opposed to glide. More recently, several studies have demonstrated that early active mobilization techniques, ranging from synergistic wrist motion (SWM) to active mid-range finger flexion, offer improved tendon nutrition, higher healing rates, increased ultimate range of motion (ROM), reduced adhesion formation, and the ability to more rapidly achieve tensile strength, due to proximal gliding of the repaired tendon.^{15–28}

Despite advances in the rehabilitation of flexor tendon repairs since the early 90's, a Cochrane review by Thien, Becker, and Theis²⁹ concluded that there remains insufficient evidence from randomized control trials to define which is the best mobilization strategy. A recent systematic review by Starr and colleagues³⁰ in 2013 indicated that despite efforts to decrease the formation of intrasynovial adhesions and facilitate the restoration of the gliding surface, complications, including adhesions formation, continued to occur in 6% of the cases undergoing early active rehabilitation protocols guidelines and in 9% of those undergoing passive mobilization protocols.

It is relevant to note that any disturbance of tendinous motion or ligamentous control can alter skeletal alignment and result in functionally limiting complications, following tendon repairs.³¹ During the rehabilitation period, one must consider the critical balance between these structures and understand how flexor tendon adhesions can influence gliding of the extensor and intrinsic tendons. During flexion or extension of a digit, to-and-fro motion of the flexors and extensors and volar and dorsal translation of the intrinsics are critical to allowing digits to function normally. Primary pathology of adhesions or joint contractures may slowly progress, altering flexor, extensor, and intrinsic tendon vectors leading to a kinematic chain imbalance and the development of secondary complications.^{1,32–35}

Although tendon adhesions may be accompanied by other postoperative complications, such as gap formation and rupture, this paper will focus on reviewing relevant literature to understand the pathomechanics associated with tendon adhesions and reduced gliding amplitude in zone II. Kinematic imbalance and decreased function associated with tendon adhesions in zone II were also investigated by means of cadaver dissections and suturing of the tendons to simulate and comprehend the effect of tendon adhesions at different levels based on the orientation of the flexor digitorum profundus (FDP)/flexor digitorum superficialis (FDS). Tendons were sutured at three different locations within zone II, ensuring tendon gliding remained unrestricted under the pulleys when both tendons were pulled simultaneously: 1) distal to A2 pulley; 2) proximal to A3 pulley; 3) distal to A3 pulley. In addition, due to a lack of literature elucidating variations in rehabilitation approach that respect the anatomic differences of flexor tendon injuries within zone II, this paper will provide therapeutic suggestions to reduce or minimize the formation of these secondary complications.

Biomechanical implications associated with tendon repair including tendon ruptures, dehiscence, or gap scar formation have been excluded in this paper since these complications dictate surgical intervention.

Zone II Flexor Tendon Injuries: Anatomy and Common Complications

Zone II includes both the FDS and the FDP tendons as they travel within the fibrosesous sheath from the A1 pulley to the FDS insertion site. Bunnell in Williamson and Richards³⁵ referred to this zone of injury as “no man's land” due its anatomic complexity and to the inherent complication of injured tendons adhering to its surrounding soft tissue resulting in poor rehabilitation outcomes.³⁶ Zone II differs in complexity from other zones of injury due to its complicated anatomical structure and function, the unique spatial relationships between FDS and FDP tendons (→Fig. 1), and the variation in sources and distribution of synovial nutrition, make this particular region more prone to adhesion formation.^{36,37}

Complications observed in flexor tendon repair are commonly divided into early and late categories. Those in the early category include infections and wound healing complications, suture pullout, tendon repair or pulley ruptures, and triggering of the tendon repair in the sheath. Later complications include tendon adhesions, rupture of the repair site, joint contractures, quadriga, lumbrical plus deformity, and bowstringing.^{1,34,35} In our clinical experience, complications in flexor tendon rehabilitation are likely to arise in different stages of the postoperative management, which is divided into three categories based on the stages of healing and tensile strength^{35,38} 1) orthotic intervention phase (4–6 weeks), 2) active phase (6–8 weeks), and 3) strengthening phase (8–12 weeks).

During the orthotic intervention phase, complications may include tendon adhesion, reduced differential tendon motion between FDS and FDP in relation to the fibrosesous sheath, and/or contracture formation. However, the secondary pathomechanical changes often do not become noticeable until the active mobilization phase. This may also continue into the strengthening phase, in which there is a noticeable decrease in tendon excursion, resulting in a loss of ROM, strength, and function. As a result, if necessary steps are not taken earlier on in phase I, these complications may persist and lead to secondary limitations in motion and function.

Cadaveric Simulations of Common Complications

Tendon Adhesions

Tang and Shi³⁹ were the first to recognize the variation in the biomechanical features of zone II, and divided it into four subzones and developed treatment methods to suit each subzone. Considering the differences between these levels, it is important to recognize potential variations in complications following tendon injury and repair and modify the rehabilitation program accordingly. Cadaveric experimentation demonstrated that, from a rehabilitation standpoint, zone II can be divided into three sub groups to define

Fig. 1 Orientation changes of flexor digitorum superficialis and flexor digitorum profundus tendons in zone II (arrows).

postoperative treatment: zone IIa—FDS inferior to FDP; zone IIb—FDS lateral to FDP; and zone IIc—FDS superior to FDP (►**Fig. 2**). Density of adhesion formation will be most profound where there is the least amount of differential tendon motion within the fibrous sheath, thus affecting joint motion. In general, the bulk of adhesion will likely have difficulty gliding beneath the A2 and A3 pulleys, reducing angular motion at the interphalangeal (IP) joints.

Our clinical observation has demonstrated that if the adhesions occur in zone IIc and the tendons become adhered to each other, it is likely that there will be limited differential gliding between the FDS and FDP tendons within the fibrous sheath. Therefore, both tendons will act as FDS only, limiting motion at the distal interphalangeal joint (DIPJ) during composite flexion, leading to a straight finger grasp and reduced grip strength⁴⁰ (►**Fig. 3**). If adhesions occur distally in subzone IIb, the individual will be more likely to have greater FDS motion despite adhesion formation, while FDP excursion will be slightly improved (►**Fig. 4**). Finally, if the injury occurs in zone IIa, both proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints will have greater gliding amplitudes (►**Fig. 5**).

To minimize adhesion formation as aforementioned, rehabilitation exercises during the “orthotic intervention phase” should allow for the greatest amount of differential excursion of the repaired tendons with the minimal amount of resistance to tendon gliding.^{25,28,38,41,42} Savage,²¹ in 1988, demonstrated that repaired tendons showed the least amount of adhesions and higher repair strength when individuals synergistically flexed and extended fingers with active wrist movement, protocol known as “synergistic wrist motion” (SWM). This concept was further validated by Cooney and colleagues, in 1989,²⁸ when differential tendon gliding was analyzed, while using three different rehabilitation protocols. The concept of SWM was then popularized by Strickland and Cannon in 1993,⁴³ and continues to be widely used to date, particularly following a more recent randomized prospective trial by Trumble and

colleagues.⁴⁴ In their study, patients were randomly assigned to either early active motion with place and hold (SWM) or a passive motion protocol. Patients in the active motion group had

Fig. 2 Subdivision of zone II. Zone IIA: flexor digitorum superficialis inferior to flexor digitorum profundus, zone IIB: flexor digitorum superficialis lateral to flexor digitorum profundus, zone IIC: flexor digitorum superficialis superior to flexor digitorum profundus.

Fig. 3 Clinical observation of tendon adhesion in zone IIC (A). Cadaveric simulation of tendon adhesion in zone IIC (B) demonstration flexor digitorum superficialis and flexor digitorum profundus tendons acting as one tendon on the proximal interphalangeal joint along with restricted distal interphalangeal joint flexion (C).

significantly more active composite flexion, with 94% of the patients showing ≥ 125 degrees of composite flexion, versus 62% of the patients in the passive protocol group. Additionally, patients in the active group were able to return to work on full duties with no restriction at 82 days, as opposed to 103 days in the passive protocol group.⁴⁴

Tanaka and colleagues²⁵ proposed a modified SWM protocol. They demonstrated in cadaveric models that tendons repaired with a 4-strand core suture and an epitendinous running suture can overcome a significant amount of synergistic tendon gliding,

Fig. 4 Clinical observation of tendon adhesion in zone IIB (A). Cadaveric simulation of tendon adhesion in zone IIB (B) demonstrating greater flexor digitorum superficialis excursion, along with some flexor digitorum profundus function (C).

ranging from complete wrist flexion combined with full finger extension to complete wrist extension combined with metacarpophalangeal (MCP) extension and IP joints flexion. These authors reported that this ROM exercise allowed for a greater tendon excursion than previously reported SWM protocols, with a mean of 20.9 mm of total FDP excursion from wrist flexion to 60 degrees of extension and a peak force of 1.77 N with 60 degrees of wrist extension, significantly below the force of 7 N needed to produce initial gap formation, as previously reported.⁴² Additionally, placing the wrist in extension while flexing fingers has been demonstrated to significantly decrease

Fig. 5 Clinical observation of tendon adhesion in zone IIA (A). Cadaveric simulation of tendon adhesion in zone IIA (B). Greater flexion of both the proximal interphalangeal and distal interphalangeal joints (C).

the overall work of flexion, and subsequently, the risk of gap formation.^{25,36}

In addition to the concept of SWM, early active motion protocols composed of short-arc active finger flexion within the constraints of a dorsal block orthosis were introduced as early as 1989, by Cullen et al and Small et al.^{16,27} A few years later, Tang and colleagues²⁶ promoted the concept of active mid-range flexion within an orthosis in which the wrist was positioned in slight extension. Such an approach was claimed to permit the greatest amount of proximal tendon gliding and

Fig. 6 Synergistic wrist motion, as per Tanaka and colleagues.²⁵

differential excursion in zone II repairs, while still protecting the repair site from excessive loading leading to failure.²⁶ They demonstrated that tendons repaired with Tang or Cruciate suturing technique can withstand a fair amount of resistance, 53.4 N and 46.3 N, respectively, and introduced the concept of active mid-range composite flexion. Furthermore, Wu and Tang⁴⁵ demonstrated that active mid-range flexion imposes minimal resistance to tendon gliding. In recent times, Frueh and colleagues⁴⁶ demonstrated in a retrospective study that active mid-range flexion had lower rupture rate and better ROM in the short term in comparison to passive mobilization solely.

Since differential motion between FDP and FDS tendons in relation to the angular motion at both joints remains unknown, we recommend a combination of SWM protocols, as per Tanaka and colleagues²⁵ (→**Fig. 6**), and early active mid-range flexion, as per Tang⁴⁷ (→**Fig. 7**), during the orthotic intervention phase. This combination seems to permit the greatest tendon excursion and differential tendon gliding of FDS and FDP, while protecting the repair site against forces leading to eventual repair failure and rupture. To minimize excessive loading associated with joint stiffness on repaired tendons, patients should perform passive composite flexion

Fig. 7 Active mid-range flexion, as per Tang⁴⁷.

Fig. 8 “Warm-up” exercises: passive flexion and active extension in orthosis.

and active extension within the confines of the orthosis, as “warm-up” exercises prior to performing active motion as described earlier (►Fig. 8).

If there are clearer signs of tendon adhesions during phase 1 of the rehabilitation, in which passive flexion is much greater than active flexion, isolated IP joint flexion exercises within the orthosis can be initiated in the hopes to increase differential tendon gliding (►Fig. 9). Caution should be exercised, however, in the use of this technique during phase 1, particularly if the strength of the repair is questionable, and subsequently, resisted IP joint flexion is not warranted at any time during phase 1. Persistent adhesions in phase 2 of the rehabilitation, when the orthosis is first discontinued, warrant active digital flexion with the wrist joint positioned in variable degrees of extension to continue increasing differential motion. Additionally, PIP and DIP joint blocking exercises along with various tendon gliding exercises will enhance tendon motion. This will result in greater tendon motion and less tendon restriction permitting greater motion at both these joints. Persistence of restricting adhesions, limiting differential motion, is treated with a variable resistance during phase 3, by placing the wrist joint in an extended position, to minimize the work of flexion.²⁵ During phase 3, specific resisted exercises may also be used, such as resisted DIPJ flexion with the assistance of a dynamic orthosis (►Fig. 10).

Fig. 9 Isolated proximal interphalangeal and distal interphalangeal flexion exercises in orthosis to address early adhesions.

Proximal Interphalangeal Joint Flexion Contractures

A second common complication of zone II flexor tendon rehabilitation is the formation of PIP joint flexion contractures. Due to the difference in cross-sectional area of the flexors and extensors, the extensor system is deficient in terms of strength and excursion in relation to the flexors⁴⁸ and thus extensor deficiency is further magnified as a result of flexion contracture. Additionally, flexion contractures of the PIP joint may be a result of scarring of the volar plate, tendon bowstringing due to pulley disruption, skin contracture, and flexor tendon adhesion.⁴⁹ Biomechanically, flexion contracture at the PIP joint causes a volar translation of the lateral bands and the MCP joint demonstrates a stronger hyperextension tendency during extension, leading to an additional flexor force at the PIP joint.⁵⁰ Since proximal excursion of the extensor digitorum communis is unrestricted at the MCP joint, attenuation of the sagittal bands are likely to produce an additional complication. Furthermore, certain tendon orthotic intervention techniques, particularly those involving dynamic traction into flexion, are prone to producing flexion contractures at the PIP joint.^{9,34}

By preventing the development of a PIP joint flexion contracture during phase 1 of tendon rehabilitation, formation of these late secondary complications can be minimized. Pettengill & Van Strein^{8,9} outlined various methods proposed by several authors

Fig. 10 Resisted distal interphalangeal joint flexion.

Fig. 11 Resisted extension in orthosis.

in their attempt to regulate development of these flexion contractures early on in the orthotic intervention phase, including the use of straps and orthotic intervention material to maintain both the PIP and DIP joints in full extension, as first introduced by the Belfast regimen, in 1989^{16,27}. This will not only prevent contractures of soft tissue structures, such as volar plate and skin, but also allow repaired sites, in case both FDP and FDS have been cut in flexion, to rest at different location and not adhere to one another. Additionally, considering the inherent weak extensor tendon apparatus over the PIP joint, we recommend the initiation of resisted PIP extension as soon as flexion contracture is noted, preferably by positioning the MCP joint in flexion (► **Fig. 11**).

During phase 2 of tendon rehabilitation, if a flexion contracture is already evident, intermittent corrective orthotic intervention including passive extension of the PIP joint with the MCP joint in flexion to position the healing tendon in a protected manner, initially serially static and then dynamic or static progressive, depending on the density of the contracture. Additionally, an MCP joint dorsal blocking orthosis may be applied during functional activities to redirect extensor force during enhancing the excursion of the extensor tendons at the PIP joint.^{7-9,51}

Fig. 12 Lumbrical plus phenomenon may occur when tendons are adhered to the fibrous sheath. Upon flexing the digit, the flexors can only cause flexion at the metacarpophalangeal joint. Gliding of the lumbrical muscle during activation of flexor tendons is unrestricted thus proximal interphalangeal joint demonstrates strong extension tendency.

Lumbrical Plus

A third secondary complication that has been clinically observed following repair of a zone II injury is the “lumbrical plus” phenomenon. The lumbricals originate off the FDP tendon and act as both flexors of the MCP joint and extensors of the IP joints. This phenomenon can be caused by shortening of the FDP tendon at the time of repair.⁵² In this case, the lumbrical plus finger may be treated by means of tendon graft, and is, therefore, beyond the scope of this paper.

Lumbrical plus phenomena may also be caused by flexor tendons adhered to the fibrous sheath in zone II, in which differential tendon excursion is reduced. In these cases, patients may co-contract their lumbricals and interossei excessively as they attempt to make a fist, commonly observed in repaired tendons that have been immobilized or delayed in engaging in mobilization protocols.^{9,34} As the flexor tendon reaches its limit of available excursion, the intrinsics continue to contract resulting in MCP joint hyperflexion and IP joint extension, or “lumbrical plus” phenomenon (► **Fig. 12**). Passive flexion of the PIP joint with active hold produces a “spring back” extension at the IP joints.

Lumbrical plus phenomenon may also be associated with tightening of the lumbricals, due to prolonged positioning of these muscles in a more relaxed manner. As previously mentioned, early implementation of differential gliding of the FDS and FDP tendons is quite effective in maximizing excursion of the tendons and minimizing tendon adhesions, which will in turn minimize lumbrical plus phenomenon.⁵³ Additionally, in these cases, the modified SWM by Tanaka et al²⁵ will position the lumbricals in maximum stretch (MCP joint extension and IP joints flexion), addressing the aforesaid issue.

Special attention should be given to lumbrical plus phenomenon in the little finger. Our clinical observation demonstrates that little fingers are notoriously more prone to developing a lumbrical plus phenomena, particularly in the case of a deficient superficialis (► **Fig. 12**).

Lumbrical plus phenomenon is predominant in phase II, when the orthosis is discontinued. To treat this, PIP and DIP joint blocking exercises with MCP joint in a variable extension to flexion positions are recommended. An MCP extension

orthosis should be considered, if the finger does respond to the aforementioned plan. If lumbrical plus persists in phase 3, when resisted exercises are added to the home program, patients are encouraged to buddy tape affected finger to the adjacent one and use EMG biofeedback combined with electrical muscle stimulation.⁷

Other Considerations: Treatment Modalities in Tendon Rehabilitation

We have described various tendon mobilization techniques addressing zone II to reduce the pathomechanical changes caused by secondary complications associated primarily with tendon adhesions. In addition to these mobilization techniques, thermal modalities such as heat and ultrasound along with electrical stimulation are helpful in maximizing the treatment outcome. These modalities are beneficial in phase 2 and 3 of tendon rehabilitation program, but may also be used as early as phase 1, if warranted.⁵⁴

Discussion

The goal of surgical restoration and postoperative rehabilitation of flexor tendons is to regain ROM and strength that will allow normal use of the hand. To successfully rehabilitate tendon injuries, specific surgical and therapeutic considerations such as type of injury, suturing technique, and postoperative positioning and motion of the wrist and MCP and IP joints are necessary, particularly immediate postoperative positioning, which seems to play a major role in forming flexion contractures. Upon suturing of tendons, intraoperative evaluation of tension-free positioning of the joints to determine the permissible arc of motion and avoid gapping simplifies the therapeutic management. Despite of the advancement in surgical techniques and therapy, confidence of achieving normal motion is unpredictable.

Trauma and surgery jointly compounds formation of edema, reduction in joint motion, and adhesion formation, which subsequently affects normal gliding of flexor tendons in a fibrous sheath.^{35,38,40} As a result, the tendons have reduced differential excursion as compared with the uninjured tendons, thus affecting normal functioning of the kinematic chain of the hand. Certain physiological changes begin to occur as early as 0 to 3 days after repair and continue to become profound overtime if the treatment fails to address these changes.³⁶ While secondary mechanical changes caused by loss of differential tendon motion may not become evident until orthotic intervention is discontinued in phase 2, the hand therapist should identify these complications during the orthotic intervention phase through weekly monitoring of active and passive ROM. If ROM is decreased or there is a significant discrepancy in active and passive ROM, the therapist should modify the intervention regimen and consider the recommendations aforementioned to prevent permanent disability. Ultimately, severe forms of these complications require surgical intervention, if functioning of the hand is greatly affected.

Tendon excursion studies in cadavers have shown that as much as 9 cm arc of motion is required for a combined flexion

of wrist and fingers, whereas just 2.5 cm of tendon excursion produces full digital flexion with the wrist in neutral.^{55–58} An *in vivo* study by Edsfieldt and colleagues⁵⁹ analyzing forces generated during finger exercises corroborated with these studies demonstrating that “place and hold” and active finger flexion with the wrist in the neutral position or minimal tenodesis extension produced the lowest forces. Additionally, *in vivo* studies have revealed that active movements generate 1.0 to 2.0 mm per 10-degree of arc of motion, whereas passive exercises generated only 0.4 to 0.9 mm per 10-degree, likely associated with tendon buckling.^{60–62} Sapienza and colleagues⁶³ validated these results in a recent cadaveric study analyzing tendon excursion and load during passive and active simulated motion. They demonstrated that active motion generated greater tendon excursions than passive motion, while hook flexion generated the greatest total tendon excursion and differential gliding without placing excessive loads on tendons.⁶³ Based on these studies, it is obvious that to move the digits maximally, in relation to the position of the wrist, a significant amount of excursion of the flexor tendons must be available. If there is a progressive loss of tendon excursion then there will be a substantial loss of joint motion. Hence, the goal of therapy must be to maximize differential tendon excursion, while protecting the repair site.

Therefore, combining SWM and active mid-range digit flexion, as previously suggested, has added benefit on the differential tendon excursion with minimal load on the healing tendons, corroborated by various tendon biomechanical studies.^{3,4,25,35,38,59,62} Postsurgical resistance to the gliding of the tendons in a fibrous sheath dictates that mobilization of these tendons be advanced by using gradual increments of wrist synergistic motion and active finger flexion in the phase 1 and combination of wrist and digital flexion, wrist and digital extension progressively in phases 2 and 3, depending on the of tendon suturing technique. This method of mobilization enhances differential tendon motion in zone II and will hopefully prevent permanent loss of ROM due to adhesion formation.

Conclusion

Despite advances in surgical and therapeutic management, restoration of flexor tendon function remains challenging. The density of adhesion formation is more profound when there is inadequate differential tendon motion between FDS and FDP during initial healing phases. There have been minimal publications addressing different complications in zone II injuries and treatment considerations or modifications that need to be implemented to maximize tendon excursion. Tendon adhesions in zones II have shown to produce various pathomechanical changes, leading to a kinematic chain imbalance. Based on the mechanical and physiological factors, special attention should be given to the various early presentations of adhesions in the subzones of zone II, flexion contractures and lumbrical tightness to properly modify classical protocols, and prevent permanent changes. Therefore, a well-structured rehabilitation program using a combination of early controlled active mobilization and SWM

will potentially maximize differential tendon excursion between FDS and FDP, minimize complications, and maximize hand function.

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