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# The A1 Pulley as a Fulcrum for Flexor Tendon Excursion: a Histopathological Study

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**SUMMARY**

**Purpose.** Stenosing tenosynovitis of the flexor tendons which readily causes the alleged "trigger finger" is a common and yet not completely understood phenomenon. The A1 Pulley is the pivot point at which the force protracted by the flexors of the digits acts upon the fulcrum at the leading edge of the pulley. We performed a histopathological study aimed at assessing the A1 Pulley of digits 2 through 5 in cadaver hands in an effort to delineate the changes the A1 pulley undergoes through the cycle of life and its repetitive motion tasks.

**Methods.** Thirty-Nine A1 Pulley specimens were included in the study. These specimens were assessed as to physical width utilizing a caliper and as to histological characteristics following embedment and pertinent staining.

**Results.** Our findings suggest the proximal leading edge of the A1 Pulley to be significantly ($p < 0.05$) thicker than the more distal aspect of the A1 pulley when pulley data were stratified for handedness and gender. We found insignificant differences within each digit as to differences between the radial and ulnar aspects of the denoted numerated digit.

**Conclusions.** We believe our study sheds further light on the mechanism leading to digital triggering and that further studies may substantiate our preliminary findings as to the differences in radial *versus* ulnar aspects of each designated digital A1 pulley.

**KEY WORDS**

*A1 pulley; excursion; flexor tendon; fulcrum; histopathological.*

## INTRODUCTION

Stenosing tenosynovitis of the adult is a well documented pathology of the flexor tendons[1]. This condition leads to an inability to extend the flexed digit ("triggering"), which is usually seen in individuals older than 45 years of age (1).

When associated with a collagen disease, several fingers may be involved (most often the long and ring fingers). Patients may note a lump or knot in the palm. The lump may be the thickened area in the first annular part of the flexor sheath or a nodule or fusiform swelling of the flexor tendon just distal to it. The nodule can be palpated by the examiner's fingertip and moves with the tendon. Usually, the tendon nodule is just proximal to the A1 Pulley at the metacarpophalangeal joint level (2, 3).

The normal A1 pulley is composed of 3 layers (2): layer I, an inner, avascular, concave unicellular or bicellular gliding layer containing cartilage-like cells; layer II, a middle layer, also avascular, characterized by spindle-shaped fibroblasts; and layer III, an outer, richly vascularized layer, continuous with the membranous tendinous sheath. A classification describing the severity of histologic abnormalities observed in trigger digit A1 pulleys is described in the literature (3). Mild abnormalities (grade 1) were those with a fibrocartilaginous gliding surface almost intact. The margin between the

fibrocartilaginous and membranous portions of the pulley was well delineated. In moderate abnormalities (grade 2), the avascular fibrocartilaginous gliding surface appeared fissured and thinner. The inner layer (I) was interrupted and replaced by fibrous tissue, with fissures that did not cross through the middle layer (II). A mild vascular network hyperplasia was observed in the outer layer (III), which began to invade the fibrocartilage. In severe abnormalities (grade 3), the fibrocartilaginous gliding surface was thin, discontinuous, or even completely destroyed. The vascular network hyperplasia became excessive and reached the synovial space of the flexor tendon sheath. The histologic features were documented by light, transmission and scanning microscope (3) and were correlated with the severity of the clinical symptoms.

The anatomic course of the 2nd to 5th digit flexor tendon is different for each of these digits. When one relates the flexor origin to the placement of the A1 Pulley of each finger the forces acting on the brink of the pulleys are not symmetrical. Hence, the ulnar pull on the leading proximal edge of the index finger A1 Pulley is most probably greater than that on the radial side of the leading edge of the pulley due to the angle at which the pulling flexor approaches the index finger. In contrast, at the leading edge of the A1 pulley of the 5th digit it may be assumed that greater forces act on the radial side of the pulley rather than on the ulnar side - a force further enhanced by ulnar deviation of the wrist.

Thus, since the human hand has a multitude of functions, force created by a strong grasp is exerted by the flexor tendons when performing a powerful grip. This concept may allow understanding of the processes causing eventual triggering by the mechanism of force transfer to the leading edge of the A1 pulley. In this instance, it may be postulated that these forces are not necessarily equally dispersed along the leading edge of the A1 pulley.

Our study was aimed at assessing the anatomical course of the flexor tendons from their origin to the A1 pulley specifically and more so to the leading edge of the pulleys. Within this study and following the anatomical assessment of the angles at which the flexor tendons approach their respective A1 Pulleys we aimed at assessing whether the leading edge of the A1 Pulley of each digit undergoes changes in the radio-ulnar dimensions compensating for excessive transmission of force in a radio-ulnar pattern as well as discerning whether a significant difference is found between the leading and more distal aligning parts of the A1 Pulley.

## MATERIALS AND METHODS

This study was approved by the Kaplan Medical Center Ethics Committee.

Dissection of fourteen cadaver-forearms was performed at the Cadaver Lab of the Technion Medical School in Haifa, Israel. The cadavers were of elderly persons (all specimens were harvested from cadavers over the age of 70), embalmed in formaldehyde. Meticulous dissection of the forearm and hand was performed in each of the upper extremities.

Of 56 available specimens, 39 were found to be of acceptable quality and hence included in the study. The excluded samples were either dry, damaged, did not contain both pulley bony insertions, or anatomic borders of the pulley could not be discerned due to the formaldehyde preparation. A digital caliper (Mitutoyo Inc., Kanagawa, Japan) was used to measure each of the 6 zones of the A1 pulley (**figure 1**) to estimate whether there are differences in tissue thickness. Three measurements were conducted to each of the 6 zones and an average of these measures was used as a final value. Histological examination of the proximal and distal cross-sections of 14 pulleys was performed in order to compare thickness and histological differences from each aspect (Radial/Ulnar, proximal/distal) of the A1 pulley. Two pulley samples were obtained for comparative measurement of thickness using a light microscope. Similar values were measured with the light microscope as compared to the caliper measurements in these samples.

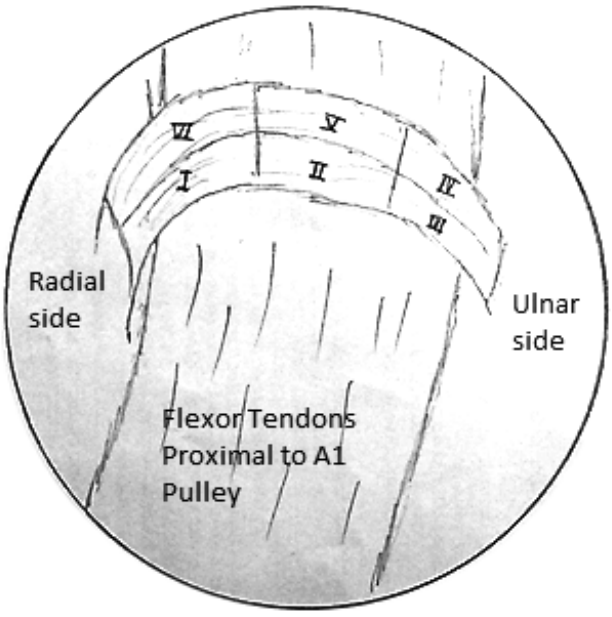


**Figure 1.** Schematic depiction of the flexor tendons entering the A1 pulley.

Division to zones of the pulley: I proximal-radial; II proximal-central; III proximal ulnar; IV distal-ulnar; V distal-central; VI distal-radial.

## RESULTS

Thirty-Nine A1 Pulley specimens were included in the study, and each was divided into six zones (**figure 1**). Of the six zones of every pulley, the central distal zone was the thinnest in 82% (32/39) of the A1 pullies. The average A1 pulley annular width of the 2nd to 5th digits (with standard deviation) was 293 ± 142 µm, 311 ± 149 µm, 300 ± 151 µm, 216 ± 95 µm, respectively.

We found that the stratified values of the three proximal zones of each pulley showed a significant ($p = 0.0129$) difference when compared to the three distal zones in each individual pulley. Statistical analysis using the ANOVA test was performed. When all values were pooled together the P-value was calculated to be $p = 0.0549$. When calculated differences were compared within each pulley (stratified data) the P-value obtained was $p = 0.0129$, and a significant difference was found between the proximal leading edge and distal aligning edge of the pulley.

A consistent thickness difference between the proximal radial and ulnar sides of the pulley as compared to the distal-central part with a value of 25 ± 14%, was found as well. We did not find a consistent enlargement of either the radial or ulnar side.

Histological cross sections of proximal and distal portions of the A1 pulley demonstrated consistent characteristics. Asymmetrical radial or ulnar thickening was observed in all pulley specimens, it is mostly represented by widening of the middle layer of avascular, spindle-shaped fibroblasts, and in addition, the thickest zone of the pulley had the most developed inner, avascular, concave unicellular or bicellular gliding layer containing cartilage-like cells as can be seen in **figures 2** and **3**.

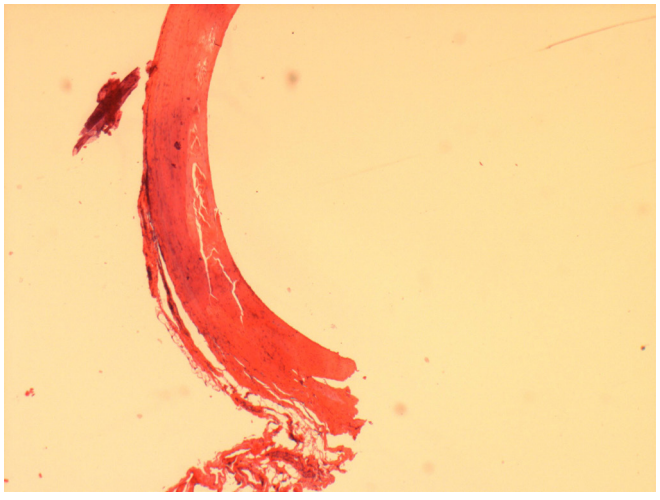

**Figure 2.** x20 magnification of 3rd digit proximal-ulnar zone of A1 pulley. The ulnar side is thicker and inner cartilage-like cells layer is well developed.

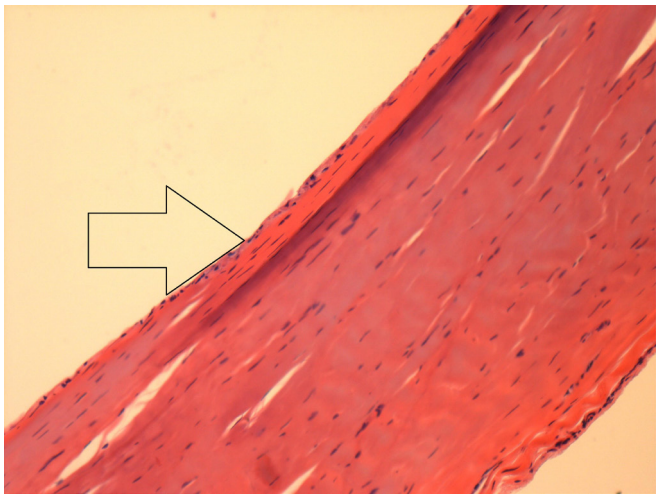

**Figure 3.** x100 magnification of 4th digit distal-radial zone of A1 pulley. The cartilage-like cell layer can be seen (arrow).

## DISCUSSION

Stenosing tenosynovitis of the flexor tendons which readily causes the alleged "trigger finger" is a common and yet not completely understood phenomenon (1-3). The A1 Pulley is the pivot point at which the force protracted by the flexors of the digits acts upon the fulcrum at the leading edge of the pulley. Our findings show that the proximal leading edge of the A1 Pulley is significantly ($p = 0.0129$) thicker than that of the distal aligning part of the A1 pulley of flexors of the 2nd to 5th digits. We believe this finding corroborates our initial conception that flexor forces acting on the A1 Pulley leading edge may explain histological changes within the pulley itself (**figures 2, 3**) as well as possible subtle changes in tendon histology which were not subject to assessment within the scope of this study.

This study was performed on cadavers of elderly people (all cadavers were above seventy years of age) and hence we believe this affected our findings. In elderly persons, the anatomical structures have undergone changes due to the natural process of aging. Changes that are otherwise not apparent in younger less challenged individuals, or those in which the youth of their tissue make them resilient to the forces acting upon them may not be noticed. The age of the subjects from which these samples were taken from may be both a drawback and advantage for this study. It could be stipulated that in an elderly patient, changes may be evident at the leading edge of the pulley as a mere age-related finding, or one may conjure that changes which are evident in this age group may be present in a subtler representation in the younger age group. We believe the latter to be true and hence view our use of elderly specimens as a

key to the findings of this study. Although ultrasound failed to show such changes in the A1 Pulley of normal digits (2), non-contracted trigger digits did show flexor tendon thickening just proximal to the A1 pulley leading edge, strengthening our claim that this specific area may be the culprit of the triggering. Contracted digits did show the tendons to have areas rendered as "Notta nodes" but since this finding was noted as significant only in the contracted digit setting, we believe it to be a local tissue reaction within the flexor tendons and their sheath to the force exerted in vivo and continuously at a designated area of the involved flexor tendons.

The finding within our study defining the leading edge of the A1 pulley as the possible perpetrator of triggering may be combined with those of other studies (2) in trying to delineate the optimal site for both steroid injections and possibly percutaneous trigger finger release. It remains to be discerned whether in non-contracted, minor degree triggering partial accurate percutaneous pulley resection may suffice (*i.e.*, releasing only the leading edge of the A1 Pulley). Tung *et al.* (3) failed to show a correlation between the compliance of the A1 Pulley when measured by passing a metal rod through the pulley to the relative frequency of triggering in each individual digit. In conjunction with our findings, we believe this failure may be attributed to the fact that the leading edge of each pulley was not assessed but rather the pulley in its entirety.

Our macroscopic findings were corroborated with the microscopic findings of a thicker layer of connective tissue at the leading edge of the pulley (**figure 2**). Our findings could be further corroborated by implementing a Transmission Electron Microscopy study implemented to assess the differences in tissue characteristics in defined areas of the A1 Pulleys. Sbernadori *et al.* (4) found that while in normal pulleys "the whole deep surface was covered uniformly by an amorphous extracellular matrix", in samples taken from trigger digits "there was the same general surface appearance but, also, areas, varying in shape and dimension where loss of the extracellular matrix had exposed the collagen fibers and a few cells of the middle layer of the pulley. There were also changes typical of "chondroid-metaplasia". A further study utilizing Transmission Electron Microscopy may be in place dividing the A1 Pulley surface into designated areas and quantifying the surface changes in each area. Hence, our study was limited by the quality of histologic assessment and by the use of embalmed cadavers.

While most hand surgeons would advocate the time-honored practice of complete A1 Pulley release, this may not necessarily be called for. Hence, the search for an ultimate complete percutaneous release technique (5-8) may be redundant of any true clinical relevance.

## CONCLUSIONS

Our findings may offer a partial and incomplete explanation for the occurrence of trigger finger. Further studies implementing advanced technology and possibly an in vivo model are in place so that our findings are further corroborated.

## FUNDINGS

None.

## DATA AVAILABILITY

Upon request to Corresponding author.

## CONTRIBUTIONS

EB, AO: design of the study. AM, SI, AO: dissections and pathology. AM, EB, MV, AO: interpretation of the results and writings.

## CONFLICT OF INTERESTS

The authors declare that they have no conflict of interests.

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