

# Diagnosis of the A3 pulley injury using ultrasound

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## Abstract

The pulley rupture is the most common injury in sport climbing. The correct diagnosis of A3 pulley ruptures represents a challenge. We therefore investigated a new approach to this pathology.

Eighteen cadaver fingers were examined using high resolution, dynamic ultrasound before and after inflicting different combinations of singular and multiple pulley ruptures. The behaviour of the volar plate (VP) with respect to the proximal interphalangeal joint (PIP) and the flexor tendons before and after pulley rupture were studied.

A direct visualization of the A3 pulley was only achieved in 61% of the fingers. The VP became significantly thicker and shorter with a flexing of the finger as well as after A3 pulley rupture. The distance between tendon and VP became significantly more pronounced after A3 pulley rupture.

The distance measurement between VP and tendon was found to be a valid indirect method for A3 pulley rupture diagnosis.

## Résumé

La rupture de la poulie est la lésion la plus fréquente en escalade sportive et c'est un véritable défi d'établir le bon diagnostic sur les ruptures de la poulie A3. C'est pourquoi nous avons envisagé une nouvelle approche de cette pathologie.

Dix-huit doigts prélevés sur des cadavres ont été examinés à haute résolution, avec ultra-sons dynamiques avant et après avoir effectué plusieurs possibilités de rupture de poulie, unique ou multiples. On a alors étudié le comportement de la plaque palmaire (VP) en respectant l'articulation interphalangienne proximale (PIP) et les tendons fléchisseurs avant et après la rupture de la poulie.

Il a alors été possible de visualiser correctement et directement la poulie A3 sur 61% des doigts. La plaque palmaire était plus épaisse et plus courte en fléchissant le doigt, tout comme après une rupture de la poulie A3. La distance entre le tendon et la plaque palmaire était beaucoup plus importante après une rupture de la poulie A3.

La mesure de la distance entre la plaque palmaire et le tendon constitue une méthode indirecte, mais valide, afin d'établir un diagnostic de rupture de poulie A3.

## Introduction

The A2 and A4 pulley are fibro-osseous pulleys that originate on the proximal and medial phalanx respectively (Doyle, 1988), while the A3 pulley originates on the volar plate (VP) of the proximal interphalangeal joint (PIP joint), allowing the A3 pulley to move more freely away from the phalanges during finger flexion (Bayer, Schweizer, Muller-Gerbl, & Bongartz, 2012; Doyle, 1988; Watanabe, Hashizume, Inoue, & Ogura, 1994; Williams, McCarthy, & Bickel, 1998). Following pulley rupture, finger flexion generally features motion of the tendon away from the bone in the so-called “bowstring phenomenon” (Marco, Sharkey, Smith, & Zissimos, 1998; Peterson & Bancroft, 2006; Walbeehm & McGrouther, 1995). However, the contrasting origin of the A3 pulley results in its comparatively minor influence on any bowstringing motion of the tendon during finger flexion. Consequently, diagnosis of A3 pulley ruptures based on the degree of corresponding tendon bowstringing is nearly impossible. Precise knowledge of the extent of the injury is paramount to selection of the correct treatment for pulley ruptures (Schöffl, Hochholzer, Winkelmann, & Strecker, 2004).

Previously, it was believed that observation of a more pronounced bowstring phenomenon was indication of the rupture of several pulleys, especially involving the A3 (complex pulley rupture), but the threshold for distinguishing is not consistent in the literature (Bodner et al., 1999; Hauger et al., 2000; Klauser et al., 2002; Martinoli, Bianchi, Nebiolo, Derchi, & Garcia, 2000). In order to address the challenge of diagnosing A3 pulley ruptures, a novel indirect approach via investigation of the volar plate was proposed. The purposes of this study were to investigate the motion of the volar plate with respect to diverse combinations of pulley ruptures and with special interest in the role of the A3 pulley.

## Material and Methods

In this study, the second and third digits from nine left hands from nine organ donors were investigated, for a total of 18 finger specimens. All organ donors were female. All hands had been frozen at  $-5^{\circ}\text{C}$  without further treatment, and less than two days after death. All donors and their families provided informed consent regarding the use of the specimens in a scientific study. This study was presented to the ethics committee of the Friedrich Alexander University Erlangen-Nuremberg, and approval was received prior to its initiation. The mean age of the donors was 73.2 years. The hands were severed in a frozen state at the middle of the radius and then thawed for a duration of four hours. All measurements were completed using a GE logic E9® (GE Healthcare®, Buckinghamshire, Great-Britain) with a linear 18 MHz probe. The Cross-Beam settings were set to “low,” and the Speckle-Reduction was left at setting “2”. For standardized measurements, each finger was attached to a fixateur externe mounted on a wooden plate using two Mini-Schanz screws in the proximal phalanx. The fingertip was then attached to a deflection pulley using a cable tie, enabling the researcher to move or fix the finger during the examination. The flexor tendons were stitched to themselves to create a loop through which metal hooks were pulled. The tendons were then subjected to a 10 N force (Schöffl, Hugel, Schöffl, Rascher, & Jungert, 2016) and then examined using ultrasound. The tendon-bone (TB) distances over the A2 pulley and A4 pulley were measured at the middle of their respective phalanges. The length of the VP was measured from its most proximal to its most distal point, and the width was measured at its broadest point. We defined a point on the proximal phalanx where the curvature of the condyle transitions from concave to convex. We then measured the distance between this point and the middle of the volar aspect of the VP (VP-bone). Then we determined the distance between the VP and the tendon as an extension of the VP-Bone measurement onto the tendon (VP-tendon). All fingers were investigated in full extension and at  $30^{\circ}$  flexion. After that, the fingers were removed from the measuring device in order to produce artificial pulley ruptures: six times the A2, four times the A4 and

eight times the A3 pulley. Then a second pulley was artificially ruptured: seven A2/A3 pulley ruptures, six A3/A4 pulley ruptures, and five A2/A4 pulley ruptures. Then the third pulley was cut, leading to eighteen A2/A3/A4 pulley ruptures for the final ultrasound measurements.

## Results

A cut-off of 2 mm TB distance over the A2 pulley corresponded to diagnostic sensitivity and specificity measures of 94% and 100% respectively for A2 pulley ruptures, and a cut-off of 2 mm TB distance measured over the A4 pulley corresponded to sensitivity and specificity measures of 90% and 97% respectively for A4 pulley ruptures.

TB distance over the A2 and A4 pulley were found to be independent of A3 pulley rupture based on comparison between complex A2/A3, A3/A4 pulley ruptures (2.6 mm) and single A2, and A4 pulley ruptures (2.4 mm) respectively. Following rupture of all three pulleys, TB distances were observed to increase to a maximum. After single A3 pulley rupture, the VP became significantly longer and thinner. Furthermore, the VP tendon distance increased to a significant degree, while the decrease observed in VP bone distance was not significant. When analysing the length and thickness of the VP before and after a singular A2 and A4 pulley rupture respectively, there was no significant difference. The same is true for the VP bone distance and the VP tendon distance.

Regarding complex pulley ruptures, a significant difference in VP tendon distance between ruptured and intact pulley systems was only observed in those complex pulley ruptures featuring an A3 pulley rupture. Following complex pulley rupture of all three pulleys (A2, A3 and A4), visualization of the VP became extremely difficult due to interference from air trapped between tendon and VP.

## Discussion

In clinical practice, the correct diagnosis of A3 pulley injuries has proven to be challenging. Due to the influence of accurate diagnosis on both prognosis and treatment, this investigation aimed to fill the diagnostic gap through determination of an effective, accessible approach for A3 pulley rupture diagnosis.

For A2 and A4 pulley ruptures, the established method of diagnosis revolves around an indirect measurement of the TB distance over the pulley of interest. In the past, researchers have proposed tailoring this method for diagnosis of additional A3 pulley ruptures when observing a further increase in TB distance (Klauser et al., 2002; Martinoli et al., 2000). Our investigations of single and complex pulley ruptures involving the A3 pulley determined that they have no influence on TB distance over either the A2 or A4 pulley in any instance.

Saito et al. (Saito & Suzuki, 2011) described in their study that the movement of the VP depended on the A3 pulley. In an MRI study performed by Bayer et al. (Bayer et al.), a general reduction of VP translation distance and augmentation of VP tendon distance were established as suitable indirect signs of A3 pulley rupture.

Following A3 pulley rupture, we observed the VP to alter in shape, returning to a longer and thinner state as a consequence of lacking the upward pull from the fibres of the A3 pulley. In our study, we observed an increase in the VP tendon distance from 0.5 mm for an intact A3, to 0.9 mm after pulley rupture. For instances in which pulleys in addition to the A3 pulley were ruptured, this VP tendon distance increased further. Using a cut-off of 0.9 mm, we were able to predict the A3 pulley rupture with a sensitivity of 76% and a specificity of 94%.

Although we observed the length and width of the VP to change significantly after pulley rupture as described in the literature (Bayer et al.; Saito & Suzuki, 2011), we believe that this is an impractical marker for determining an A3 pulley rupture, as this requires comparison to pre-rupture data for the given specimen – which, usually, is not available.

Even though the results of this study were conclusive, it needs to be stressed that there were several limitations. First of all, the findings of a cadaver study cannot be directly translated to *in vivo* settings. Secondly, because of the limited availability of cadaver specimens, we only investigated 18 fingers, and all were female. Thirdly, air entrapment between tendon and bone occurred, obscuring the images produced through ultrasound.

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