

Stress-shielding as a cause of insertional tendinopathy: the operative technique of limited adductor tenotomy supports this theory

JW Orchard¹, JL Cook² & N Halpin³

¹Injury Risk Management Research Centre, University of New South Wales, Australia.

²Musculoskeletal Research Centre, La Trobe University, Victoria, Australia. ³Sports Physician, Parramatta, NSW.

The aetiology of tendinopathy is poorly understood. A new hypothesis proposed argues that tendinopathy may not be purely a tensile injury, rather that altered mechanics such as compression or stress-shielding may be important. Both tendon compression and a decrease in tendon load (stress-shielding) will induce change in a tendon similar to that seen in an insertional tendinopathy.

Stress-shielding as a cause of tendinopathy is supported by the clinical success of operative release of adductor longus. This surgery releases the superficial section of the normal adductor longus tendon at a point distal to the insertion. This may have the effect of transferring stress from the superficial section of the tendon to the stress-shielded deeper portion, and the induction of normal loads in both the deeper and superficial portions of the tendon may assist in tendon recovery. This interesting hypothesis and clinical intervention require further investigation

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Introduction

There has been much progression in our understanding of tendinopathy in the past 10 years, although it is fair to say that there has been more conclusive disproving of old myths compared to positive improvements of knowledge. This is exhibited by comparing feature editions on the subject in the review journal *Clinics in Sport Medicine*. The very titles of these editions are a prime example. In July and October 1992 there were two editions entitled Tendinitis I & II, whereas in 2003 there was a single edition entitled Tendinopathy. The title Tendinitis was a consequence of the mistaken belief in the past that tendinopathy was primarily an inflammatory disorder. The new title of Tendinopathy is a more broad term but denotes, correctly, that we do not fully understand the aetiology.

Comparing the editions of the *Clinics* from a decade apart, one short viewpoint paper stands out in the recent edition as being a completely novel concept, entitled "Compression etiology in tendinopathy"¹. This paper discusses the phenomenon of insertional tendinopathy, which commonly affects the supraspinatus tendon, common extensor origin at the elbow, adductor

longus tendon, patellar tendon and Achilles tendon. Surgical, histological and radiological observation is that the pathological part of the tendon insertion is the deep (joint) side of the tendon, with the superficial portion of the tendon insertion generally normal. There may also be abnormalities on the bony side of the enthesis (Figure 1). In patellar tendinopathy, which is a prototype for the insertional tendinopathy, Almekinders et al have shown, using strain gauges in cadaver tendons, that the superficial portion of the tendon insertion is consistently under greater strain levels than the deep portion². This is logical, as the superficial portion of the tendon is always farther away from the central axis of rotation of the joint than the deep portion.

We agree with Almekinders et al that these findings challenge the concept that insertional tendinopathy is a simple repetitive strain injury resulting from tensile forces, in which case it should affect the superficial portion of the tendon. Almekinders et al propose two alternate theories to explain their observations: that there is either a 'compression' or 'stress-shielding' aetiology for insertional tendinopathy^{1,2}. These are logical alternate explanations. The compression theory suggests that the deep surface of the tendon is damaged by compressive rather than tensile forces. The stress-shielding theory considers tendinopathy to be a combined overuse-underuse injury, where the superficial portion of the tendon bears too much of the tensile load, whilst the deep portion of the tendon bears too little of the same load. A low load may induce changes in tendon that are similar to that seen in tendinopathy and in compressed tendon.

Much has also been made of the inconsistent results of surgery for patellar tendinopathy³. Surgical technique varies substantially for operations performed on the patellar tendon, and most of the other insertional tendinopathies. We would like to draw attention to the surgical technique for adductor tendinopathy (Figures 2 and 3), which involves a limited adductor tenotomy^{4,6}.

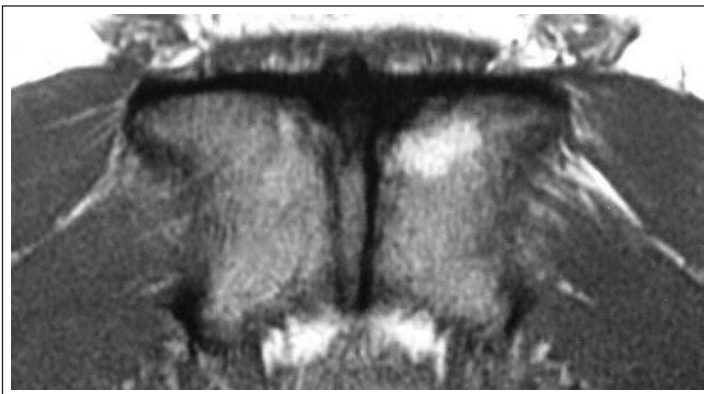


Figure 1: Axial T2 MRI scan (from below) showing tendon changes primarily in the deep surface of the left adductor longus tendon attachment, with additional changes in the adductor brevis tendon attachment and left pubic bone marrow oedema.



Figure 2: Left adductor longus tendon seen at surgery, prior to tenotomy (pectineus and gracilis muscle fibres can be seen slightly superficial to the tendon on either side).



Figure 3: Left adductor longus tendon (retracted proximally) after partial tenotomy. Deep muscle fibres of adductor longus (not brevis) can now be seen after the tendon has been cut (refer to figure 5).

Adductor tendinopathy and patellar tendinopathy are both typically insertional tendinopathies, although a major difference is that generally in patellar tendon the pathological changes are greater on the tendon side than the bone side of the insertion, with the reverse occurring with respect to the adductor longus tendon. The adductor longus is also fortunate to have multiple nearby agonists, particularly adductor brevis which lies immediately deep to adductor longus (Figure 4).

There are some important (and perhaps surprising) points that should be made about the operative technique of adductor tenotomy. The procedure involves a partial tenotomy which is only performed on the superficial fibres of the adductor longus tendon, those under larger tensile load in the proposed model⁵. The tenotomy is done at a level below the inguinal skin fold, a distance of 2-4cm from the tendon insertion⁴⁻⁶. The adductor longus tendon is much longer on the superficial side than the deep side⁷. When the surgeon makes

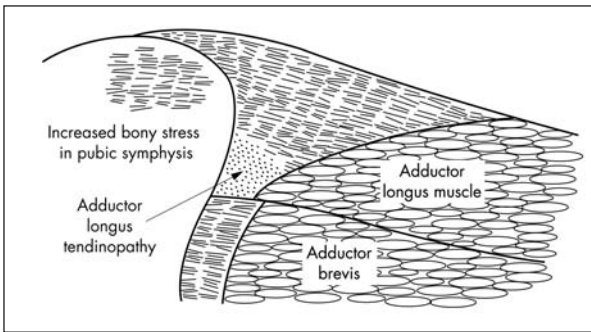


Figure 4: Adductor tendinopathy (oblique sagittal section), noting adductor longus tendon (superficial) lying over adductor brevis tendon (deep). Both tendons attach to the pubic bone, which may also have pathological changes. Note that the adductor longus tendon is much longer on its superficial surface than its deep surface. If there are degenerate areas within the tendon, the deep surface of the attachment to the bone is affected, as in other insertional tendinopathies.

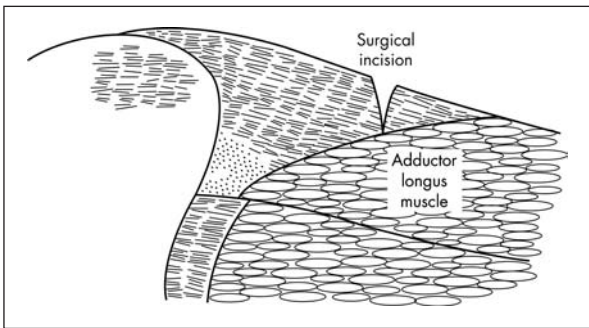


Figure 5: Adductor release surgery (oblique sagittal section). The incision is made 2-4cm from the bony insertion, at a level where a cut perpendicular to the tendon produces a tenotomy of the superficial tendon fibres only. The adductor longus muscle is not cut. The operation is performed away from the bone-tendon junction, which is the region of pathology.

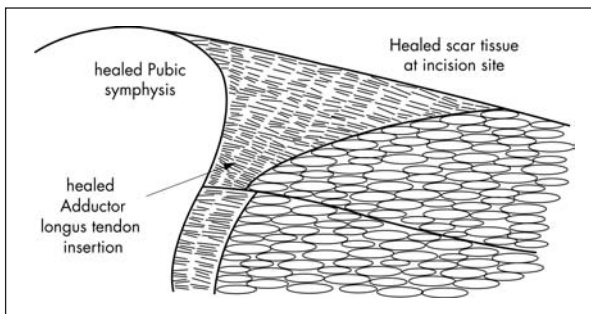


Figure 6: Possible outcome after adductor release surgery (oblique sagittal section). The superficial fibres of adductor longus have repaired under less tension, as the body has good ability to lay down new collagen fibres and form tendon-like scar. However, whilst this process has been occurring, perhaps the deep fibres of the tendon have also been able to repair, because they are no longer stress-shielded.

what appears to be a complete tendon division at a level distal to the insertion, it is actually only the superficial fibres of the adductor longus that are being divided, as there is adductor longus muscle deep to the tendon at this level⁵⁻⁷ (see Figures 3 and 5).

The success of adductor tenotomy is, like many operations in sports medicine, only based on clinical case series studies rather than randomised control trials⁴⁻⁶. However, the clinical success of an operation, which is clearly being performed on an area of normal tissue rather than an area of pathological tissue, suggests that this operative technique somehow advantageously affects the mechanics of a disordered tendon. By contrast, most other surgical techniques for other insertional tendinopathies, despite their variation in techniques, directly involve removing or attempting to repair a damaged area of tendon, sometimes arthroscopically from the underside of the tendon.

We propose that a possible effect of limited adductor tenotomy is that it transfers tensile stress from the strong superficial portion of the adductor longus tendon to the deep portion of the tendon (see Figures 4-6). It is possible that in adductor tendinopathy the deep portion of the tendon is stress-shielded and the technique described above corrects this mechanical disadvantage of the deep portion of the tendon. If this operation is shown by more rigorous scientific trials to be successful, it will add weight to the stress-shielding theory of tendinopathy as proposed by Almekinders and others.

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