SHOCK WAVE
THERAPY IN
PRACTICE

ENTHESIOPATHIES II

RADIAL SHOCK WAVE TREATMENT OF TENDINOPATHIES

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LEVEL10

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INTRODUCTION

/ Ulrich Dreisilker

The great response from medical colleagues and physical therapists to the first publication on the subject of ESWT for enthesiopathies / tendinopathies ("Enthesiopathies," Level10 Verlag, October 2010) was the motivation for this second volume. This book is intended to be helpful and stimulating for all physicians, physical therapists and sports scientists interested in shock wave therapy for tendinopathies.

Further development of ESWT.

Extracorporeal shock wave therapy (ESWT) is constantly being developed further: numerous scientific studies have shown how successful radial shock wave therapy (RSWT) is for tendinopathies and that it achieves even better results when accompanied by stretching / load-bearing exercises.[30-35] Shock wave cross-friction for tendinopathies enables low energy dosing, which is sufficient to stimulate tendon fibre proliferation and collagen synthesis. Doppler sonography (PDI) for imaging pathological capillaries (neovascularisation) is another milestone in the diagnosis and therapy monitoring of tendinopathies.

It is well known that muscle and tendon form a functional element together. Understanding tendon pathologies requires knowledge of muscle pathophysiology, on which M. Gleitz has reported extensively.[1] This volume makes multiple references to Gleitz' work as well as to the contribution from K. Karanikas ("Mechanical influence on the muscle-tendon complex," p. 45).

Tendon insertion disorders are widespread.

Tendon insertion disorders are not only a problem of older patients and degenerative changes from overexertions in daily life. They also occur as the result of improper training plans and the over- or under-training of athletes. While treatment and sports medical advice have fortunately improved for high-performance and elite sports in recent years, there is a great need to catch up in the care of recreational athletes and amateurs.

The myofibrils themselves are formed from the contractile filaments actin (thin) and myosin (thick) (fig. 4). The thick filaments contain the myosin, which consists of a long shaft with two ball-like heads on the end.

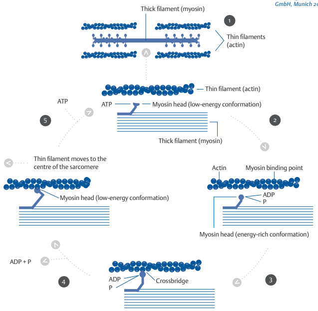
Additional proteins, such as troponin and tropomyosin, are deposited in the furrows between the two actin strands and function as control proteins. In the non-contracted state of the muscle fibre, the heads of the myosin filaments stand perpendicular to the actin filaments.

These heads bind to the actin filaments upon nerve signals, split the fuel ATP and draw the actin filaments together. In this phase, chemical energy is converted into mechanical energy. After this action, the heads of the myosin detach from the actin filaments again and swing back to their initial position. ATP is consumed in this process.

Externally visible muscle shortening occurs. This mechanism is known as the cross-bridge cycle and is implemented by the neural excitation of muscle fibres with the help of the transmitter acetylcholine.

HYPOTHESIS ON FORCE GENERATION DURING MUSCLE CONTRACTION | *Fig. 4*

Campbell, Neil A. & Reece, Jane B.: Biologie, Pearson Deutschland GmbH, Munich 2009, p. 1486.



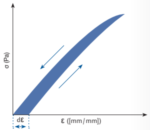
1 > The myosin head with bound ATP is in the low-energy conformation.
2 > The myosin head hydrolyses ATP to ADP and inorganic phosphate (P) and is then in an energy-rich conformation.
3 > The myosin head binds to actin and forms a cross-bridge.
4 > Through the release of ADP and P, the myosin head tips into the low-energy conformation and draws the thin filament in the direction of the centre of the sarcomere.
5 > The binding of another ATP molecule leads to the release of the myosin head. This can then assume the energy-rich conformation again, and a new cycle can begin.

THE SERIAL (SE) AND PARALLEL ELASTIC ELEMENTS (PE) – CONNECTIVE TISSUE

Serial (SE) and parallel elastic elements (PE) have many common structural properties. The distinguishing features essentially concern the elasticity, plasticity, viscosity and especially the stiffness of the individual structures. The tendons, for example, exhibit great stiffness and good elasticity. Tendons are able to convert stored energy into kinetic energy during an elongation. Fasciae have less stiffness and greater plasticity, so that a greater hysteresis loop remains after an elongation. This is comparable to the structure of a plastic bag, which does not return to its original form after being stretched and keeps the deformation (fig. 5).

Modified from Brinckmann et al. 2012 p. 39 Orthopaedic Biomechanics MV-Verlag.

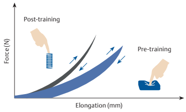
TENSION-ELONGATION DIAGRAM OF CONNECTIVE TISSUE | *Fig. 5 a*



The tension-elongation diagram of the connective tissue follows a hysteresis loop (according to J.A. Ewing, variant delayed behaviour on the input size).

TRAINED AND UNTRAINED CONNECTIVE TISSUE | *Fig. 5 b*

Modified from Brinckmann et al. 2012 p. 39 Orthopaedic Biomechanics MV-Verlag.



Trained connective tissue with good elasticity (tendon) has a smaller hysteresis loop (like a spring). Connective tissue with plastic properties has a larger hysteresis loop (like a sponge). Patients with a weakness in their connective tissue have a large hysteresis.

The different properties of the tendon (serial element) and the fasciae (parallel elastic element) are explained in the following.

TENDON

The tendon is part of the connective-tissue, non-contractile elements of the muscle. It is integrated in the continuum of the connective-tissue structures of the muscle, which begins on the bone in the tendo-osseous transition and continues to the tendon. Connective tissues of the tendon, in the form of the epimysium, perimysium and endomysium, transition into the connective tissue of the muscle body.