SHOCK WAVE THERAPY IN PRACTICE

SHOCK WAVES IN SPORTS MEDICINE

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LEVEL 10

CONTENTS

EDITORIAL

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Extracorporeal Shock Wave Therapy (ESWT) for treatment of musculoskeletal disorders was introduced about 25 years ago, more or less by accident. Following the first human Extracorporeal Shock Wave Lithotripsy (ESWL) application to treat kidney stones in 1980, research was focused to investigate the effects and side effects of shockwave transmission in the tissues between the skin and the kidney stone.[1] Because bone and kidney stones were thought to have similar physical characteristics (hard material), specific interest was initially put on bone. It was demonstrated that ESWL was able to destroy or fracture bones in vitro.[5]

Contrary to this, in vivo bone formation was observed following ESWL in several animal studies.[2] Consequently, in 1991, the first human musculoskeletal ESWT applications were described in a cohort of 82 delayed or non-union fractures and 70 successful results were reported.[5]

The next phase (first half of the 1990s) was driven by the assumption that symptomatic radiopaque lesions in the rotator cuff (calcifying tendinopathy) and degenerative enthesiopathic insertional tendon lesions (plantar fasciitis / heel spur and tennis elbow) could possibly be successfully targeted.[2]

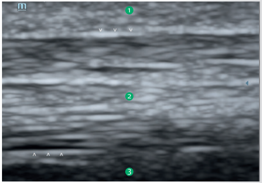
In the middle of the 1990s, the first devices for specific orthopaedic ESWT were introduced into the market. Compared with the ESWL devices, easier handling was achieved by direct coupling (gel) between the applicator and the skin instead of water immersion. Reduced maximum energy and aiming by ultrasound instead of fluoroscopy were big advantages. Also the size of the apparatuses and their costs were reduced.

In 1995, we introduced focused ESWT into sports medicine (Figure 1) and published the first related article, demonstrating 31 % overall pain reduction for ESWT applied to different overload sports injuries and 46 % of these athletes were painfree when treated with low energy (0.03–0.08 mJ/mm²).[3]

When using transducers of > 12 MHz, the resolution of the primary and secondary bundles increases clearly. The boundary of the tendon, defined by its external peritendineum (arrow tips), can now be well discerned.

1 = Cutis and subcutis
2 = Fibrillar echotexture of Achilles tendon
3 = Fatty tissue

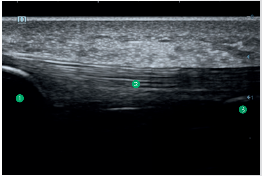
SIGNATURE OF A HEALTHY ACHILLES TENDON (LONG AXIS IN ZOOM MODE) | *Figure 1c*



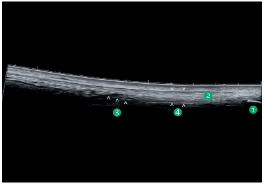
By using the extended field of view function, the area of the patellar tendon that can be depicted increases from 4 to approx. 6 cm. However, due to splaying of the sound waves, the parts close to the boundaries are no longer hit by the sound waves in orthograde direction, thus causing anisotropy of the tendon.

1 = Patella
2 = Patellar tendon
3 = Tibial head

NORMAL RESULT FOR PATELLAR TENDON (LONG AXIS WITH EXTENDED FIELD OF VIEW) | *Figure 2*



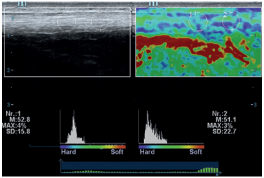
NORMAL RESULT FOR ACHILLES TENDON (LONG AXIS WITH PANORAMIC IMAGE) | *Figure 3*



With the panoramic imaging function it is possible to depict and/or document the changes of a tendon along its entire length. However, the resolution is lower here compared with that of the normal B image.

1 = Calcaneus
2 = Achilles tendon
3 = Transition from muscle to tendon
4 = Peritendineum

STRAIN ELASTOGRAPHY OF A NORMAL ACHILLES TENDON | *Figure 4*

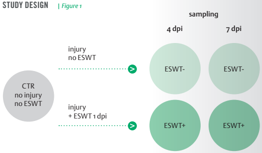


The stiffness of the tendon can be evaluated using strain elastography. This examination method allows a direct comparison of the elasticity (strain ratio) along the tendon.

STUDY DESIGN | Figure 1

Overview of experimental groups (n ≥ 6). Except for the control group (CTR), all animals received a cardiotoxin-induced injury in the hind limb muscle. Two of the injured groups (ESWT+) were treated with a single ESWT session on day one post injury. At day four and at day seven post injury, muscles from one treated (ESWT+) and one untreated (ESWT-) group were sampled to analyse the progress of regeneration.

dpi: days post injury



Tissue samples were either fixed in Karnovsky's fixative, followed by resin embedding for semithin sectioning, or snap frozen via 2-methylbutane for cryosectioning. Quantitative analyses were carried out on non-overlapping photographs of the lesion centres taken on either 1.5 µm semithin sections or 10 µm cryosections (three sections per lesion cut at a minimum distance of 1 mm).

Quantitative evaluation of the regeneration processes involved the following variables: minimal Feret diameter (F_{min}), defined as the closest possible distance between two parallel tangents of an object (e.g. a muscle fibre), as well as cross sectional area (CSA) of newly formed muscle fibres, as identified by the presence of centrally located nuclei, myonuclear content (number of central, peripheral and total nuclei per fibre) and CSA/nuclei ratio. Data of all groups were compared by statistical analysis of variance (ANOVA, Kruskal-Wallis).

With regard to both size-dependent variables, F_{min} and CSA, animals of the ESWT+ groups exhibited significantly larger muscle fibres compared with the ESWT-group animals on day four as well as on day seven post injury. Comparison of mean fibre size in the regenerating lesions with those of uninjured (healthy state) CTR group muscle similarly revealed a clear advantage of ESWT+ over ESWT-. While fibre sizes in ESWT+ lesions reached

42 % and 67 % of the healthy state value on day four and day seven post injury, respectively, the corresponding values for ESWT- lesions were only at 35 % and 58 %, respectively. This represents a mean diameter increase by 18 % on day four and 14 % on day seven post injury as a result of a single ESWT treatment (Figure 2, Table 1).
Central nuclei within muscle fibres indicate recent fusion of satellite cells and thus regeneration. Mean numbers of both central nuclei and total nuclei per regenerating fibre were significantly higher in ESWT+ lesions compared with the ESWT- groups at both time points investigated. Similarly, when analysing the overall ratio of fibres with three or more nuclei, the ESWT+ lesions significantly outmatched ESWT- lesions. While in the latter, on day four post injury only 25 % of all regenerating fibres had three or more nuclei (mean at 1.9 nuclei/fibre), 45 % of all ESWT+ group animals displayed fibres containing three or more nuclei (mean at 2.6 nuclei/fibre). Similar results were obtained on day seven post injury: ESWT- group 23 %, mean 1.9 nuclei/fibre, and ESWT+ group 46 %, mean 2.6 nuclei/fibre. Notably, the ESWT associated increase in nuclei per fibre is consistent with the increases in fibre area and diameter. There was no statistical difference in the CSA/myonuclei ratio between the ESWT+ and ESWT- groups at day seven post injury, indicating a regular, healthy regeneration process in the ESWT+ groups (Figure 2, Table 1).

Table 1
Evaluated variables of regenerating muscle fibres in ESWT+, ESWT- and control (CTR) group muscles. dpi: days post injury, n.a.: not available, n.s.: not significant. Data from [62].

OVERVIEW AND RESULTS OF EVALUATED VARIABLES[62] | Table 1

	4 dpi			7 dpi			
	ESWT-	ESWT+	P value	ESWT-	ESWT+	P value	CTR
F_{min}, µm	17.0 ± 1.0	20.0 ± 2.0	<.001	28.0 ± 2.0	32.0 ± 1.0	<.001	48.0 ± 3.0
CSA, µm²	336 ± 50	477 ± 99	<.001	890 ± 110	1130 ± 90	<.001	2450 ± 282
central nuclei/fibre	1.7 ± 0.1	2.2 ± 0.2	<.001	1.4 ± 0.1	1.8 ± 0.2	<.001	0.0 ± 0.0
≤ 2 nuclei/fibre, %	75.4	54.8	<.001	77.4	53.5	<.001	n.a.
≥ 3 nuclei/fibre, %	24.6	45.2	<.001	22.6	46.5	.001	n.a.
$pax7^+$, % of total nuclei	2.8 ± 0.7	3.7 ± 0.6	<.001	3.5 ± 0.6	5.4 ± 0.5	<.001	0.2 ± 0.2
myoD+, % of total nuclei	1.6 ± 0.2	3.6 ± 0.5	<.001	3.2 ± 1.0	5.1 ± 0.9	<.001	0.0 ± 0.0
$myogenin^+$, % of total nuclei	1.5 ± 0.3	2.8 ± 0.4	<.001	2.4 ± 0.3	3.9 ± 0.8	<.001	0.1 ± 0.1
$H3P^+$, % of total nuclei	3.0 ± 0.6	6.7 ± 1.1	<.001	7.0 ± 1.4	8.2 ± 1.6	n.s.	0.1 ± 0.1