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Dose-related effects of shock waves on rabbit tendo Achillis

A SONOGRAPHIC AND HISTOLOGICAL STUDY

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We aimed to determine whether extracorporeal shock waves of varying intensity would damage the intact tendo Achillis and paratenon in a rabbit model. We used 42 female New Zealand white rabbits randomly divided into four groups as follows: group a received 1000 shock-wave impulses of an energy flux density of 0.08 mJ/mm^2, group b 1000 impulses of 0.28 mJ/mm^2, group c 1000 impulses of 0.60 mJ/mm^2, and group d was a control group.

Sonographic and histological evaluation showed no changes in group a, and transient swelling of the tendon with a minor inflammatory reaction in group b. Group c had formation of paratendinous fluid with a significant increase in the anteroposterior diameter of the tendon. In this group there were marked histological changes with increased eosin staining, fibrinoid necrosis, fibrosis in the paratenon and infiltration of inflammatory cells.

We conclude that there are dose-dependent changes in the tendon and paratenon after extracorporeal shock-wave therapy and that energy flux densities of over 0.28 mJ/mm^2 should not be used clinically in the treatment of tendon disorders.

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There have been several recent reports on the use of extracorporeal shock waves in the treatment of pseudarthrosis[1-3] calcifying tendinitis[4-7] and tendinopathies of the elbow.[8] Claims have also been made for the use of shock waves in the removal of cement in revision arthroplasty and even for the treatment of avascular necrosis.[9]

The beneficial effects of low-energy shock waves may be related to Melzack's[10] concept of hyperstimulation analgesia, in that a moderate sensory stimulus can relieve pain when it is administered directly to the site, and a relatively short stimulus (seconds to minutes) may relieve chronic recalcitrant pain for a long period. High-energy shock waves have also been shown to cause mechanical disintegration of calcific deposits in tendons. There have been no reports on alteration or damage to tendon and peritendinous tissues after shock-wave therapy, although there have been extensive descriptions of pulmonary alveolar injuries,[11] renal sub- and pericapsular haematoma,[12-14] and hepatic necrosis or haematoma.[15,16]

We aimed to evaluate the extent to which extracorporeal shock waves may be harmful to such tissues, in particular tendo Achillis.

Materials and Methods

After ethical approval by the university commission we studied 84 tendons in 42 New Zealand rabbits, randomly assigned to four treatment protocols as follows:

Group a – 1000 shock-wave impulses of an energy flux density of 0.08 mJ/mm^2 (low energy – 24 tendons).

Group b – 1000 impulses of 0.28 mJ/mm^2 (medium energy – 24 tendons).

Group c – 1000 impulses of 0.60 mJ/mm^2 (high energy – 24 tendons).

Group d – no shock wave therapy (control group – 12 tendons).

Randomisation was by sealed envelopes opened immediately before starting application of the shock wave.

The term 'energy flux density' is used to indicate that shock-wave energy 'flows' through an area perpendicular to the direction of propagation, and is measured as mJ/mm^2. The commonly used unit of kV gives no information on the energy at the focus, and is no longer recommended for the description of medical shock-wave fields.[5]

Shock-wave application. The shock waves were applied by an experimental device (Osteostar; Siemens AG, 91052 Erlangen, Germany) in which an electromagnetic shock-wave generator was integrated in a mobile fluoroscopy unit. The shock waves are generated by a strong electric current

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Table I. Semiquantitative rating of sonographic changes in the tendon

0	No alteration. Change in diameter of less than 0.5 mm compared with before treatment
I	Possible alterations. Change in diameter between 0.5 and 1.0 mm
II	Definite alteration in ultrasound pattern; the presence of paratendinous fluid and change in diameter of over 1.0 mm

through a flat coil, which induces a magnetic field in an overlying metal membrane. Just as similar poles repel each other, the generated magnetic fields in the membrane and the coil lead to sudden movements of the membrane, which induce shock waves in the surrounding liquid. An acoustic lens is used to focus the shock waves at the centre of the C-arm. The focal area of the shock waves is defined as the area within which 80% of the maximum energy is reached. It has a length of 50 mm in the direction of the shock waves, and a radius of 3.5 mm perpendicular to this axis.

Each rabbit was anaesthetised by the intramuscular injection of ketamine and atropine sulphate, with continued ketamine and xylazine, and the hindlimb was shaved. The ankle was fixed in the neutral position. Using ultrasound control, the tendon was marked by a metal clip 1 cm proximal to its calcaneal insertion. The marked tendon was then placed in the centre of the C-arm using fluoroscopy and the shock-wave unit docked to the lower leg by means of a water-filled cylinder. Ultrasound gel (University Hospital, Mainz, Germany) was used as a contact medium between the cylinder and the skin. The administration of 1000 shock-wave impulses took a mean of 32 minutes (20 to 42), the variation being due to the learning curve for intravenous anaesthesia. After recovery, minor skin damage was treated, and settled well.

Sonographic assessment. At one-week intervals, we obtained high-resolution ultrasound images of the tendons under sedation with Promazin using a Siemens SL 400 machine with a 7.5 MHz linear array probe. Strictly longitudinal sections were taken by an experienced examiner and recorded on a thermoprinter (Video Copy Processor P66E; Mitsubishi Electrical Corporation, Tokyo, Japan). Evaluation of the sonograms was performed without knowledge of the treatment procedure, and based not only on measurement of the diameter of the tendon, but also a semiquantitative scale of tendon function during flexion/extension of the ankle. Morphological features and changes in the anteroposterior diameter of the tendon were scored as described in Table I.

Histopathological examination. A number of each group of rabbits were killed by intravenous xylazine, ketamine and thiopental, also at weekly intervals (Table II). Specimens were taken and fixed in 4% paraformaldehyde and processed to provide 5 μm paraffin sections which were stained with haematoxylin and eosin and alcian blue. The sections were assessed by a pathologist without knowledge of the treatment group using the criteria of Backman et al,[17] to grade staining affinity, nuclear appearance and fibrillar appearance of the tendon on a four-point scale from no change to marked change. The thickness, degree of fibrosis, degree of oedema, capillary changes, and degree of inflammatory response of the paratenon were recorded.

Statistical methods. We used the exact chi-squared and the Fisher-Yates tests for small samples. The Fisher-Yates test was used for frequencies in two-by-two contingency tables and the exact chi-squared test when the number of either row or column was greater than two and the observed frequencies were small.

Results

Throughout the experimental period, the animals remained in generally good condition and were not immobilised. No ani-

Table II. Semiquantitative assessment of histopathological changes (mean values). Grading: 0, no change; 1, slight change; 2, moderate change; and 3, marked change

			Tendon			Paratenon			
Group	Follow-up (days)	Number	Staining affinity (grade 0 to 3)	Nuclear appearance (grade 0 to 3)	Fibrillar appearance (grade 0 to 3)	Thickness (grade 0 to 3)	Occurrence of fibrosis (grade 0 to 3)	Capillaries (grade 0 to 3)	Inflammation (grade 0 to 3)
a	1	6	0.16	0.0	0.16	0.0	0.16	0.0	0.0
	7	6	0.16	0.16	0.0	0.0	0.0	0.16	0.0
	14	6	0.0	0.16	0.0	0.0	0.0	0.0	0.0
	28	6	0.16	0.0	0.16	0.0	0.0	0.16	0.0
b	1	6	1.16*	0.0	1.0*	1.16*	1.33*†	1.67*†	1.5*†
	7	6	1.0*	0.0	0.5	0.83	0.5	0.83	1.0
	14	6	0.5	0.16	0.33	0.0	0.5	0.33	0.0
	28	6	0.0	0.0	0.16	0.0	0.33	0.0	0.0
c	1	6	1.5*	1.0	1.5*†	2.5*†	0.83*	3.0*†‡	1.5*†
	7	6	2.83*†‡	3.0*†‡	3.0*†‡	1.83*†	0.5*†	2.83*†‡	0.5
	14	6	2.0*†	1.83*†‡	2.16*†‡	1.0*†‡	2.83*†‡	1.5*†‡	2.5*†‡
	28	6	1.83*†‡	1.33*†‡	1.5*†‡	0.5	1.5*†	0.5	0.33
d		12	0.16	0.0	0.25	0.0	0.0	0.16	0.0

* significance *v* control group (subgroup d)
† significance *v* subgroup a
‡ significance *v* subgroup b

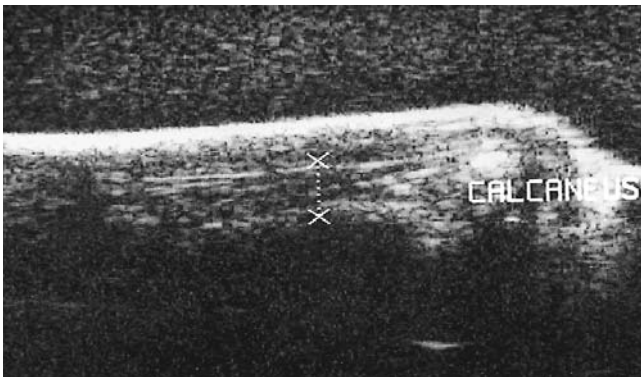

Fig. 1a

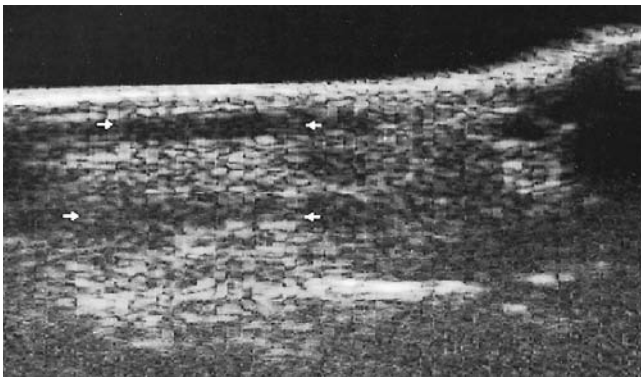

Fig. 1b

Ultrasonograms of rabbit tendo Achillis showing (a) a normal appearance, with an anteroposterior diameter of 3.0 mm, and (b) at one day after high-energy shock waves. There is an increase in anteroposterior diameter, to 4.3 mm, with fluid accumulations (arrows).

mal showed signs of muscular or neurological dysfunction.

Sonography. The pretreatment tendo Achillis showed as an echo-rich continuous structure of parallel reflections. Proximally, the distal gastrocnemius muscle was visible and distally there was a typical transition to the bony structure of the calcaneus. Subcutaneous and peritendinous fatty tissue was echo-poor compared with the tendon itself (Fig. 1).

In groups a and b (low and medium energy) changes of less than 20% were described with no morphological alterations other than a 1 mm increase in anteroposterior tendon diameter (Figs 2a and 2b). There were no statistically significant differences between groups a and b.

In group c (high energy) the tendon diameter was always increased by over 1 mm at one day after application of the shock wave and most cases showed peritendinous collections of fluid (see Fig. 1b). These changes returned to normal within 28 days in all animals (Fig. 2c), and no sonographic changes were seen in the surrounding muscles.

Group c differed significantly from groups a and b at days 1 ($p < 0.001$) and 7 (a *v* c: $p = 0.034$; b *v* c: $p = 0.016$) after the application of the shock wave, but there were no significant differences after 14 or 28 days.

Groups a, b and c differed significantly from the control group ($p < 0.001$) at day 1. By day 7 there was a significant difference for group a ($p = 0.037$) and group c ($p < 0.001$). At day 14, only group c differed significantly from control values ($p = 0.002$), and by day 28 there were no significant differences.

Histopathological examination. In the control animals the tendon and paratenon had a normal histological appearance with homogenous staining of collagen bundles. For group-a (low energy) specimens there were no differences from control specimens at any follow-up period, with no capillary ruptures and no inflammatory reactions (Figs 3 and 4).

In group b (medium energy) there were changes in the staining affinity, thickening of the paratenon, and dilated capillaries. At day 1, inflammatory reactions included increased numbers of polymorphonuclear granulocytes in the peritendinous tissue and sporadic extravasation of erythrocytes. At day 7, the inflammatory peritendinous reaction had decreased. Alcian blue staining, which is specific for glycosaminoglycans, showed a slight increase in intensity, but structural integrity was intact. After 14 and 28 days the sections showed mostly normal findings (Fig. 5).

In group c (high energy) there was massive extravasation of erythrocytes which caused capillary disruption within 24 hours, and the peritendineum was oedematous and thickened. There was no intratendinous bleeding, even adjacent to the subcutaneous or peritendinous haematomas. At day 7, the tendon fibres were swollen with an intensely eosinophilic reaction, extensive areas of necrosis and absence of fibrocyte nuclei, even in the peritendinous tissue, and some extravasated erythrocytes were completely homogenised. At day 14, a fibroblast proliferation was seen in the peritendineum, and within the tendon areas with normal nuclei alternating with necrotic areas without any nuclei. Where fibrocytes were found their tissue orientation and structure appeared irregular.

Even at day 28 the staining reaction with alcian blue was more intense than in the control group, indicating an increased production of glycosaminoglycans or reduced metabolism due to fibroblast damage. At this stage there were no inflammatory reactions or erythrocyte extravasation (Fig. 6).

A semiquantitative assessment of the histopathological changes in each treatment group is shown in Table II. There were significant differences from the control group in the appearance of the tendon and paratenon in groups b and c, and at most follow-up intervals group c differed significantly from groups a and b.

Discussion

Shock-wave damage to the kidney, liver and lung has been described,[14-16,18,19] and chondroid metaplasia of muscle and an effect on bony healing have also been reported.[9] In a recent review article[20] we have listed animal studies showing positive effects on osteogenesis[21-24] and negative influences,[25-30] including osteocyte damage, dysplasia of

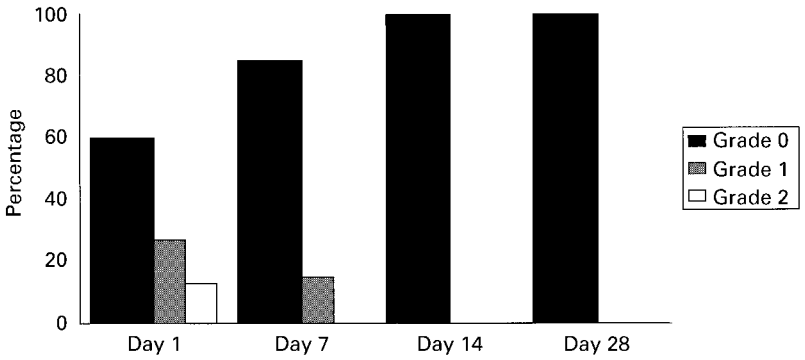

Fig. 2a

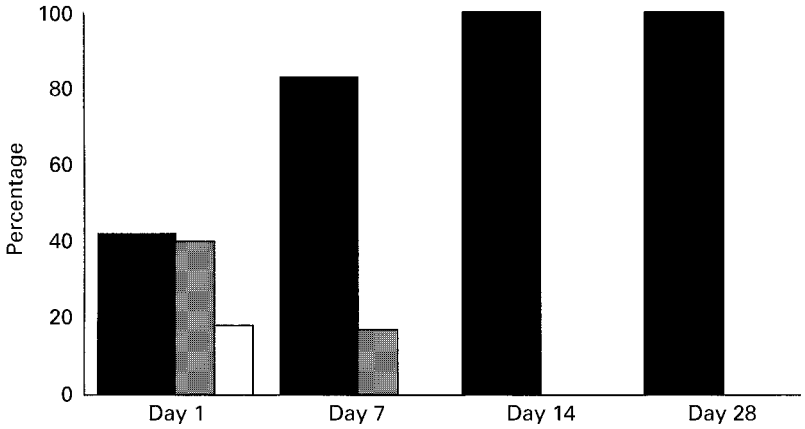

Fig. 2b

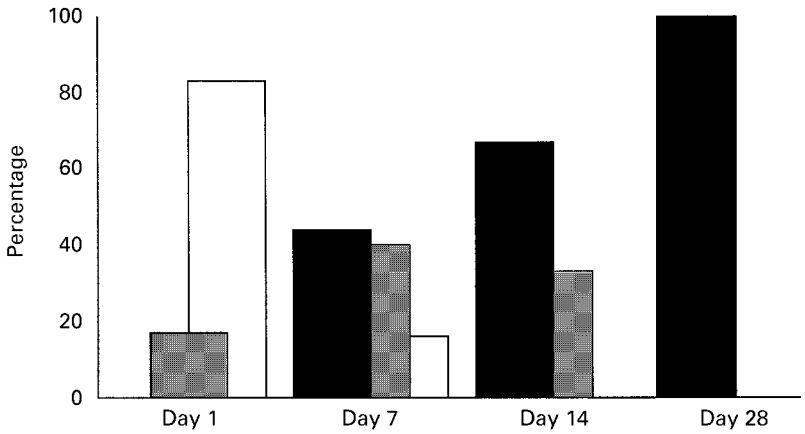

Fig. 2c

Changes in tendo Achillis by the criteria shown in Table I in (a) group a (low energy), (b) group b (medium) and (c) group c (high).

the growth plates, delay of fracture healing and a decrease in mechanical stability. We concluded that there was no clear evidence of any osteogenetic effects from high-energy shock waves.

To our knowledge, there are no published reports on the effect of shock waves on tendons, but there is much information on tendon reaction after mechanical injury. Repair processes start from both the surrounding soft tissues (extrinsic healing) and within the tendon itself (intrinsic healing). Of tendons with no sheaths, tendo Achillis in the rabbit has been most intensely investigated,[31] with accounts of necrosis, granulocyte invasion, thickening of the paratenon, capillary proliferation, proliferation of extracellular matrix with chondroid metaplasia and late replacement of granulation tissue by scar tissue. Full repair to normal cannot be expected.[32-35]

We used an animal model to study the chronological reaction of rabbit tendo Achillis and paratenon after extracorporeal shock waves. Ultrasound did not show any direct injury to the tendon, but there was a dose-dependent

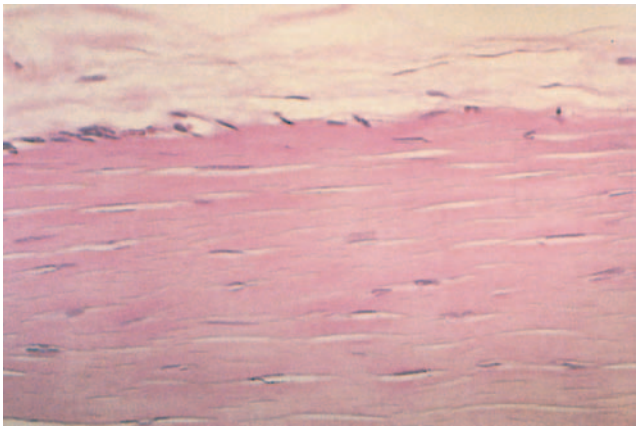

Fig. 3

Normal tendon structure in control animals. There are parallel collagenous fibres and fibrocyte nuclei in the tendon, and above it loose peritendinous tissues with no inflammation (haematoxylin and eosin ×134).

increase in anteroposterior diameter with evidence of accumulation of fluid in the adjacent soft tissues. We saw no damage to muscle and bony structures.

The histopathological changes depended on the intensity of the shock waves, and were similar to those after blunt trauma including degenerative signs, inflammatory cells, increased numbers of capillaries, and oedema and fibrosis in the paratenon. The extent of these changes varied between the animals in each treatment group, but there was a marked increase in changes in both tendon and paratenon with greater shock-wave intensity, confirming similar observations for various soft tissues.[14-16,36,37]

The results from our animal model cannot be directly extrapolated to man, partly because the energy levels used are not directly comparable to the energies used clinically. Nevertheless, our results considered in the light of published post-traumatic findings allow some conclusions as follows:

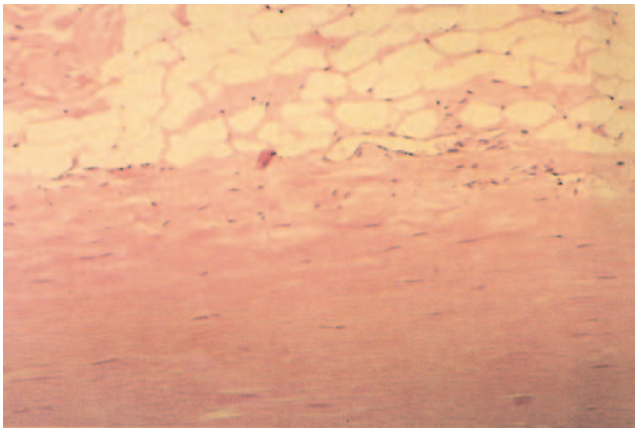

Fig. 4a

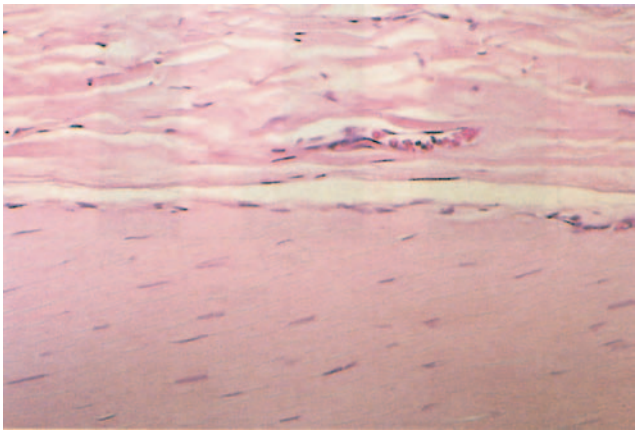

Fig 4b

Group a (low energy). Figure 4a – At day 1, there is normal histology of the tendon and peritendineum (haematoxylin and eosin ×66). Figure 4b – At day 7, there is a normal architecture of the tendon (haematoxylin and eosin ×134).

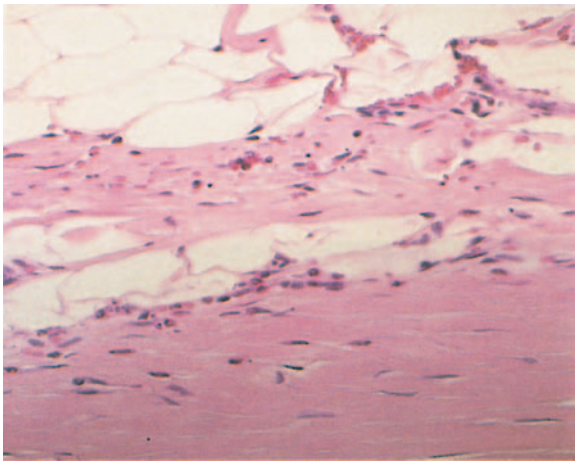

Fig. 5a

Fig. 5b

Group b (medium energy). Figure 5a – At day 1, there is normal tendon tissue in the lower part, but the superficial part shows oedematous peritendineum and accentuated cell-rich inflammatory infiltration with predominantly polymorphonuclear granulocytes. Figure 5b – At day 7, there is regression of the inflammatory reaction, and normal architecture of both the tendon and paratenon (haematoxylin and eosin ×66).

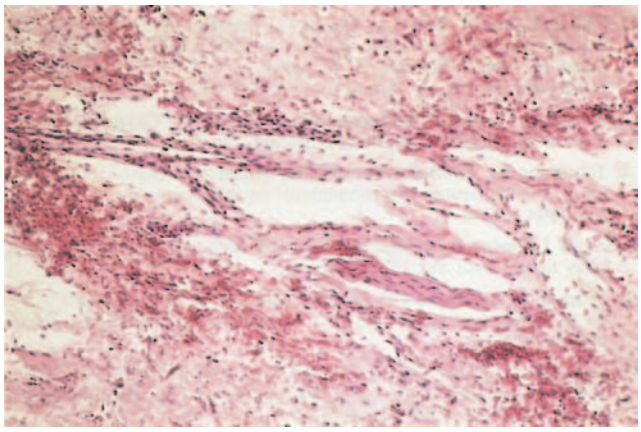

Fig. 6a

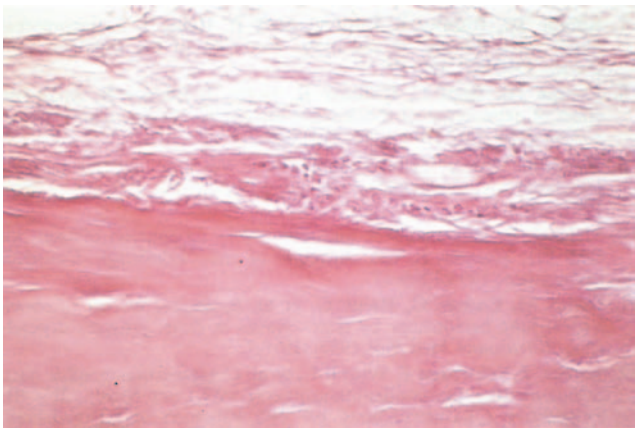

Fig. 6b

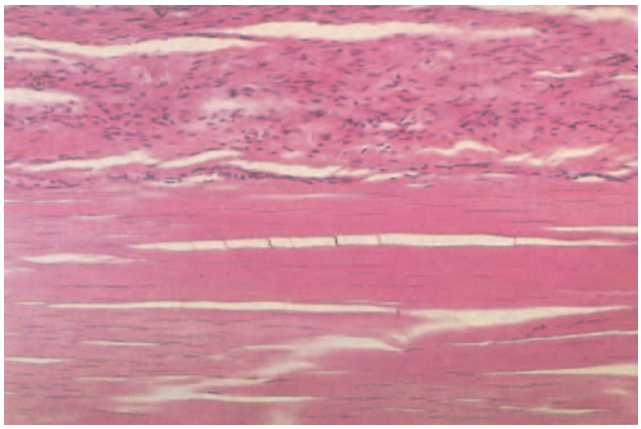

Fig. 6c

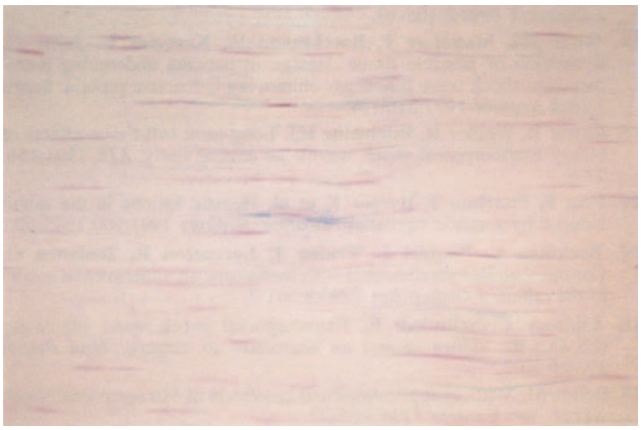

Fig. 6d

Group c (high energy). Figure 6a – At day 1 the peritendineum shows oedematous swelling, extensive erythrocyte extravasation and an early inflammatory response (haematoxylin and eosin ×66). Figure 6b – At day 7 there is extensive fibrinoid necrosis of the tendon and peritendineum. The collagenous fibres are structureless and hypereosinophilic; nuclear integrity is disrupted. The peritendineum is oedematous and necrotic (haematoxylin and eosin ×66). Figure 6c – At day 14, there is increased cellularity of the loose peritendinous connective tissue with a marked fibroblast reaction. The fibre content is increased indicating slight fibrosis. There is no inflammatory reaction within the tendon, but a great reduction in the number of fusiform fibrocyte nuclei (haematoxylin and eosin ×66). Figure 6d – At day 28 the fibrocyte nuclei have a normal appearance within the tendon. There is an increase in staining affinity of the intercellular matrix. In control sections glycosaminoglycans are practically invisible (alcian blue ×134).

1) Energy flux densities of up to 0.28 mJ/mm^2 gave no evidence of marked damage to the tendon and adjacent tissues, all changes being reversed within four weeks.
2) At 0.60 mJ/mm^2 there was marked damage to the tendon and paratenon. The increase in tendon diameter, the fibrinoid necrosis and the inflammatory and reparative peritendinous reactions had not completely disappeared after four weeks. These would decrease mechanical strength, with danger of partial or complete tears of the tendon.

Our clinical experience of over 300 patients followed for at least two years and assessed by ultrasound or MRI showed no traumatic tendon changes after extracorporeal shock waves at a maximum energy flux density of 0.28 mJ/mm^2 (unpublished results). The fear of the rupture of already damaged tendons leads us to recommend that shock-wave therapy of high energy should not be used to treat tendon disorders in man until further studies have been performed.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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