■ ASPECTS OF CURRENT MANAGEMENT

Extracorporeal shock-wave therapy in the management of chronic soft-tissue conditions

C. A. Speed

Extracorporeal shock waves are focused, single-pressure pulses of microsecond duration. They were first utilised for medical purposes over two decades ago in the treatment of renal calculi by lithotripsy. Soon afterwards shock waves were used in the management of ununited fractures.^{1,2} In the 1990s extracorporeal shock-wave therapy (ESWT) became popular in Germany for certain soft-tissue disorders, including calcifying tendonitis of the rotator cuff, humeral epicondylitis and plantar fasciitis. It is now employed worldwide for the treatment of musculoskeletal complaints. In the USA the Federal Drug Administration gave permission for its use in the treatment of chronic proximal plantar fasciitis in 2000.^{3,4}

Although ESWT has become increasingly popular there is considerable debate as to its appropriate usage and efficacy. This review addresses issues relating to its use in soft-tissue complaints including the technical features, economic aspects, effects on tissue, potential adverse effects and the current evidence for its use.

Technical aspects

Shock waves are three-dimensional pressure pulses of microsecond duration with peak pressures of 35 to 120 MPa. Many of the physical effects are considered to be dependent upon the energy involved, measured in millijoules, which is calculated by taking the integral time over the pressure-time function at each particular location of the pressure field.⁵ This is affected by the pulse pressure, the density of the propagation medium and the area in which the shock wave is existent. For medical use, shock waves are concentrated into small focal areas of 2 to 8 mm in diameter in order to optimise the therapeutic effects and to minimise the effects on other tissues.⁶ The focus is defined as the site at which the maximum peak-positive acoustic pressure is attained. The concentrated shock-wave energy per unit area, the energy flux density (EFD), is a term used to reflect the flow of shock-wave energy perpen-

dicular to the direction of propagation and is taken as one of the most important descriptive parameters of shock-wave 'dosage'.⁵

The basic requirements of a shock-wave system are that it should have sufficient power to generate effective shock waves, be versatile in its ability to generate waves of a reliable range of energies, have a method to target the shock waves to a specific site and have minimal adverse effects.

The characteristics of shock waves are determined by the type of device used. They are generated by an electric storage capacitor with variable high voltage which is charged and subsequently rapidly discharged by electroacoustic transducers.⁷ The generators can involve electrohydraulic, electromagnetic or piezoelectric mechanisms. The type of the source determines the shape of the pulse.^{7,8} Piezoelectric generators have the benefit of accurate focusing, but generally generate high peak pressures, while electrohydraulic and electromagnetic devices can generate a range of peak pressures. However, electrohydraulic generators, the original type used, are limited by considerable shock-to-shock variation in the focal pressure and in difficulties with localisation of their effect. The shape of their pulse cannot be variably controlled as in electromagnetic and piezoelectric pulse generators (Table I).⁹ Although specific waveforms can be produced and may have different effects on tissue, such characteristics may not be maintained during the passage through tissues.

Regardless of the method of generation, shock waves are concentrated by means of focusing reflectors on the target site. Localisation of the shock waves can be performed using imaging modalities, typically fluoroscopy or ultrasound. These may be external to the system 'off line', or integrated within the treatment head, 'in line'. The latter is preferable since it allows more accurate targeting.

Dosage

While the terms 'high', 'medium' and 'low' energy are commonly used in the literature,

■ C. A. Speed, Honorary Consultant Rheumatology, Sports & Exercise Medicine, Addenbrooke's Hospital, Hills Road, Cambridge CB2 2QQ, UK.

©2004 British Editorial Society of Bone and Joint Surgery
doi:10.1302/0301-620X.86B2.14253 \$2.00
J Bone Joint Surg [Br]
2004;86-B:165-71.

Table I. A comparison of the devices used to generate shock waves

	Advantages	Disadvantages
Piezoelectric	Long service life Good focusing accuracy Low acoustic power treatment can be performed without anaesthesia	Low power may result in inefficacy or repeat treatments Difficult to build in x-ray localisation systems
Electrohydraulic	Can generate a range of peak pressures	Substantial pressure fluctuations Limited service life
Electromagnetic	Can generate a range of peak pressures Integration of in-line localisation devices is facilitated by cylindrical design Deep penetration of shock waves is possible Precisely defined focal point Large aperture distributes energy over a large skin surface, potentially reducing adverse effects	Cost

Table II. Classification of shock-wave therapy according to energy flux density (EFD) ranges

Authors	Level	EFD range (mJ/mm ²)
Mainz	Low	0.08-0.27
	Medium	0.28-0.59
	High	>0.60
Kassel	Low	<0.12
	High	>0.12

there is no clear consensus on the EFDs involved in relation to these terms. Two classifications have been proposed (Table II).

Individual treatments are usually described according to the number of shocks administered, the generator frequency and the energy level setting. The last varies according to the machine used, with each energy setting representing a specific EFD.

Effects of shock waves on soft tissues

Shock waves may have beneficial or adverse effects on soft tissues (Table III). The passage of shock waves to their target can result in damage to tissue along the axis of the field of the shock wave. This can result in localised bleeding, and petechiae and haematomas have been seen after treatment, particularly with high-energy pulses.^{10,11}

Cavitation, the movement of new and pre-existing gas bubbles in a fluid, is thought to play a significant role in the development of tissue changes in lithotripsy resulting from a shock wave gas bubble interaction.¹² Such bubbles expand within a few microseconds of the shock wave, and usually then collapse within 100 microseconds, generating a second, spherical, shock wave. Cavitation can have mechanical and chemical effects which may be therapeutic, as with disintegration of calcification, or potentially deleterious. Mechanical alterations are principally due to shearing effects, while chemical phenomena appear mainly to be due to the development of free radicals.^{13,14}

Shock waves can destroy cells acutely as a result of the production of free radicals and cell lines appear to differ in their susceptibility to destruction.¹⁵ Most cells which survive the shock continue to function and divide normally, irrespective of the cell cycle at the time of exposure.¹⁶ However, ultrastructural changes have been demonstrated on electron microscopy, including changes in the cytoplasm and the mitochondria,¹⁷⁻¹⁹ most of which require EFDs in the region of 0.5 mJ/mm².

Alterations in the cell membrane, including its permeability, occur at lower EFDs of 0.12 mJ/mm².²⁰

Most of the observed changes with the use of ESWT in man have been observed on examination of non-musculo-

Table III. Potential mechanisms of beneficial and adverse effects of ESWT

	Potential adverse effects	Proposed beneficial effects
Direct tissue trauma and/or cavitation	Bleeding Production of free radicals Mechanical shearing Ultrastructural damage	Stimulation of tissue healing – mechanism unknown Disintegration of calcium Transient inflammatory response
Altered cell-membrane permeability	Cell death	Suppression of pain transmission Other effects?
Direct effect on nociceptors		Denervation: antinociceptive effect
Peripheral nerve stimulation	Arrhythmias, peripheral paraesthesiae	Hyperstimulation, blocking gate control mechanism

Table IV. Randomised, controlled trials of ESWT in specific soft-tissue conditions

Condition	Reference	Symptom duration	Total sample size	ESWT regime	Shock wave generator*	Focusing of ESWT	First outcome point (weeks)	Evidence for benefit
Calcific tendonitis	30	Pain >12 months	80	A: No treatment Others 200 impulses under LA† B: 0.1 mJ/mm ² C: 0.3 mJ/mm ² D: As C, 2 sessions over 1 week	EH	Fluoroscopic	12	Yes
Non-calcific tendonitis of the rotator cuff	31	Pain >6 months	40	Sham v 2000 impulses at 0.11 mJ/mm ² repeated at weekly intervals for 3 sessions	EM	Ultrasound	6 and 12	No
	32	Pain >3 months	74	Sham v 1500 impulses at 0.12 mJ/mm ² monthly for 3 months in 74 adults with chronic tendinosis of the rotator cuff persisting for at least 3 months. No LA	EM	Ultrasound	12 and 24	No
Plantar fasciitis	4	6 months	260	Sham or 1500 shocks at >0.18 mJ/mm ² . Multicentre LA	EH	None. Heel moved during treatment	12	Yes
	39	6 weeks	160	ESWT weekly for 3 weeks to a total dose of at least 1000 mJ/mm ² or placebo to a total dose of 6.0 mJ/mm ² .	EM	Ultrasound		No
	40	3 months	88	Sham or 1500 shocks at 0.12 mJ/mm ² monthly for 3 months. No LA	EM	Ultrasound	12	No
	38		60	Six treatments (1/7 to 10 days) of either 1200 shocks of 0.03 to 0.4 mJ/mm ² or sham treatment (0 mJ/mm ²) No LA	EH	Ultrasound	12	Yes
	41	12 months	30	1000 shocks of 0.06 mJ/mm ² or sham weekly for 3 weeks. No LA	EM	Fluoroscopy	12	Yes
Lateral epicondylitis	34	Randomised controlled, pain 12 months	100	3000 or 30 impulses at 0.08 mJ/mm ² . No LA	EM	None	3, 6, 24	Yes
	35	6 months	270	2000 shocks at 0.07 to 0.09 mJ/mm ² . No LA	Varied	Focusing uncertain	12, 52	No
	36	3 months	75	1500 pulses ESWT at 0.12 mJ/mm ² or sham therapy, monthly for 3 months. No LA	EM	Ultrasound	12	No

* EH, electrohydraulic; EM, electromagnetic

† LA, local anaesthetic

skeletal tissues, particularly the kidney, and most of the information available on the effects on musculoskeletal tissues is based on animal studies.²¹⁻²³ Studies of the effect of shock waves on tendo Achillis of the rabbit have indicated a dose-dependent effect, a transient inflammatory reaction and swelling of the tendon with 1000 impulses of higher (0.28 mJ/mm²) but not lower (0.08 mJ/mm²) doses.²⁴ Fibrinoid necrosis, paratenon fibrosis and marked inflammatory changes were seen with even higher doses (0.60 mJ/mm²).

Animal studies have demonstrated that considerable damage is done to lung parenchyma by shock waves. This appears to be related to almost total reflection of the waves when they reach a tissue/air interface, resulting in local damage.²⁵ There is also a potential for the development of cardiac arrhythmias. The pressure threshold for this appears to be in the range of 1 to 10 MPa.^{26,27} When multiple shock waves are administered at high frequency (100 Hz), arrhythmias can occur away from the focus. Stimulation of peripheral nerves also occurs within the focus of the

Table V. Results from a dose-ranging study of ESWT in 80 subjects with calcific tendonitis reproduced from reference 33

Group (n = 20 in each)	Constant score			
	Baseline	3 months	95% CI	p value
0 (control)	44.5 ± 8.3	47.8 ± 11.4	42.0 47.3 52.6	
1 (low energy) x 1 session	39.4 ± 11.2	51.6 ± 20.1	42.4 51.7 61.1	>0.5
2 (high energy) x 1 session	39.0 ± 11.8	63.7 ± 14.6	56.8 63.8 70.8	<0.0001
3 (high energy) x 2 sessions	43.5 ± 13.1	68.5 ± 13.1	62.1 68.5 74.8	<0.001

Table VI. Results of a randomised, controlled trial of ESWT in 40 subjects with non-calcific tendonitis reproduced from reference 34

Group/parameter	Control group		Treatment group		95% CI (group difference)
Constant score (age-corrected)					
Pretreatment	42.20 ± 13.04	(n = 20)	40.70 ± 13.29	(n = 20)	-6.93 to 9.93
At 6 weeks	64.17 ± 25.17	(n = 18)	60.95 ± 29.62	(n = 19)	-15.17 to 21.61
At 12 weeks	64.39 ± 32.68	(n = 18)	66.50 ± 37.92	(n = 20)	-25.53 to 21.31
Number of successful treatments 8 (n = 18)			10 (n = 20)		N/A
Subjective improvement (%)					
At 6 weeks	26.32 ± 28.67	(n = 19)	28.42 ± 32.02	(n = 19)	-22.10 to 17.89
At 12 weeks	31.05 ± 31.43	(n = 19)	40.00 ± 38.35	(n = 20)	31.77 to 13.87
Pain during rest (VAS 0 to 10)					
Pretreatment	5.40 ± 3.00	(n = 20)	5.35 ± 2.54	(n = 20)	-1.73 to 1.83
At 6 weeks	2.78 ± 2.71	(n = 18)	2.74 ± 3.03	(n = 19)	-1.88 to 1.97
12 weeks	3.22 ± 2.82	(n = 18)	2.30 ± 3.03	(n = 20)	-1.01 to 2.85
Pain during activity (VAS 0 to 10)					
Pretreatment	7.95 ± 1.96	(n = 20)	7.75 ± 1.48	(n = 20)	-0.91 to 1.31
At 6 weeks	5.72 ± 2.80	(n = 18)	5.74 ± 2.51	(n = 19)	-1.79 to 1.76
At 12 weeks	6.11 ± 3.23	(n = 18)	4.85 ± 3.07	(n = 20)	-0.81 to 3.33

shock wave. Thermally-induced adverse effects are unlikely as a result of only minor changes in temperature and the administration of pulses of only low frequencies.²⁸

Proposed mechanisms for explanation of clinical benefit

The mechanisms of the action of shock waves on soft-tissue conditions are unknown, but possibly include direct stimulation of the 'healing' processes, neovascularisation, disintegration of calcium and neural effects. These may involve alterations in the permeability of cell membranes preventing the development of potentials to transmit painful stimuli, direct suppressive effects on nociceptors and a hyperstimulation mechanism which blocks the gate control mechanism,^{23,24,29} but these possibilities remain speculative.

Methods of treatment

There are currently no standardised guidelines for the use of ESWT in soft-tissue conditions. A broad range of regimes with respect to the choice of machine, positioning and localisation of the patient, doses, treatment frequencies and the use of local anaesthesia has been employed.

Clinical evidence

A number of studies have now been published relating to the clinical effects of ESWT on soft-tissue injuries, most of which have been controlled. In this article only randomised controlled trials are considered in relation to specific soft-tissue conditions (Table IV).

Calcific tendonitis

Loew et al³⁰ subjected 80 patients with calcific tendonitis of the shoulder to varying patterns of treatment with shock waves. They were randomised to receiving either no treatment, a single session of 2000 shocks of either 0.1 mJ/mm² or 0.3 mJ/mm² or two sessions of 2000 shocks x 0.3 mJ/mm². The Constant scores both before and at three months after the treatment are given in Table V. One subject in the no treatment group, six in group 1, 12 in group 2 and 14 in group 3 reported subjective improvement in pain. There was radiological reduction in the calcium deposits in two of group 0, in four of group 1, 11 of group 2 and in 12 of group 3.

Non-calcific tendinopathy of the rotator cuff. Schmitt et al³¹ performed a prospective double-blind randomised controlled study of shock-wave therapy under local anaesthesia in

40 subjects with recalcitrant non-calcific tendonitis of supraspinatus. The ESWT group received 6000 shocks (EFD 0.11 mJ/mm^2) under ultrasound guidance, using an electromagnetic generator. In the control group a foil was placed between the patients and the shock-wave head to prevent transmission. Improvements were seen in both groups, with no significant difference between the two (Table VI).

We noted similar results in a double-blind randomised controlled trial of 1500 pulses of ESWT at 0.12 mJ/mm^2 or sham therapy, monthly for three months, in 74 adults with chronic tendinosis of the rotator cuff.³² Sham therapy involved deflation of the treatment head, no coupling gel and the use of minimal energy pulses (0.04 mJ/mm^2) in order only to replicate the noise of the machine, but avoidance of contact with the region of interest. Both groups showed significant and sustained improvements from two months onwards. There was no significant difference between the groups with respect to the degrees of change in shoulder pain and disability index (SPADI) scores or night pain over a period of six months.

A mean change in the SPADI (SD; range) of 16.1 (27.2; 0 to 82) in the treated patients and of 24.3 (24.8; -11 to 83) in the sham group was noted at three months. The mean changes at six months in the treatment and sham groups were 28.4 (25.9; -24 to 69) and 30.4 (31.2; -12 to 88), respectively. Similar results were noted for night pain.

Lateral epicondylitis. Buchbinder et al³³ reported a Cochrane review of shock-wave therapy for lateral elbow pain. They identified only two trials of ESWT *versus* placebo.^{34,35} Both studies included similar groups with chronic recalcitrant pain in the lateral elbow for more than six months and clinical signs of lateral epicondylitis. In one of the studies, Rompe et al^{34,35} compared 3000 shocks of 0.08 mJ/mm^2 using an electromagnetic generator, with $30 \times 0.08 \text{ mJ/mm}^2$ as the control in a total of 100 subjects. The study does not appear to have been blinded and it is unclear as to whether data were analysed on an intention-to-treat basis. A significant improvement in pain and function was noted only in the 'treatment' group when reviewed after three months.

The other study, by Haake et al,³⁶ was a double-blind multicentre randomised, controlled trial involving 270 patients who received either 2000 shocks at 0.07 to 0.09 mJ/mm^2 or a sham procedure in which a coil prevented transmission of the shock waves. Treatments were administered at weekly intervals for three weeks. Different devices for the production of shock waves were used at different treatment centres. No difference was noted between the treatment and placebo groups using the Roles and Maudsley subjective pain rating score and grip strength after six and 12 weeks, and after 12 months. The success rate was 25.8% in the ESWT group and 25.4% in the placebo group, the difference being 0.4% with a 95% confidence interval (CI) of -10.5 to +11.3. An improvement was observed in two-thirds of the patients in both groups 12 months after intervention.

We have subsequently reported similar findings to those of Haake et al,³⁶ in a double-blind randomised, controlled trial of ESWT in 75 adults who had recalcitrant lateral epicondylitis for more than three months with a mean duration of symptoms of 15.9 and 12 months in the ESWT and sham groups, respectively.³⁷ The subjects were randomised to receive either active treatment with an electromagnetic generator (1500 pulses of ESWT at 0.12 mJ/mm^2) or sham therapy, monthly for three months. All were assessed before each treatment and one month after completion of the course by visual analogue scores for pain during the day and at night. Both groups showed similar significant improvements from two months. In the ESWT group the mean (SD, range) pain score was 73.4 (14.5; 38 to 99) at the beginning and 47.9 (31.4; 3 to 100) at three months. In the sham group the mean (SD; range) pain score was 67.2 (21.7; 12 to 100) at the beginning and 51.5 (32.5; 3 to 100) at three months. After three months, there was an improvement of 50% in 35% of the ESWT group and in 34% of the sham group.

Plantar fasciitis. Shock-wave treatment has gained approval of the FDA for the treatment of recalcitrant plantar fasciitis in the USA³ on the basis of a double-blind multicentre randomised trial in 260 subjects with chronic plantar fasciitis of more than six months' duration.⁴ Outcome was assessed by investigator-rated pain on pressure using a dolorimeter, estimation by the subject of the time and distance of pain-free walking, evaluation of the use of analgesics and the patient rating of start-up pain. The treatment was by a single dose of ESWT at a high-energy level (1500 shocks at $>0.18 \text{ mJ/mm}^2$), using an electrohydraulic generator. The shock waves do not appear to have been focused and the heel was continually manipulated by the physician during the treatment session. The placebo involved a styrofoam block and a fluid-filled intravenous bag, without the use of coupling gel. Local anaesthesia was used, although this differed between the treatment and placebo groups. Data were not analysed on an intention-to-treat basis. Three months after the one treatment, 56% more of the patients who had been treated had a successful result by all four of the criteria of evaluation when compared with the patients treated with a placebo. Improvement in start-up pain was noted in 59.7% of the ESWT group and in 48.2% of the placebo group. Pain-free activity improved similarly in both groups and investigator-induced pain was better in 62.2% and 43% in the ESWT and placebo groups, respectively.

Cosentino et al³⁸ performed a single-blind randomised, controlled trial of ESWT in 60 subjects with chronic heel pain and heel spurs using an electrohydraulic generator and localised by ultrasound. The subjects were randomised to receive 1200 shocks of either a considerable energy range of 0.03 to 0.4 mJ/mm^2 or sham treatment (0 mJ/mm^2). Six treatments were administered at intervals of seven to ten days. Significant decreases in subjective pain at rest, on start-up and at the end of daily activities were reported at

the end of the course of treatment and at follow-up at one and three months. No change in the radiographic dimensions of heel spurs was noted.

In contrast to the results of these two studies, Buchbinder et al³⁹ performed a double-blind randomised, placebo-controlled trial of ultrasound-guided ESWT in 160 subjects with symptomatic proximal plantar fasciitis of at least six weeks' duration which had been demonstrated by ultrasound. Patients were randomly assigned to receive either ultrasound-guided ESWT weekly for three weeks to a total dose of at least 1000 mJ/mm² (n = 81), or a similar placebo to a total dose of 6.0 mJ/mm² (n = 85). After six and 12 weeks, there were significant improvements in overall pain in both the treated and placebo groups (mean (SD) improvement, 18.1 (30.6) and 19.8 (33.7) at six weeks (p = 0.74) for between-group difference and 26.3 (34.8) and 25.7 (34.9) at 12 weeks (p = 0.99), respectively). Similar improvements in both groups were also observed for morning and activity pain, walking ability, the Maryland foot score, problem elicitation technique, and the SF-36 score. There were no statistically significant differences in the degree of improvement between the two groups for any measured outcomes.

We performed a double-blind randomised, controlled trial of ESWT of moderate dose (1500 shocks at 0.12 mJ/mm²) or sham treatment, monthly for three months in 88 adults with chronic plantar fasciitis.⁴⁰ The sham treatment was similar to that used in the study of ESWT in lateral epicondylitis described above.³⁷ Both groups showed significant improvement over the course of the trial, but there was no statistically significant difference between them with respect to the changes seen in any of the outcome measures over the six-month period. After three months, 37% of the subjects in the ESWT group and 24% in the sham group showed a positive response as measured by an improvement of 50% with respect to pain during the day. Positive responses with night pain occurred in 41% and 31% in the ESWT and sham groups, respectively.

Positive responses in start-up pain occurred in 37% and 36% in the ESWT and sham groups, respectively.

In another study, Rompe et al⁴¹ performed a prospective single-blind randomised, controlled trial of low-dose ESWT (1000 shocks of 0.06 mJ/mm²) or sham treatment administered weekly for three weeks in 30 patients with chronic plantar fasciitis which had persisted for at least 12 months. The sham treatment involved no contact with skin and the absence of gel. A significant improvement in pain and function was noted only in the ESWT group after three months, but six subjects withdrew and the data were not analysed on an intention-to-treat basis.

Interpreting the literature. There are considerable differences between the results of various studies which have assessed the efficacy of ESWT in soft-tissue conditions. These may be explained by a number of factors, which may be grouped into technical, the subject populations, variations in the severity of the disease and the design of the study.

Technical aspects include different designs of machine and the heterogeneity of factors such as shock-wave intensity, focal energy, geometry of the shock-wave focus, frequency of treatment and the use of different forms of 'sham therapy'. Different machines and generators may well have dissimilar effects because of differences in the shock waves produced. While the intensity of treatment delivered by some machines necessitates the use of local anaesthesia, others do not require this. The accuracy of localisation of treatment is also a potential source of variation, and is open to operator error, although this has not been evaluated extensively.

Differences in study populations, and in particular the duration of symptoms, may also play a role, as may the specific soft-tissue condition which is being treated. The degenerative tennis elbow may have a true difference in response to that of calcific conditions or to chronic plantar fasciitis. Soft-tissue conditions may be mimicking another complaint (e.g. referred pain presenting as lateral epicondylitis), and for this reason studies which include conditions proven by imaging would improve the quality of randomised controlled trials. The design of the study is also important. The assessment of various management approaches in soft-tissue conditions is often limited by small sample sizes, heterogeneous study populations, surrogate outcome measures, inadequate blinding and inappropriate sham therapies. A pre-assessment period helps to ensure that symptoms are stable, in order to exclude natural history as a source of improvement.⁴²⁻⁴⁴ The use of different outcome measures can also prevent direct comparisons between studies. Measurement of different outcomes gives different and complementary information and all have a role to play in the evaluation of a treatment. While the literature is abundant with non-randomised controlled studies of ESWT there remains an inadequate number of randomised, controlled trials examining technical factors and dosage.

ESWT is a potentially helpful addition to the options for the management of soft-tissue conditions. Currently, there is evidence of benefit from some regimes of ESWT in calcific tendonitis of the rotator cuff and chronic plantar fasciitis. However, there is a need for further proper research into the effects of ESWT on soft tissues, for randomised, controlled trials in other soft-tissue conditions and investigation of the technical factors relating to the treatment and optimal regimes of dosage in specific conditions.

References

1. **Schleberger R, Senge T.** Non-invasive treatment of long-bone pseudarthrosis by shock waves (ESWL). *Arch Orthop Trauma Surg* 1992;111:224-7.
2. **Vachalnou VD, Michailov P.** High energy shock waves in the treatment of delayed and nonunion of fractures. *Int Orthop* 1991;15:181-4.
3. **Henney JE.** From the food and drug administration. *JAMA* 2000;284:2711.
4. **Ogden JA, Alvarez R, Levitt R, Cross GL, Marlow M.** Shock wave therapy for chronic proximal plantar fasciitis. *Clin Orthop* 2001;387:47-59.
5. **Wess O, Ueberle F, Durhben RN, et al.** Working group technical developments: consensus report in high energy shock waves in medicine. In: Chaussy C, Eisenberger F, Jochem D, Wilbert D, eds. *High energy shock waves in medicine*. Thieme Stuttgart, 1997.

6. Auge BK, Preminger GM. Update on shock wave lithotripsy technology. *Curr Opin Urol* 2002;12:287-90.
7. Coleman AJ, Saunders JE. A review of the physical properties and biological effects of the high amplitude acoustic fields used in extracorporeal lithotripsy. *Ultrasonics* 1993;31:75-89.
8. Coleman AJ, Saunders JE. A survey of acoustic output of commercial extracorporeal shock wave lithotripters. *Ultrasound Med Biol* 1989;15:213-27.
9. Delius M. Medical applications and bioeffects of extracorporeal shock waves. *Shock Waves* 1994;4:55-72.
10. Haake M, Boddeker IR, Decker T, et al. Side-effects of extracorporeal shock wave therapy (ESWT) in the treatment of tennis elbow. *Arch Orthop Trauma Surg* 2002;122:222-8.
11. Sistermann R, Katthagen BD. Complications, side effects and contraindications in the use of medium and high-energy extracorporeal shock waves in orthopaedics. *Z Orthop Ihre Grenzgeb* 1998;136:175-81.
12. Coleman AJ, Saunders JE, Crum LA, Dyson M. Acoustic cavitation generated by an extracorporeal lithotripter. *Ultrasound Med Biol* 1987;13:69-76.
13. Delius M, Draenert K, Draenert Y, Börner M. Effect of extracorporeal shock waves on bone: a review of shock wave experiments and the mechanism of action of shock wave action. In: Sibert W, Buch M, eds. *Extracorporeal shock waves in orthopaedics*. Berlin: Springer-Verlag, 91-108.
14. Munver R, Delvecchio FC, Kuo RL, et al. In vivo assessment of free radical activity during shock wave lithotripsy using a microdialysis system: the renoprotective action of allopurinol. *J Urol* 2002;167:327-34.
15. Brummer F, Suhr D, Hulser D. Sensitivity of normal and malignant cells to shock waves. *J Stone Dis* 1992;4:243-8.
16. Gambihler S, Delius M, Brendel W. Biological effects of shock waves: cell disruption, viability, and proliferation of L1210 cells exposed to shock waves in vitro. *Ultrasound Med Biol* 1990;16:587-94.
17. Brauner T, Brummer F, Hulser DF. Histopathology of shock wave treated tumour cell suspensions and multicell tumour spheroids. *Ultrasound Med Biol* 1989;15:451-60.
18. Russo P, Mies C, Huryk R, Heston WDW, Fair WR. Histopathologic and ultrastructural correlates of tumour growth suppression by high energy shock waves. *J Urol* 1987;137:338-41.
19. Kohri K, Iguchi M, Kurita T. Effect of high energy shock waves on tumour cells. *Urol Res* 1990;18:101-5.
20. Steinbach P, Hofstadter F, Nicolai H, Rossler W, Wieland W. In vitro investigations on cellular damage induced by high energy shock waves. *Ultrasound Med Biol* 1992;18:691-9.
21. Haupt G, Chvapil M. Effect of shock waves on the healing of partial-thickness wounds in piglets. *J Surg Res* 1990;49:45-8.
22. Ortham Z, Alper M, Akman Y, Yavuz O, Yalciner A. An experimental study on the application of extracorporeal shock waves in the treatment of tendon injuries: a preliminary report. *J Orthop Sci* 2001;6:566-70.
23. Wang C-J, Huang H-Y, Pai C-H. Shock wave-enhanced neovascularisation at the tendon-bone junction: an experiment in dogs. *J Foot Ankle Surg* 2002;41:16-22.
24. Rompe JD, Kirpatrick CJ, Kullmr K, Schwitalle M, Krschek O. Dose related effects of shock waves on rabbit tendon achillis: a sonographic and histological study. *J Bone Joint Surg [Am]* 1998;80-A:546-52.
25. Hartman C, Child SZ, Mayer R, Schenk E, Carstensen EL. Lung damage from exposure to the fields of an electrohydraulic lithotripter. *Ultrasound Med Biol* 1990;16:675.
26. Dalecki D, Keller BB, Carstensen E, et al. Thresholds for premature ventricular contraction in frog hearts exposed to lithotripter fields. *Ultrasound Med Biol* 1991;17:341-6.
27. Delius M, Hoffmann E, Steinbeck G, Conzen P. Biological effects of shock waves: induction of arrhythmia in piglet hearts. *Ultrasound Med Biol* 1994;20:279-85.
28. Filipczynski L, Piechoki M. Estimation of the temperature increase in the focus of a lithotripter for the case of high rate administration. *Ultrasound Med Biol* 1990;16:149-56.
29. Schelling G, Delius M, Gschwender M, Grafe P, Gambihler S. Extracorporeal shock waves stimulate frog sciatic nerves indirectly via a cavitation mediated mechanism. *Biophys J* 1994;66:133-40.
30. Loew M, Daecke W, Kusnierczak D, Ranmauzaden M, Ewerbeck V. Shock-wave therapy is effective for chronic calcifying tendinitis of the shoulder. *J Bone Joint Surg [Br]* 1999;81-B:863-7.
31. Schmitt J, Haake M, Tosch A, et al. Low-energy extracorporeal shock wave treatment (ESWT) for tendinitis of supraspinatus: a prospective randomised study. *J Bone Joint Surg [Br]* 2001;83-B:873-6.
32. Speed CA, Richards C, Nichols D, et al. Extracorporeal shock-wave therapy for tendinitis of the rotator cuff: a double-blind, randomised, controlled trial. *J Bone Joint Surg [Br]* 2002;84-B:509-12.
33. Buchbinder R, Green S, White M, et al. Shock wave therapy for lateral elbow pain (Cochrane Review). In: The Cochrane Library, Issue 3, 2002. Oxford Update Software.
34. Rompe JD, Hopf C, Kullmer K, et al. Low energy extracorporeal shock wave therapy for persistent tennis elbow. *Orthop* 1996;20:23-7.
35. Rompe JD, Hopf C, Kullmer K, Heine J, Burger R. Analgesic effect of extracorporeal shock wave therapy on chronic tennis elbow. *J Bone Joint Surg [Br]* 1996;78-B:233-7.
36. Haake M, König IR, Decker T, et al. No effectiveness of extracorporeal shock wave therapy in the treatment of tennis elbow: results from a prospective randomized placebo-controlled multicenter trial. *J Bone Joint Surg [Am]* 2002;84-A:1982-91.
37. Speed CA, Nichols D, Richards C, et al. Extracorporeal shock wave therapy for lateral epicondylitis: a double blind randomised controlled trial. *J Orthop Res* 2002;20:895-8.
38. Cosentino R, Falsetti P, Manca S, et al. Efficacy of extracorporeal shock wave treatment in calcaneal entheophytosis. *Ann Rheum Dis* 2001;60:1064-7.
39. Buchbinder R, Ptasznik R, Gordon J, et al. Ultrasound-guide extracorporeal shock wave therapy for plantar fasciitis: a randomized controlled trial. *JAMA* 2002;288:1364-72.
40. Speed CA, Nichols D, Richards C, et al. Extracorporeal shock wave therapy for plantar fasciitis: a double randomised controlled trial. *J Orthop Res* 2003;in press.
41. Rompe JD, Hopf C, Nafe B, Burger R. Low-energy extracorporeal shock wave therapy for painful heel: a prospective controlled single-blind study. *J Orthop Trauma Surg* 1996;115:75-9.
42. Green S, Buchbinder R, Glazier R, Forbes A. Systematic review of randomised controlled trials of interventions for painful shoulder: selection criteria, outcome assessment, and efficacy. *BMJ* 1998;316:354-60.
43. Speed CA, Hazleman BL. Shoulder pain. *Clin Evi* 2002;7:1122-39.
44. Crawford F. Plantar heel pain and fasciitis. *Clin Evi* 2003;9:1327-38.