Extracorporeal Shock Wave Therapy without Local Anesthesia for Chronic Lateral Epicondylitis

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Background: The use of extracorporeal shock wave therapy for the treatment of lateral epicondylitis is controversial. The purpose of this study was to evaluate the use of extracorporeal shock wave therapy without local anesthesia to treat chronic lateral epicondylitis.

Methods: One hundred and fourteen patients with a minimum six-month history of lateral epicondylitis that was unresponsive to conventional therapy were randomized into double-blind active treatment and placebo groups. The protocol consisted of three weekly treatments of either low-dose shock wave therapy without anesthetic or a sham treatment. Patients had a physical examination, including provocation testing and dynamometry, at one, four, eight, and twelve weeks and at six and twelve months after treatment. Radiographs, laboratory studies, and electrocardiograms were also evaluated prior to participation and at twelve weeks. A visual analog scale was used to evaluate pain, and an upper extremity functional scale was used to assess function. Crossover to active treatment was initiated for nonresponsive patients who had received the placebo and met the inclusion criteria after twelve weeks.

Results: A total of 108 of the 114 randomized patients completed all treatments and the twelve weeks of follow-up required by the protocol. Sixty-one patients completed one year of follow-up, whereas thirty-four patients crossed over to receive active treatment. A significant difference ($p = 0.001$) in pain reduction was observed at twelve weeks in the intent-to-treat cohort, with an improvement in the pain score of at least 50% seen in 61% (thirty-four) of the fifty-six patients in the active treatment group who were treated according to protocol compared with 29% (seventeen) of the fifty-eight subjects in the placebo group. This improvement persisted in those followed to one year. Functional activity scores, activity-specific evaluation, and the overall impression of the disease state all showed significant improvement as well ($p < 0.05$). Crossover patients also showed significant improvement after twelve weeks of active treatment, with 56% (nineteen of thirty-four) achieving an improvement in the pain score of at least 50% ($p < 0.0001$).

Conclusions: These results demonstrate that low-dose shock wave therapy without anesthetic is a safe and effective treatment for chronic lateral epicondylitis.

Level of Evidence: Therapeutic Level I. See Instructions to Authors for a complete description of levels of evidence.

Lateral epicondylitis is a common orthopaedic symptom. Initially, nonoperative management is effective in most patients; however, studies on the natural history and surgical management of the disorder have shown that surgical intervention is necessary in 4% to 11% of patients[1-3]. While surgical intervention is often successful, it carries with it pain, risks, and costs that may be avoided with successful nonoperative management.

Despite the widespread use of extracorporeal shock wave therapy in the treatment of lateral epicondylitis, there is a lack of evidence-based support of its efficacy[4-6]. The mechanism of action for shock wave therapy remains uncertain, but it includes the possible stimulation of the healing process in damaged tendons by disrupting avascular, damaged tissues and encouraging revascularization, release of local growth factors, and the recruitment of appropriate stem cells to the area[7,8]. Another proposed mechanism for its efficacy is hyperstimulation analgesia, where, through brief sensory stimulation, shock wave therapy can provide long-term pain relief[9,10]. Alteration of chemical mediators of pain, modulation of the pain signal, and disruption of cell membranes have all been proposed as possible generators of this analgesic effect[7,8,10]. Several structured studies of the treatment of lateral epicondylitis have failed to find a significant benefit from extracorporeal shock wave therapy[11-15]. The present study was designed to evaluate the efficacy of extracorporeal shock wave therapy

without local anesthesia in the treatment of chronic lateral epicondylitis. To date, no double-blind, placebo-controlled, multicenter study of shock wave therapy without local anesthesia in a general population with lateral epicondylitis refractory to traditional conservative management has been published as far as we know. The null hypothesis of the present study was that shock wave therapy has no effect on lateral epicondylitis.

Materials and Methods

A total of 114 patients from three large orthopaedic practices were evaluated for inclusion in the study. The study design was a randomized, multicenter, double-blind, placebo-controlled parallel treatment protocol. Patients were initially screened for inclusion and then were randomized into an active treatment group (fifty-six patients) or a placebo treatment group (fifty-eight patients). At randomization, each patient was given a unique study number and a sealed envelope with his or her study number on it. The sealed envelope contained a randomization code (A or B), which was only opened by the shock wave operator and was not shared with anyone else involved in the study. All patients provided informed consent, and the study was approved by each participant's institutional review board. Patients were not charged for treatment during the study.

Active treatment consisted of one treatment each week with 2000 impulses at 0.06 mJ/mm^2 with use of the Sonocur extracorporeal shock wave therapy system (Siemens Medical Solutions USA, Iselin, New Jersey) for three weeks. The treatment head of the device, which measures 11 × 12 cm and produces a treatment area that is 6 × 6 mm and 58 mm in depth, was directed to the point of maximal tenderness on the lateral epicondyle as identified by physician palpation and patient report. An ultrasound coupling gel was used. During treatment, the technique of so-called clinical focusing was used by adjusting the shock wave focus every 200 to 400 impulses and redirecting the shock waves to the most symptomatic site. Placebo treatment consisted of sham treatments of 2000 impulses at 0.06 mJ/mm^2 but with use of a sound-reflecting pad between the patient and the application head of the machine. Patients were treated in isolation by a technician, so that no patient could view another's treatment. Both the patients and the evaluating physicians were blinded to the treatment assignment, and only the technician knew the treatment group. No local anesthesia or other injections were used. Patients were evaluated prior to treatment and at follow-up examinations at one, four, eight, and twelve weeks and at six and twelve months after completion of treatment. Follow-up examiners were not aware of the patient's treatment group unless the patient had become unblinded during crossover treatment.

The primary efficacy end point was relief of pain elicited by provocative Thomsen testing and recorded on a visual analog scale at twelve weeks compared with baseline. Patients who did not respond to their assigned treatment with at least a 50% reduction in pain from baseline after twelve weeks could have their treatment group revealed to them. If the patient had received placebo treatment and still met the inclusion criteria, he or she could be crossed over into the active treatment group. These crossover patients were then followed for twelve weeks after treatment to evaluate their response, but they were considered lost to the placebo group. If the patient had been in the active treatment group and treatment failed after twelve weeks, other standard therapies could be considered according to the physician and standard medical practice.

The inclusion criterion for participation in the study was a history of lateral epicondylitis for a minimum of six months with pain that was resistant to at least two of three conventional therapies. These included more than four weeks of physical or occupational therapy, use of nonsteroidal anti-inflammatory medications for more than four weeks, and corticosteroid injections. Patients also had to have tenderness on palpation of the lateral epicondyle and reproducible pain provoked by resisted wrist extension (the Thomsen test) of ≥40 mm on the 100-mm visual analog scale.

The Thomsen test was performed with the shoulder flexed to 60°, the elbow extended, the forearm pronated, and the wrist extended 30°. Pressure was applied on the dorsum of the hand to stress the extensor carpi radialis and brevis. The test was performed three times, with the patient recording the pain on the 100-mm visual analog scale after the third test.

The exclusion criteria were an age of less than eighteen years, a history of a lateral elbow injection within the prior six weeks, physical therapy within the prior four weeks, and the use of nonsteroidal anti-inflammatory medications or acetaminophen for any reason within one week prior to the study. In addition, active bilateral epicondylitis, treatment with systemic therapeutic anticoagulation, or a history or radiographic findings of cervical spondylosis, upper extremity arthritis, elbow arthritis, a neurologic abnormality, rheumatoid disease, or radial nerve entrapment were criteria for exclusion. Patients receiving Workers' Compensation as well as those who had prior surgery for lateral epicondylitis or those with severe systemic disease or who were pregnant were also excluded.

The study population consisted of sixty women and fifty-four men with a mean duration of symptoms of twenty-one months prior to participation. The average age was forty-seven years. The right arm was affected in 67% (seventy-six patients), and the left arm was involved in 33% (thirty-eight patients). Of the 114 patients who met the inclusion criteria (failure of treatment with injections, nonsteroidal anti-inflammatory medications, and physical therapy), eighty-four (74%) had failed all three treatments. Overall, for 92% (105) of the patients, steroid injections failed to relieve the pain. No significant differences were detected between the placebo and active treatment groups in terms of demographic characteristics such as age, race, gender, body habitus, affected arm, chronicity of pain, medical diagnoses, or prior treatments.

The initial evaluation of the patients consisted of a thorough history and physical examination, as well as baseline studies consisting of complete blood-cell count with differential, electrocardiogram, and anteroposterior and lateral radiographs of the affected arm. Radiographs were reviewed by the

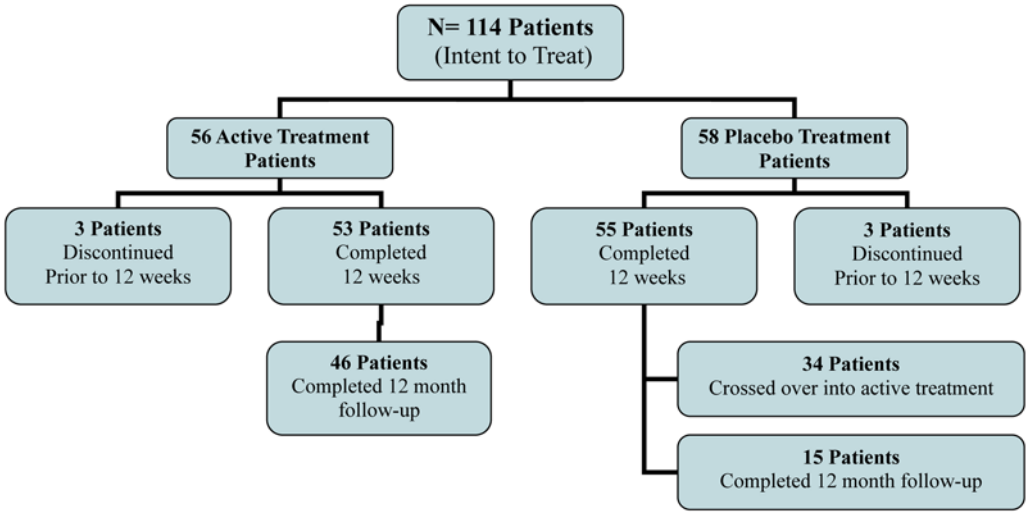

Fig. 1
Patient disposition.

treating physicians to evaluate any concomitant abnormality or possible exclusion criteria. These studies were then repeated at twelve weeks to monitor any potential change related to treatment with the shock wave device.

Prior to treatment and at one, four, eight, and twelve weeks and at six months and one year after treatment, each patient had a clinical evaluation to assess the symptoms. The evaluation consisted of Thomsen provocation testing, functional assessment with the upper extremity functional scale[16] (Table I), and a subjective evaluation of the disease status by the patient. In addition to the clinically validated functional score[16] calculated with subjective reports of the activities in Table I, a patient-specific activity score was determined for each patient at each follow-up visit as well. To calculate this activity score, patients rated their ability to perform two patient-identified activities that they found particularly difficult to do on a scale from 1 (no difficulty) to 10 (cannot perform). Grip strength was also evaluated with dynamometry at each follow-up visit through twelve weeks. Patients were queried with regard to these activities and any adverse effects at each follow-up visit.

TABLE I Activities Rated on the Upper Extremity Functional Scale*

Sleeping
Writing
Opening jars
Picking up small objects with fingers
Driving more than 30 minutes
Opening a door
Carrying a milk jug from the refrigerator
Washing dishes

*The ability of the patient to perform each activity was rated on a scale of 1 to 10, with 1 indicating no difficulty and 10, the activity cannot be performed.

Statistical Methods

The primary efficacy end point was a 50% reduction in the provocation in pain on the Thomsen test. On the basis of previous experience with extracorporeal shock wave therapy, we assumed a response rate of 80% for patients in active treatment and 50% for patients in the placebo group. With use of this information, it was determined that a sample size of forty-five patients per treatment group would have an 80% power in detecting the treatment difference with a two-sided significance level of 0.05. Assuming a retention rate of at least 80%, a total of 114 patients were then recruited into the study. To demonstrate a difference between the two treatments for the primary efficacy end point, a Fisher exact test was used. Missing responses were input as the last recorded value carried forward for intent-to-treat calculations.

On the basis of pilot study results, the power of a sample size of forty-five patients for the mean upper extremity functional score would be >90%. The analysis of variance test was used for the functional score difference and other continuous variables. Statistical significance was set for $p \leq 0.05$ for all outcome parameters.

Results

One hundred and fourteen patients were randomized into the placebo group (fifty-eight patients) and the active treatment group (fifty-six patients). In the active treatment group, fifty-three patients completed the twelve-week protocol requirements, two patients were unable to complete the requirements because of intolerance of the treatment, and one patient withdrew from the study because of thrombocytopenia, which had been documented prior to study enrollment. Fifty-five patients in the placebo group completed the twelve-week protocol requirements, and three patients withdrew from

TABLE II Efficacy Outcomes

	Active Treatment Group				Placebo Group				
	No. of Patients	Baseline*	12 Weeks*	Change†	No. of Patients	Baseline*	12 Weeks*	Change†	P Value‡
Pain	56	74 ± 15.8	37.6 ± 28.7	49%	58	75.6 ± 16.0	51.3 ± 29.7	32%	0.02
Functional scale	53	4.7 ± 1.8	2.3 ± 1.6	51%	54	4.6 ± 1.8	3.2 ± 2.1	30%	0.01
Activity score	52	7.7 ± 1.3	3.5 ± 2.2	55%	54	7.4 ± 1.2	5.0 ± 2.6	32%	0.0002
Overall impression	53	70.3 ± 16.0	32.8 ± 27.7	53%	54	66.0 ± 16.9	46.2 ± 28.11	30%	0.0013
Grip strength *(lb [kg])*	53	71 ± 26.3 (32 ± 12)	87.1 ± 10 (4.0 ± 5)	23%	54	72.5 ± 29.5 (32.9 ± 13)	81.5 ± 32.5 (37.0 ± 15)	12%	0.09

*The values are given as the mean and the standard deviation. †The change was calculated as the improvement from baseline within the treatment group. ‡P values were calculated for the difference between the groups.

the study to seek alternative treatment by twelve weeks. Additionally, thirty-four patients left the placebo group after twelve weeks to cross over into the active treatment protocol (Fig. 1).

At twelve weeks, a significant difference between treatment groups ($p = 0.001$) was observed with respect to pain reduction on the Thomsen test, with reduction in pain of at least 50% achieved in 61% (thirty-four) of the fifty-six patients in the active treatment group who were treated according to protocol compared with only 29% (seventeen) of the fifty-eight patients in the placebo group. The average pain score for the active treatment group decreased from 74 at baseline to 38 at twelve weeks on the 100-mm visual analog scale compared with a decrease from 76 to 51, respectively, for the placebo group. The difference between the groups with respect to the mean pain scores was significant ($p < 0.024$) (Fig. 2).

The mean improvement in the upper extremity functional scores at twelve weeks was 2.4 (from 4.7 to 2.3) for the active treatment group compared with 1.4 (from 4.6 to 3.2) for the placebo group; the difference was significant ($p < 0.01$). A significant improvement in the patient activity scores was also seen at twelve weeks, with a mean improvement of 4.2 (from 7.7 to 3.5) in the active treatment group compared with 2.4 (from 7.4 to 5.0) in the placebo group ($p = 0.0002$).

The mean improvement in grip strength was 14.6 lb (6.6 kg) (from 71 to 87.1 lb [32.2 to 38.2 kg]) in the active treatment group at twelve weeks compared with 8.6 lb (3.9 kg) (from 72.5 to 81.5 lb [32.9 to 37.4 kg]) in the placebo group. However, this difference was not significant ($p = 0.09$).

The rating of the overall impression of their disease state by the patients in the active treatment group improved significantly from 70.3 at baseline to 32.8 at twelve weeks. Compared with the ratings of the placebo group, the difference was significant ($p = 0.0013$) (Table II).

At six months, a similar improvement was seen among the forty-seven patients in the active treatment group, some of whom had been unblinded after twelve weeks. Thirty-five patients in the group (twenty-eight of whom remained blinded) had maintained at least a 50% reduction in pain at that time. With the patients who were not evaluated included as treatment failures, the rate of patients who maintained the level of

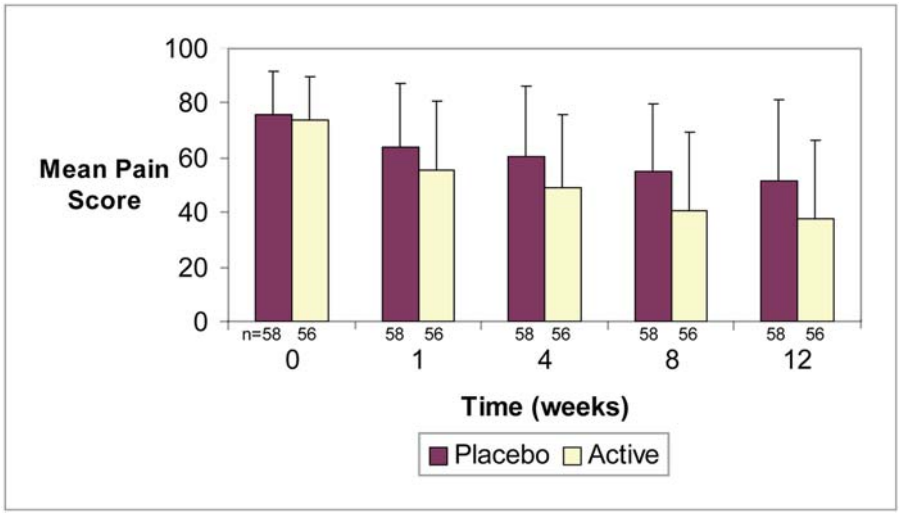

Fig. 2

Pain assessment to twelve weeks.

pain relief was 63% (thirty-five) of the fifty-six patients. When only those who remained blinded were considered, the rate was 50% (twenty-eight patients). Only one patient who had achieved a 50% reduction in pain at twelve weeks was found not to have maintained this level of relief at six months. The mean pain score in the active treatment group at six months was 24, with a median score of 8. The functional scores in the active treatment patients had also improved at six months, with a mean improvement of 2.8 (from 4.7 to 1.9).

By six months, most patients in the placebo group had been lost to crossover and were therefore not available for comparison. Of the sixteen placebo patients who had not crossed over and were seen at six months, thirteen had achieved a 50% reduction in pain. However, this represents only 22% (thirteen) of the fifty-eight patients in the intent-to-treat placebo cohort. The mean pain score in the placebo group at six months was 18, with a median score of 6.

At one year, forty-six patients in the active treatment group were evaluated again. Forty-three (93%) reported at least a 50% reduction in pain. With those from the intent-to-treat cohort who were not evaluated included as treatment failures, the rate of patients who had achieved and maintained at least a 50% reduction in pain was 81% (forty-three of fifty-three patients). The mean pain score was 10, with a median score of 4. Of the fifteen patients in the placebo group who had not crossed over and were seen at one year, all fifteen had achieved a 50% reduction in pain. This, however, represents only 26% (fifteen) of the fifty-eight patients in the original placebo cohort.

Thirty-four patients from the placebo group crossed over after twelve weeks to receive active treatment. All patients were nonresponsive to placebo treatment and fulfilled the same criteria for inclusion as at the beginning of the study. These patients were then given the same active treatment protocol weekly for three weeks and were followed for twelve weeks, according to the protocol previously outlined. Eight patients (24%) dropped out prior to completing the twelve-week follow-up evaluation. The reasons for dropping out were an inability to tolerate treatment (one patient), failure to return for follow-up (one patient), patient request (five patients), and relocation from the region (one patient).

The mean pain score for the crossover patients before they began active treatment was significantly lower than it had originally been at the start of placebo treatment ($p = 0.034$). During the placebo phase, the mean pain score had changed from 78 before placebo treatment to 70 before active treatment; whereas during active treatment, the mean pain score had decreased from 70 to 28 at twelve weeks. At all visits during active treatment, the crossover patients had significantly lower pain scores than those during their corresponding placebo treatments ($p = 0.0339$ at baseline, $p = 0.0027$ at week 1, and $p < 0.0001$ at weeks 4, 8, and 12) (Fig. 3). For the primary efficacy analysis, a reduction in pain of at least 50% was achieved by 56% (nineteen) of the thirty-four crossover patients compared with 0% during the placebo phase. The mean functional scores with active treatment also improved. During the twelve-week placebo period, they had improved from 4.7 to 4.0, and, after twelve weeks with active treatment, they improved from 3.9 to 1.98. The difference between the two treatment groups (the original placebo patients and the same patients who crossed over to active treatments) with respect to this improvement did not reach significance ($p = 0.25$); however, within the active treatment group, the change from baseline was significant ($p < 0.0001$).

Adverse Effects

No changes from baseline were seen in either the active treat-

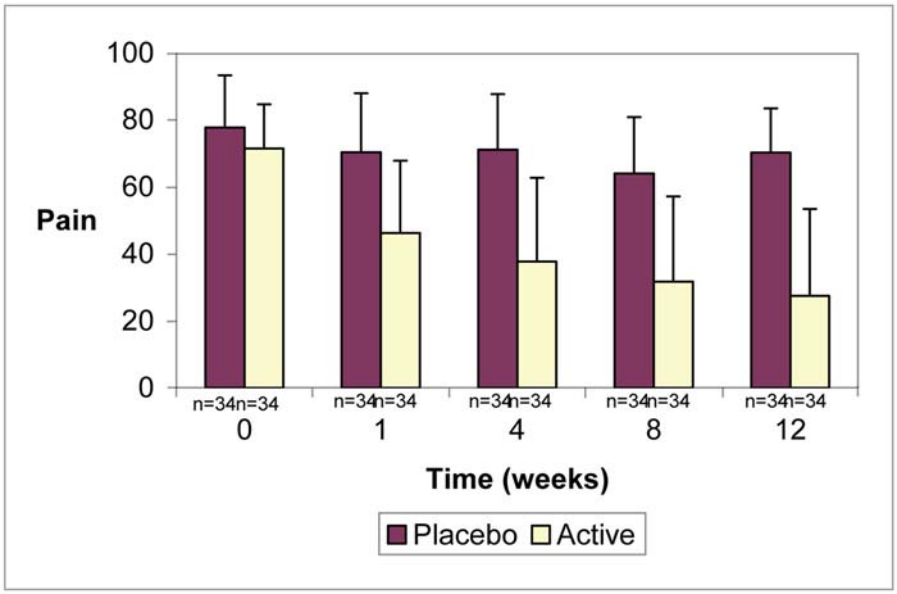

Fig. 3

Summary of pain data for crossover patients.

TABLE III Device-Related Adverse Events at the Twelve-Week Follow-up Evaluation

	Active Treatment Group			Placebo Group		
	No. of Patients*	No. of Occurrences	Percentage of Patients†	No. of Patients*	No. of Occurrences	Percentage of Patients†
Pain	28	60	50	13	32	22
Nausea	10	10	18	0	0	0
Local reaction	6	8	11	5	5	9
Sweating	5	5	9	0	0	0
Dizziness	4	4	7	0	0	0
Hypertonia	3	5	5	3	3	6
Hypesthesia	3	5	5	1	2	2
Paresthesia	3	4	5	8	12	14
Joint stiffness	2	2	4	0	0	0
Myalgia	2	2	4	0	0	0
Tremor	2	2	4	0	0	0
Vasodilation	2	2	4	0	0	0
Pallor	1	1	2	0	0	0
Accidental injury	0	0	0	2	3	3
Headache	0	0	0	2	7	3
Peripheral edema	0	0	0	1	1	2
Twitching	0	0	0	1	1	2
Sinusitis	0	0	0	1	2	2

*The number of patients who had the adverse event. †Based on the intent-to-treat cohort, with fifty-six in the active treatment group and fifty-eight in the placebo group.

ment group or the placebo group with respect to the hematologic, radiographic, or electrocardiographic studies obtained at baseline and at the twelve-week follow-up examination. No serious adverse effects from the device were found. Twenty-eight patients (50%) in the active treatment group compared with thirteen patients (22%) in the placebo group experienced moderate treatment-related pain that was transient. Ten patients (18%), all in the active treatment group, experienced nausea during treatment. Two active treatment patients had to stop treatment sessions prior to receiving the full 2000 impulses because of these symptoms. One withdrew from the study, and one was able to resume and tolerate the treatment later. One other active treatment patient withdrew because of pain and a slight tremor in the treated arm after completing the first treatment. No lasting adverse effects were noted, and all of these effects had resolved by the final follow-up evaluation. A full list of the adverse events probably or possibly related to the shock wave treatment is provided in Table III.

Discussion

In this study, we found extracorporeal shock wave therapy to be effective in the treatment of chronic lateral epicondylitis that had been refractory to other nonoperative treatment modalities. To our knowledge, this is the only double-blind, placebo-controlled multicenter trial of shock wave therapy for lateral epicondylitis to show efficacy in a general population. Active treatment resulted in significant improvement compared with placebo with respect to the reduction of pain, functional scores, patient activity score, and subjective rating of the disease state by the patient at twelve weeks. While grip strength improvement with active treatment was not significantly different from that with placebo treatment, the functional score, a validated measure of upper extremity function that combines activities requiring power grip and wrist and/or finger extension, showed a significant improvement. The improvements gained by active treatment were maintained in almost all of the patients who were followed for twelve months. Furthermore, the placebo patients who had not responded and crossed over to active treatment showed significant improvement compared with their own scores during placebo treatment.

The support for the use of shock wave therapy for lateral epicondylitis in the literature has been highly questionable[6,9,10,17-19]. In a meta-analysis of extracorporeal shock wave therapy in the musculoskeletal system, Ogden et al. reviewed the cases of more than 8000 patients with a wide variety of musculoskeletal conditions[5]. For lateral epicondylitis, the published and abstracted studies involved 1672 patients. Eleven prospective studies identified in that meta-analysis, which were not blinded or did not have control groups, described

success rates of 48% to 72%. In contrast, several recent high-quality, prospective, randomized trials of extracorporeal shock wave therapy did not find similar results and concluded that there was no benefit to treatment over placebo[11-15].

Speed et al. assessed moderate-dose shock wave therapy for patients with lateral epicondylitis of at least three months' duration in a double-blind, randomized, placebo-controlled trial and found that the success rates for active shock wave treatment (35%; fourteen of forty patients) and placebo (34%; twelve of thirty-five patients) were not significantly different[12]. The dosing protocol used in that study was moderate-dose shock wave therapy (1500 impulses at 0.12 mJ/mm^2) given at monthly intervals for three months. The dose and dosing interval are different from the weekly application of low-dose (2000 impulses at 0.06 mJ/mm^2) shock wave therapy used in our study and in most other trials of shock wave therapy. Despite the higher individual doses given in their monthly regimen, the total energy delivered per month was one-half of that delivered in the current study. As the histologic response of tissue treated with shock wave therapy is a dose-dependent phenomenon, this may have influenced the findings of their study[8,18].

Similarly, Melikyan et al. evaluated higher-energy shock wave therapy without local anesthesia for the treatment of lateral epicondylitis in a randomized, double-blind study[14]. The patients were treated with a single fractionated dose of 1000 mJ/mm^2 of shock wave therapy split over three sessions of 333 mJ/mm^2 each. The authors found no significant benefit to shock wave treatment over placebo in any parameter including the Disabilities of the Arm, Shoulder and Hand score, pain, grip strength, analgesic use, or subsequent surgical rate. That study differs from the current study in many ways. The energy level and dosing protocol are different, and the concomitant use of nonsteroidal anti-inflammatory medications was allowed. These medications inhibit the efficacy of extracorporeal shock wave therapy[9,18]. All patients in that study were already awaiting surgery, were treated at a single institution, and had been selected for inclusion by a single surgeon, which may have been a source of bias.

Chung and Wiley recently evaluated shock wave therapy as a primary treatment for previously untreated lateral epicondylitis in a double-blind, placebo-controlled trial[15]. The success rate in the active treatment group (39%; twelve of thirty-one patients) was not significantly higher than that in the placebo group (31%; nine of twenty-nine subjects). They used a treatment protocol that was similar to ours, but the study group was much smaller and the patients had a shorter duration of follow-up and had not previously been treated for epicondylitis. The study protocol also allowed concurrent use of nonsteroidal anti-inflammatory medications as well as a stretching program, both of which could confound their conclusions.

Haake et al. evaluated low-dose extracorporeal shock wave therapy with local anesthesia in a randomized, double-blind, placebo-controlled trial[13]. In that large study, no difference between shock wave therapy and sham treatment was noted for any of the treatment efficacy end points. Both the active treatment and placebo groups showed equal success rates at 25.8% (thirty-two of 124 patients) and 25.4% (thirty-one of 122 patients), respectively. These results are similar to the success rate spontaneously achieved in our placebo group, in which 29% (seventeen of fifty-eight patients) attained a 50% reduction in pain. While that study and ours are both multicenter, randomized, double-blind, placebo-controlled trials with similar inclusion and exclusion criteria and dosing protocols, the study by Haake et al. used local anesthesia injected at the treatment site. The use of a local anesthetic may alter the tissue effect of the shock wave therapy, interfere with hyperstimulation analgesia, or simply inhibit the aiming of the treatment head at the point of maximal tenderness. Regardless, this confounding factor was not used in our study and may be a reason for the very different outcomes.

Rompe et al. recently reported the results of a treatment protocol nearly identical to that used in the current study but limited to patients with epicondylitis secondary to playing tennis[18]. While smaller in numbers, the results are very similar to our findings with success in twenty-five (65%) of the thirty-eight patients in the active treatment group compared with eleven (28%) of the forty patients in the placebo group. Unlike our study, that study was done at a single center, was limited to tennis-related disease, and did not use a crossover design.

The decision not to use local anesthesia in our study, while absolutely needed to eliminate its possible influence on the effect of shock wave therapy and to allow clinical refocusing, may have biased the efficacy of our blinding to some extent. Although the patients were treated in isolation and did not know whether the treatment should be uncomfortable, a sham treatment with a reflecting pad is a slightly different experience than active treatment. This may be inferred by the higher rate of transient local pain with active treatment (50%) compared with placebo (22%). However, half of the active treatment patients did not experience pain with treatment, indicating that their experience was not radically different from that of most of the placebo group.

Other authors have found that a substantial number of patients with lateral epicondylitis improve with time, and this certainly occurred to a certain extent within our placebo group[3,12,13]. In the patients who remained in the placebo cohort, high success rates were seen at six months (81%) and one year (100%); however, this represents only 22% and 26%, respectively, of the intent-to-treat cohort, a rate that has been seen in other studies[13]. The high success rate of those who remained in the placebo group is likely due to the bias resulting from the fact that those who improved spontaneously did not cross over into active treatment. In fact, of those who remained in the placebo group after twelve weeks, only one patient had a pain score that would have qualified for inclusion into the crossover cohort.

Extracorporeal shock wave therapy as utilized in the current study, without the use of local anesthesia, is a safe and effective treatment of chronic lateral epicondylitis. In patients who have had failure of conventional treatment of lateral epi-

condylitis, shock wave therapy can significantly improve the pain scores, functional scores, and the subjective impression of the disease state. These results are contradictory to those found when extracorporeal shock wave therapy is used with local anesthesia or with concomitant use of nonsteroidal anti-inflammatory medications. Further research and clinical trials may be necessary to evaluate the ideal dosing and influence of confounding factors on the efficacy of shock wave therapy. ■

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