Long-Term Effect of Shock Wave Therapy on Upper Limb Hypertonia in Patients Affected by Stroke

P. Manganotti, MD, PhD; E. Amelio, MD

Background and Purpose—Spasticity is a disabling complication of stroke and different noninvasive treatments are used to reduce muscle hypertonia. Shock waves are defined as a sequence of single sonic pulses largely used in the treatment of diseases involving bone and tendon as well as muscular contractures. The effect and duration of extracorporeal shock wave therapy (ESWT) was investigated on muscle hypertonia of the hand and wrist.

Methods—A total of 20 patients affected by stroke associated with severe hypertonia in upper limbs were evaluated. Placebo stimulation was performed 1 week before active stimulation in each patient. Evaluation was performed using the National Institutes of Health and Ashworth scales and video monitoring with a digital goniometer before and immediately after placebo or active stimulation. Motor nerve conduction velocity from abductor digiti minimi were recorded. Patients were monitored at 1, 4, and 12 weeks after active treatment.

Results—After active ESWT, patients showed greater improvement in flexor tone of wrist and fingers compared with placebo stimulation. At the 1- and 4-week follow-up visits, a significant decrease of passive muscle tonicity was noted on muscles in all patients receiving active treatment. At 12 weeks after therapy, 10 of the 20 patients showed persistent reduction in muscle tone. There were no adverse events associated with ESWT.

Conclusions—ESWT reduces hypertonia of the wrist and finger muscles for 12 weeks after treatment. The possible mechanisms of action of ESWT are discussed. (Stroke. 2005;36:1967-1971.)

Key Words: extracorporeal shock wave therapy • muscle spasticity • rehabilitation • shock waves • stroke

Increase in muscle tone in the hands and wrists is a major problem in the management of chronic hemiparetic patients and may seriously impair dressing, washing, and other activities of daily living.1 Motor neuron syndrome in stroke patients is a collection of symptoms including motor defects, increased reflexes, and muscle hypertonia, which produce a series of complications causing changes in the rheologic components of muscles and subsequent stiffness in tendons and joints.1 In stroke, the hypertonia in muscles is partially related to spinal hyperexcitability in addition to fibrosis and changes in connective tissues that are responsible for passive rigidity of muscles.1

Recent studies have reported that muscular injections of botulinum toxin type A decreases muscular tone in hypertonic muscles of the hand, with improvement in the use of the upper limb and a decrease in complications.2-5 However, in a small number of patients, the development of neutralizing antibodies can reduce the efficacy of treatment. In addition, the dosage of botulinum is not always sufficient to treat extensive and severe hypertonia in upper and lower limbs. Rehabilitation and different noninvasive treatments, particularly on the connective components, should be also considered.

Shock waves are defined as a sequence of single sonic pulses characterized by high peak pressure (100 MPa), fast pressure rise (<10 ns), and short duration (10 μs). Different studies and clinical experiments have demonstrated the efficacy of shock waves in the treatment of bone and tendon diseases, including pseudoarthrosis6–8 tendinitis calcarea of the shoulder,9,10 epicondylitis,11 plantar fasciitis,12 and several tendon diseases, especially in athletes.13,14 The persistent clinical effects of shock wave treatment on muscular contractures in athletes together with preliminary data reporting a reduction in hypertonia in neurological patients after shock wave therapy15 has suggested a possible use of shock wave treatment in patients experiencing muscular hypertonia. Thus, it can be hypothesized that muscle hypertonia can decrease after shock wave therapy. The aim of the present study was to examine the effect of shock wave treatment on hypertonic muscles in the hand and wrist in a group of patients affected by stroke.

Subjects and Methods

Twenty patients with poststroke upper limb spasticity were enrolled in the study comprising 11 men and 9 women with a mean age of 63 years (range 38 to 76 years). Fifteen patients had ischemic stroke,
and 5 patients had hemorrhagic stroke. To be eligible for the study, patients must have had a stroke ≥9 months previously. Fifteen patients had mixed cortical and subcortical lesions mainly in the territory of middle cerebral artery. Five patients had mainly subcortical lesions (2 in thalamic site and 3 in the mesencephalic and pons structures). Most patients were receiving pharmacological therapy (15 antiplatelet and 12 antihypertensive). No patients took medications that could have had an impact on the study (eg, GABAergic medications). Patients with previous or planned treatment of the limb with botulinum toxin, phenol, alcohol, or surgery were excluded. All patients provided informed consent.

Study Procedure
The study was an open study in which each patient served as his or her own control. The protocol consisted of 1 placebo treatment session in which no shock waves were applied, followed by 1 active shock wave treatment session 1 week later. This was done to ensure homogeneity in the active and placebo groups so that the true effect of shock waves could be compared in each patient. To avoid possible crossover effects, the study included a 1-week interval between placebo treatment and active shock wave therapy. In each subject, clinical measures were evaluated before and immediately after placebo, and 1 week later, identical clinical measures were performed before and immediately after and at 1, 4, and 12 weeks after the active shock wave treatment.

Evaluation of Efficacy
The National Institutes of Health Stroke Scale (NIHSS) for neurological status16 was used. The mean score was 12 (range 10 to 15). For muscle tone, tonicity of the wrist and finger flexors was evaluated at all follow-up visits using the Ashworth scale.1,17 To evaluate the tonicity of fingers in a plegic hand, we fixed the wrist and stretched the fingers of the hand.1,17 Patients experienced focal spasticity of wrist and fingers, with a mean score of 3.4 (0.7) for wrist flexor tone and a mean score of 3.2 (0.6) for finger flexor tonicity on the Ashworth Scale, with 0 indicating normal muscle tone and 4 a rigid flexion.

A digital goniometer was used to measure the passive range of motion of the wrist. The goniometer was synchronized with a video polygraphy (Micromed System; Brainquick).

Moreover, an electrophysiological study was performed to address the possibility that extracorporeal shock wave therapy (ESWT) could cause nerve damage. Distal motor nerve conduction velocity and F responses from abductor digitii minimi by ulnar nerve stimulation were recorded. F wave responses were elicited by supramaximal stimulation of the ulnar nerve once every second. Seven F wave responses were collected at each recording session. For each set of 7 stimuli, we measured the mean F wave peak-to-peak amplitude and the mean latency of the responses.18 All electroneurographic studies were performed before and after the placebo and active stimulation and at 1, 4, and 12 weeks after active stimulation.

The needle electromyograph (EMG) was also investigated. Usually, the presence of chemical denervation as the consequence of the action of botulinum is detected by needle EMG. The procedure was performed only after 4 weeks, after ESWT in all patients on the first interosseus muscle, 1 of the treated muscles of the hand. We chose this interval because it is the mean time to observe electromyographic signs of denervation after nerve damage. No baseline EMG was performed because the muscle was not denervated before treatment, and the absence of signs of denervation after ESWT associated with an invariant amplitude of potential indicated that the therapy has no effect on the peripheral nerve structure.

The clinical and electrophysiological values of each patient were submitted to ANOVAs with repeated measures. Post hoc comparisons were performed with paired t tests adjusted with the Bonferroni method. P<0.05 was considered statistically significant.

Shock Wave Therapy Instrumentation
An electromagnetic coil lithotripter (Modulith SLK; Storz Medical AG) equipped with in-line ultrasound, radiographic and computerized aiming (Lithotrack system) was used. The pressure pulses were focused in the flexor hypertonic muscles of the forearm and the interosseus muscles of the hand: 1500 shots were used to treat flexor muscles of the forearm mainly in the middle of the belly, and 3200 shots for interosseus muscles of the hand (800 for each muscle) using an ultrasound pointer guide. The energy applied was 0.030 mj/mm². Different points of application were used to treat several areas of the hypertonic muscles. Because low energy is used, the therapy is painless and does not require the use of anesthesia or analgesic drugs. Placebo treatment without shock wave energy was applied with the same instrumentation, and the same sound was used in all patients. The shock wave treatment was performed over the flexor ulnaris, flexor radius, and over intrinsic muscles of the hand using the ultrasound device and pointer.

Results
Placebo Stimulation
No significant changes in the Ashworth or NIHSS scores were noted in either finger and wrist flexors after placebo stimulation (Figure 1; Table). Likewise, no significant changes in distal motor conduction or late responses (F response) were noted after placebo stimulation (Table).

Active Extracorporeal Shock Wave Stimulation
Muscle Tone
The average baseline evaluation in the Ashworth results for the wrist flexor was 3.2±0.7. The Ashworth score was 3.2±0.6 for the hand muscles. Immediately after active treatment, the Ashworth score for the wrist flexor decreased to 2.0±0.9 (P<0.001). The finger flexion (finger flexors) showed a marked reduction of spasticity with an Ashworth change to 0.8±0.4 (P<0.001; Figures 1 and 2; Table).

Persistent effects were observed in all subjects (Figure 3; Table). With regard to the Ashworth score, the time effect was statistically significant (F[4,12]=84.707; P<0.001). Post hoc comparisons showed a significant difference in muscle tone of finger flexors between baseline and muscle tone of the finger flexors after the first week (P<0.001) and at 4 (P<0.02) and 12 weeks (P<0.05).
Post hoc comparisons showed a significant difference of muscle tone of wrist flexors between baseline and after the first ($P<0.001$) and fourth weeks ($P<0.05$). There were no differences between baseline and muscle tone of the wrist flexors after 12 weeks ($P=\text{NS}$). No different effects of the shock wave were noted between patients with different degrees of hypertonia as measured by the Ashworth scale.

Range of Motion
With regard to the range of passive motion, the effect of time after treatment was statistically significant ($F_{(4,12)}=81.457; P<0.001$). Post hoc comparisons showed a significant difference of range of motion between baseline and the first ($P<0.01; 20^\circ$ versus $50^\circ$, respectively) and fourth weeks ($P<0.05; 20^\circ$ versus $40^\circ$, respectively). There were no differences between baseline and muscle tone of the wrist flexors after 12 weeks ($P=\text{NS}$; Table).

No significant changes were found in latency or amplitude of motor action potential (Table). No significant changes were noted in latency and amplitude of late responses across the different recordings (Table). After 4 weeks, the needle EMG recording did not show any signs of spontaneous activity in the first interosseus muscle, one of the treated muscles of the hand.

The NIHSS neurological examination score did not change either before (mean 12; SD 2.5) or after (mean 12; SD 3) treatment.

Discussion
The major finding of this study is that a single, active treatment of shock wave therapy on spastic muscles of upper limb in patients affected by stroke resulted in a significant reduction in muscle tone. No effect was noted after placebo stimulation. The effect of active stimulation lasted $\approx 12$ weeks after therapy. In particular, a significant effect on the muscle tone of the finger flexors was noted. No adverse effects were observed in any patient, and no

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**Clinical and Electrophysiological Findings**

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>After Placebo</th>
<th>After ESWT</th>
<th>After 1 Week</th>
<th>After 4 Weeks</th>
<th>After 12 Weeks</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ashworth wrist flexors</td>
<td>3.4 (0.7)</td>
<td>3.3 (0.6)</td>
<td>2.0 (0.9)*</td>
<td>2.4 (0.6)*</td>
<td>2.3 (0.7)*</td>
<td>3.0 (0.5)</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Ashworth finger flexors</td>
<td>3.2 (0.6)</td>
<td>3.06 (0.5)</td>
<td>0.8 (0.4)*</td>
<td>1.2 (0.4)*</td>
<td>1.3 (0.4)*</td>
<td>1.8 (0.7)*</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Range of motion (°)</td>
<td>20 (7)</td>
<td>20 (6)</td>
<td>50 (6)*</td>
<td>50 (7)*</td>
<td>40 (6)*</td>
<td>30 (8)</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Motor nerve conduction (ms)</td>
<td>55 (6)</td>
<td>54 (7)</td>
<td>57 (5)</td>
<td>54 (8)</td>
<td>55 (7)</td>
<td>56 (7)</td>
<td>NS</td>
</tr>
<tr>
<td>Latency CMAP (ms)</td>
<td>3.5 (1.3)</td>
<td>3.6 (2)</td>
<td>3.9 (1.7)</td>
<td>3.6 (1.4)</td>
<td>3.5 (2)</td>
<td>3.5 (3)</td>
<td>NS</td>
</tr>
<tr>
<td>Amplitude CMAP (mV)</td>
<td>11 (2)</td>
<td>12 (2)</td>
<td>11 (2)</td>
<td>12 (1.8)</td>
<td>11 (2.1)</td>
<td>11 (2)</td>
<td>NS</td>
</tr>
<tr>
<td>F Wave Mean Latency (ms)</td>
<td>25 (5)</td>
<td>25 (6)</td>
<td>23 (4)</td>
<td>23 (6)</td>
<td>25 (4)</td>
<td>26 (3)</td>
<td>NS</td>
</tr>
<tr>
<td>F Wave mean amplitude (uV)</td>
<td>700 (100)</td>
<td>790 (120)</td>
<td>650 (200)</td>
<td>670 (140)</td>
<td>680 (150)</td>
<td>700 (140)</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are expressed as mean and SDs.

* $P<0.001$ with Bonferroni correction.

CMAP indicates compound motor action potential.

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Figure 2. Clinical findings in a patient.
changes in peripheral nerve conduction or late responses were observed. No signs of denervation were recorded in the hand muscles.

The mechanism of shock wave therapy on spastic muscles is still unknown. Only a limited number of recent studies have investigated the mechanisms of shock waves, which can induce enzymatic and nonenzymatic NO synthesis. NO is involved in neuromuscular junction formation in the peripheral nervous system and in important physiological functions of the central nervous system, including neurotransmission, memory, and synaptic plasticity. NO synthesis has been suggested to be one of the most physiologically important mechanisms that could explain the effectiveness of shock waves in the anti-inflammatory treatment of various tendon diseases. However, a direct effect of shock waves on fibrosis and on the rheologic components of chronic hypertonic muscles should be considered in accordance with the documented, therapeutic effects on bone and tendon diseases.

Because in the present study we did not observe any changes in the F wave amplitude, which measures spinal excitability, we can hypothesize that shock waves can also act on the passive stiffness of muscles determined by inactive connective tissues.

Nonetheless, a direct effect of mechanical stimuli of shock waves on the muscle fibers adjacent to the tendon cannot be excluded. Continuous or intermittent tendon pressure can decrease spinal excitability without long-lasting clinical or neurophysiological effects. Notwithstanding, in this patient group, the clinical changes were observed for weeks after therapy and exclude a major effect of mechanical vibratory stimulation, which is transitory and short lasting. Moreover, the late responses usually decreased by vibratory stimulation were not significantly modified by shock wave therapy, suggesting a different mechanism of action.

In addition, because no signs of denervation were noted in treated muscles, we can exclude any relationship to neuromuscular denervation in the patient cohort treated with shock wave therapy. No changes were observed in either the amplitude or latency of distal motor action potential and late responses, excluding a significant effect of shock wave therapy on peripheral nerves and spinal excitability. On the contrary, the presence of denervation in treated hypertonic muscles and a long-lasting decrease in the amplitude of the motor action potential are related to the neuromuscular block caused by several types of botulinum neurotoxins.

Shock wave therapy appears to be safe and is also noninvasive. Our findings suggest that shock wave therapy may be useful in decreasing flexor tonicity in patients with spasticity of the hand and could open new areas of research in treatment of hypertonicity. Further studies with a larger group of patients are warranted.

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References


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