Effect of radial shock wave therapy on pain and muscle hypertonia: a double-blind study in patients with multiple sclerosis

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Abstract

Background: Radial shock wave therapy (RSWT) has been extensively used in rehabilitative medicine to treat pain, and more recently muscle hypertonia, in patients with cerebral palsy and stroke.

Objectives: To assess the long-term effects of RSWT in a cohort of subjects affected by multiple sclerosis (MS) who were suffering from painful hypertonia of ankle extensor muscles.

Methods: In this randomised, double blind, placebo-controlled study, we treated 34 patients with four sessions of RSWT (once weekly) and treated 34 patients with placebo. Participants were assessed at baseline, 1 week after the first session, and 1 week and 4 weeks after the last session. We measured pain using the visual analogue scale for pain, while we assessed muscle tone using the modified Ashworth scale and evaluated spinal excitability using the H-reflex.

Results: After RSWT, muscle tone decreased 1 week after the last session and pain decreased at all the follow-up evaluations, while spinal excitability was unaffected. No significant changes were found after the placebo treatment.

Conclusions: RSWT can reduce pain and muscle tone in MS patients without adverse effects. The lack of RSWT effects on spinal excitability supports the idea that RSWT is likely to act on non-reflex hypertonia, for example reducing muscle fibrosis.

Keywords: Ankle, ankle muscle strength, H-reflex, hypertonia, multiple sclerosis, muscle hypertonia, muscle tone, pain, radial shock wave therapy, rehabilitation, spasticity, spinal excitability, upper motor neuron syndrome, walking speed

Date received: 26 March 2014; accepted: 1 August 2014

Introduction

Spasticity can be defined as a form of hypertonia due to a velocity-dependent increase in tonic stretch reflexes, resulting from abnormal spinal processing of proprioceptive input.1 Spasticity is one of the clinical signs of upper motor neuron (UMN) syndrome; however, in addition to reflex hypertonia, patients with UMN syndrome are also suffering from a non-reflex hypertonia, due to connective tissue changes.2

Since the late 1980s, extracorporeal shock wave therapy (ESWT) has been widely and successfully used in the treatment of pain in various musculoskeletal disorders.3 Moreover, ESWT has been successfully used for the treatment of hypertonia in subjects with UMN syndrome.4,5 ESWT devices use pressure waves that are generated through electromagnetic, electro-hydraulic and piezoelectric sources. These waves have the point of higher pressure at the centre of their focus, which is placed within the treated tissue; thus, they are defined as focused shock waves.6,7

In 1999, a new technology using a ballistic source to generate pressure waves was introduced. This technology is called radial shock wave therapy (RSWT). The ballistic source consists of a tube within which compressed air (1–4 bar) is used to fire a bullet that strikes a metal applicator, placed on the patient’s skin. The applicator transforms the kinetic energy of the bullet into radially-expanding pressure waves with a
low penetration power (< 3 cm). These unfocused shock waves have their point of highest pressure at the tip of the applicator, outside the treated tissue.\textsuperscript{6,7}

It was previously shown that both focused (ESWT) and unfocused (RSWT) shock waves produce cavitation bubbles in the treated tissue. The cavitation is consequent to the negative phase of wave propagation. The rapid collapse of the cavitation bubbles leads to secondary pressure waves. Cavitation-mediated mechanisms could have a central role in the actions of both ESWT and RSWT.\textsuperscript{7}

RSWT is extensively used in rehabilitative medicine to treat painful musculoskeletal disorders, such as: medial tibial stress syndrome,\textsuperscript{8} lateral epicondylitis\textsuperscript{9} and plantar fasciitis.\textsuperscript{10} Three recent works suggest that RSWT can reduce hypertonia in patients with UMN syndrome that was due to cerebral palsy\textsuperscript{11,12} and to stroke.\textsuperscript{13} This clinical improvement has been related to a direct effect of RSWT on muscle fibrosis and other components of non-reflex hypertonia.\textsuperscript{11}

Muscle hypertonia affects up to 80% of subjects with multiple sclerosis (MS) and is often painful.\textsuperscript{14} Pain probably reflects the prolonged abnormal contractions due to spasticity, but it also depends on the musculoskeletal consequences due to spasticity and the other components of the UMN syndrome.\textsuperscript{1} The relationship between spasticity and pain is made even closer by the fact that pain increases spasticity, creating a spiralling course of more pain and disability.\textsuperscript{15}

In the hope to combine the two effects of RSWT on pain and hypertonia, we used RSWT in the present study to treat the painful hypertonia of ankle extensor muscles (triceps surae) in a cohort of subjects affected by MS. The first aim of the study was to assess the clinical effect of RSWT on pain (primary outcome) and also to assess hypertonia (secondary outcome) in a randomised placebo-controlled parallel arm trial. The second aim was to investigate the mechanisms by which RSWT exerts its effects. In order to differentiate the possible effects of RSWT on the reflex and non-reflex components of hypertonia, we assessed spinal excitability using H-reflex studies.

Materials and methods

Inclusion criteria

Patients were enrolled at the Department of Neuroscience of the University of Genova, Italy, according to the following criteria:

1. MS diagnosed according to the revised McDonald’s criteria\textsuperscript{16} with a Kurtzke Expanded Disability Status Score (EDSS) > 4;
2. Hypertonia of ankle extensor muscles ranging from 1–4, according to the modified Ashworth scale (MAS);
3. Pain during ankle mobilization rated > 4 in the visual analogue scale for pain (VAS), which ranges from 0 (no pain) to 10 (unbearable pain);
4. No clinical relapse, and no use of corticosteroid or botulinum toxin, in the last 6 months.

The study was approved by the local ethics committee. A total of 120 subjects (68 women) were examined for study eligibility. At the end of the evaluation, 68 subjects (40 women; mean age ± SD = 51.4 ±12.2 years) met the inclusion criteria and joined the study.

Clinical outcome measures

The primary outcome was pain referred to the treated lower limb, which was measured using the VAS for pain. Responders were those subjects who experienced a pain reduction after treatment greater than 33%, versus that at time zero (T0) prior to treatment.\textsuperscript{17}

The secondary outcome was muscle tone of the ankle extensor muscles, measured in the supine position by means of the MAS (the patient’s ankle was moved from a position of maximal extension to maximal dorsiflexion, over a duration of about 1 second). To accommodate the ‘1+’ modification for numerical analysis, grade 1 was recorded as 1 and ‘1+’, as 1.5.

Further outcome measures were ankle muscle strength and walking speed. Ankle strength in extension was rated according to the Medical Research Council (MRC) for muscle strength. Walking speed was assessed by the 10-m walking test (10-MWT).

The same physician, who was blinded to the protocol, performed all clinical assessments.

Electrophysiological study

This assessment was performed to investigate the possible effects of RSWT on the stretch reflex excitability. Subjects were tested while lying in a bed, relaxed in a prone position, with their feet over the
edge of an examining table. Special care was taken to assure that muscles acting on the ankle joint were at complete rest. The posterior tibial nerve was stimulated by a surface bipolar electrode placed in the popliteal fossa. Rectangular pulses of 2 ms duration were administered by means of a constant-current stimulator (model DS7A; Digitimer, UK).

EMG was recorded through bipolar surface preamplified electrodes (TSD150B; Biopac Systems, USA) positioned over the soleus muscle, 3 cm below the insertion of the gastrocnemii. We evaluated M-wave and H-reflex peak-to-peak amplitudes by means of the Acqknowledge software (Biopac Systems, USA). At the beginning, for each subject, the soleus H – M recruitment curve was built up, using a stimulation frequency of 0.1Hz. The electrical stimulation intensities producing H-max (the H-reflex with the maximal amplitude) and M-max (the M-wave elicited by a supramaximal stimulus) were defined and then we calculated the H-max/M-max ratio. Stimulus strength was set (in the ascending limb of the H-reflex intensity curve) to produce H-reflexes having amplitude near to H-max/2, using a frequency of 0.1Hz. Using this stimulation intensity, we collected 20 H-reflexes at 0.1Hz and then 20 H-reflexes were recorded at a frequency of 1Hz. To calculate post-activation depression (PD), the ratio of the H-reflex amplitude evoked at 1Hz to the H-reflex amplitude evoked at 0.1Hz (1Hz/0.1Hz ratio) was calculated in each single subject: The greater the 1Hz/0.1Hz ratio, the smaller the PD.

**Radial shock wave therapy**

We used a BTL-6000 SWT Topline Unit (BTL, Italy). Patients were treated only on one side. When both sides met the inclusion criteria (hypertonia of ankle extensor muscles ranging from 1 to 4, according to the MAS and pain during the ankle mobilization), then treatment was delivered to the most painful side. RSWT consisted of a 4-session course, with a 1-week interval between sessions. During each session, 2000 shots were delivered to ankle extensor muscles, including the Achilles tendon (600 shots in each gastrocnemius muscle, 600 shots in the soleus muscle and 200 shots in the Achilles tendon). We used a frequency of 4 Hz, with a pressure of 1.5 Bars. The treatment was not painful.

**Placebo treatment**

The placebo treatment was similar to RSWT; however, in the placebo sessions the shock waves were prevented from reaching the target muscles by a thin foam cushion placed on the metal applicator. The therapists delivering RSWT were not blinded. On the contrary, the medical doctors performing the clinical and H-reflex measurements were blinded.

**Study procedure**

Patients were randomly allocated to receive either the RSWT or the placebo treatment after stratification using a software-generated randomization tool. No physiotherapy treatment was performed after the treatment.

The clinical examination was performed: just before the first treatment session (T0); one week after the first session (just before the second session) (T1); one week (T2) and 4 weeks (T3) after the last session (figure).

The H-reflex investigation, performed only in the subjects treated with RSWT, was performed 2 weeks before the T0 baseline and at T2. The results obtained in patients at baseline were compared with those obtained in healthy subjects.

**Statistical analysis**

At T0, differences between RSWT and placebo groups were analysed using the unpaired \( t \)-test (age values) and Mann-Whitney U test (EDSS, MAS, VAS, 10-MWT and MRC scores).

Changes between T0 and post-treatment (T1, T2 and T3) clinical measures (MAS, VAS, 10-MWT and MRC scores) were analysed using the Wilcoxon test. H-reflex parameters (H-max/M-max ratios and 1Hz/0.1Hz ratios) obtained in patients at baseline were compared to those obtained at T2, using the paired \( t \)-test. H-reflex parameters obtained in patients at baseline were compared to those obtained in healthy subjects using the unpaired \( t \)-test.

The level of statistical significance was set as \( p < 0.05 \). All data was shown as mean ± standard deviation (SD).

**Results**

A total of 34 subjects received RSWT and 34 subjects received placebo treatment.
During the period of the study (from enrolment to 4 weeks after the last treatment’s session), the therapies potentially acting on muscle tone and pain were not modified. In the RSWT group, 25 subjects were treated with baclofen, 10 with benzodiazepines, 12 patients with pregabalin and 2 with gabapentin. In the placebo group, 23 patients were treated with baclofen, 13 patients were treated with benzodiazepines, 10 patients with pregabalin and 2 with famipridine.

Table 1 and Table 2 show the pre-treatment (T0) demographic and clinical characteristic of the 68 subjects enrolled in the study. At T0, statistical analysis did not find any significant differences in age distribution, EDSS and clinical outcome measures (VAS for pain, MAS, MRC for muscle strength and 10-MWT) between the subjects treated with RSWT and those treated with placebo.

Table 2 shows the time course of the clinical outcome measures. After RSWT, VAS scores significantly decreased at all follow-up evaluations, with the maximal effect at T2, when the mean pain score decreased from 6.49 to 3.44. Responders were: 26% of the subjects at T1, 68% of the subjects at T2 and 32% of the subjects at T3. MAS scores significantly decreased only at T2, while significant changes were not observed by MRC and 10-MWT. After the placebo treatment, we found no significant changes from T0 values. There was only one responder to the placebo (3%).
Discussion
The main finding of this study, performed in MS patients with hypertonia of ankle extensor muscles associated with pain, was that four sessions of RSWT induced a significant pain reduction. This effect, peaking 1 week after the last session (at T2), was already disclosed 1 week after the first session, and persisted 4 weeks after the last session. At the time of the maximal effect on pain (at T2), we observed a reduction of muscle tone. RSWT did not have any significant influence on muscle strength and gait speed (10-MWT). No effect was noted after the placebo treatment.

Pain that is both nociceptive and neuropathic is a common symptom in MS.18 In the patients enrolled in the present study, pain was enhanced by ankle mobilization. In the majority, spontaneous pain increased by passive and active motion of the ankle; a few patients had pain exclusively during ankle mobilization. This dependence on mobilization of the ankle supports the nociceptive nature of the pain, even though a neuropathic component cannot be excluded.

Spasticity can be the direct cause of pain in MS patients.14 It was shown, in healthy subjects, that lengthening a contracted muscle (eccentric contraction) can cause the disruption of some muscle fibres, with the release of substances that may excite the muscle nociceptors.19 The same process is likely to happen when a spastic muscle is stretched. However, it must be stressed that spasticity is only one of the positive signs displayed by patients with UMN syndrome; others include: muscle spasms, co-contractions and spastic dystonia. The negative signs are weakness and loss of dexterity.1 All these positive and negative features, along with soft tissue changes, perturb body weight distribution, inducing excessive stress on joint structures and causing pain.20 Sensory disturbances can also play a role. It is the mixing and matching of such components that leads to the pain perceived by the patients with UMN syndrome.

In this complex scenario, the reduction of muscle tone after RSWT is likely to have played only a partial role in the pain relief experienced by the patients investigated in this study. This view is corroborated when the time courses of the two phenomena are compared. The reduction of pain was present 1 week after the first session (T1) and 4 weeks after the last session (T3), at a time when no effect on muscle tone was detected. Therefore, we believe that the pain reduction that we observed was largely determined by the shock wave action on nociception. Although pain relief is the main result reported following RSWT, the antinociceptive mechanisms of shock waves are far from being completely understood. Important mechanisms are thought to be nitric oxide production, inhibition of cytokines and the modulation of peptides involved in nociception.21

While our observed results on pain were in line with the literature, our findings on muscle tone lasted for a shorter time than was previously reported. One week after the first session (T1) of RSWT, we did not find any effect on muscle tone; muscle tone was reduced 1 week after the last session (T2), but the effect was not maintained 1 month after the last session (T3).

Indeed, previous works (in which RSWT was used to treat hypertonia) show there were more enduring results, lasting at least 2 weeks after the last session.11–13 We suggest that the discrepancy between our results and the previous ones is probably related to the patients’ age, disease duration and the level where the UMN are damaged.

Concerning the possible mechanisms of the observed muscle tone reduction, pain relief is one candidate. It is well known that pain itself may be contributing to increased muscle tone.15 Therefore, treating the pain may reduce muscle tone. In the present study, the reduction of muscle tone was detected only when the effect on pain reached its peak (at T2), suggesting that pain reduction must exceed a threshold to determine its effects on muscle tone.

Another mechanism could be the effect of RSWT on muscle fibrosis and other non-reflex components of

| Table 3. H-reflex results in MS patients treated with RSWT and in 40 age-matched healthy subjects. |
|-----------------|-----------------|-----------------|
|                 | H-max/M-max ratio | 1 Hz/0.1 Hz ratio |
| Healthy subjects| 0.30 ± 0.14 (p = 0.000002)a | 0.47 ± 0.18 (p = 0.005)a |
| Patients at baseline T0 | 0.57 ± 0.26 | 0.62 ± 0.22 |
| Patients at T2 | 0.56 ± 0.24 (p > 0.05)a | 0.59 ± 0.13 (p > 0.05)a |

aP values refer to the comparison with the results obtained in patients at baseline.
H-max: the H-reflex with the maximal amplitude; Hz: Hertz; M-max: the M-wave elicited by a supramaximal stimulus; MS: multiple sclerosis; RSWT: radial shock wave therapy.
muscle hypertonia. This mechanism was originally suggested to explain the reduction of muscle tone induced by ESWT. Through its action on non-reflex hypertonia, however, RSWT could also reduce spasticity. The reduced extensibility, due to soft tissue changes, causes pulling forces to be transmitted more readily to the muscle spindles. In this condition, an exaggerated spindle discharge in response to muscle stretch might lead to an increased stretch reflex. Thus, the reduction of non-reflex hypertonia could modify muscle spindles’ excitability, leading to a secondary reduction of spasticity.

A third mechanism by which RSWT might act on muscle tone could be the modification of the excitability of the spinal circuits mediating the stretch reflex. Indeed, mechanical stimuli acting on muscles and tendons can decrease spinal excitability and induce long-lasting effects on spasticity. To test this possibility, we measured the excitability of the stretch reflex loop using the soleus H-reflex in all the patients treated with RSWT, and in a group of 40 age-matched controls. Specifically, we investigated the H-max/M-max ratio, which is considered an index of spasticity. Furthermore, we evaluated post-activation depression (PD), i.e. the inhibition of the H-reflex induced by a preceding conditioning stimulus that is able to activate the afferents mediating the H-reflex itself. PD was investigated by assessing the frequency-related depression of the soleus H-reflex. We decided to investigate PD, because it is highly correlated to the severity of spasticity and it has been used in the longitudinal assessment of spasticity.

As largely expected, at baseline, the soleus H-max/M-max ratio was higher and PD was lower in patients than in healthy controls. In the treated MS patients, these parameters remained stable for 1 week after the last session of RSWT (T2), when muscle tone decreased. Our data, therefore, did not support the action of RSWT on the excitability of spinal circuits underlying the stretch reflex and confirmed previous results in stroke patients after ESWT. Indeed, it is well known that MAS is not able to discriminate between reflex and non-reflex hypertonia. The lack of RSWT effects on spinal excitability supports the idea that RSWT is likely to act on non-reflex hypertonia, for example on muscle fibrosis. Further studies are needed to investigate this issue.

During the study, we did not have patients receive treatment with physiotherapy. This was done in order to refer any changes in outcome measures to RSWT itself. In connection with this, the absence of functional changes on gait (speed velocity) did not represent an unexpected result. On the contrary, it confirmed that, to achieve functional results, any physical treatment should be integrated as part of a comprehensive rehabilitation program, in which the role of physiotherapy is essential.

The present study is the first one in which shock waves were used to treat muscle hypertonia and pain in MS patients. This is also the first randomised placebo-controlled study in which radial shock waves were used to treat hypertonia in adult patients.

With its limitations related to the lack of an objective assessment of hypertonia and the limited number of subjects, the present study suggested that RSWT can reduce pain and muscle tone in MS patients, without any effect on muscle strength. Further studies are needed to confirm our present results and to evaluate their impact on quality of life. To optimise the effect in MS patients and obtain functional results, we think that RSWT should be integrated into a rehabilitation program, where physiotherapy should consist of active and passive stretching of the hypertonic muscles, strength training of the antagonist muscles, functional mobility training and gait pattern training.

Conflict of interest
The authors declare that there is no conflict of interest. The funder had no role in study design, data collection and analysis; decision to publish nor preparation of the manuscript.

Funding
This work was supported by the Fondazione Italiana Sclerosi Multipla (FISM) [the Italian Foundation for MS] (grant number 2011/R/35).

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