

Positive Effects of Extracorporeal Shock Wave Therapy on Spasticity in Poststroke Patients: A Meta-Analysis

Peipei Guo, MM,^{*,1} Fuqiang Gao, MD,^{†1} Tingting Zhao, MM,^{†1} Wei Sun, MD,[†]
Bailiang Wang, MD,[†] and Zirong Li, MD[†]

Background: Spasticity is a common and serious complication following a stroke, and many clinical research have been conducted to evaluate the effect of extracorporeal shock wave therapy (ESWT) on muscle spasticity in poststroke patients. This meta-analysis aimed to evaluate the therapeutic effect on decreasing spasticity caused by a stroke immediately and 4 weeks after the application of shock wave therapy. *Methods:* We searched PubMed, Embase, Web of Science, and Cochrane Library databases for relevant studies through November 2016 using the following item: (Hypertonia OR Spasticity) and (Shock Wave or ESWT) and (Stroke). The outcomes were evaluated by Modified Ashworth Scale (MAS) grades and pooled by Stata 12.0 (Stata Corp, College Station, TX, USA). *Results:* Six studies consisting of 9 groups were included in this meta-analysis. The MAS grades immediately after ESWT were significantly improved compared with the baseline values (standardized mean difference [SMD], -1.57; 95% confidence intervals [CIs], -2.20, -.94). Similarly, the MAS grades judged at 4 weeks after ESWT were also showed to be significantly lower than the baseline values (SMD, -1.93; 95% CIs, -2.71, -1.15). *Conclusions:* ESWT for the spasticity of patients after a stroke is effective, as measured by MAS grades. Moreover, no serious side effects were observed in any patients after shock wave therapy. Nevertheless, our current study with some limitations such as the limited sample size only provided limited quality of evidence; confirmation from a further systematic review or meta-analysis with large-scale, well-designed randomized control trials is required. **Key Words:** Extracorporeal shock wave therapy—muscle spasticity—stroke—meta-analysis.

© 2017 Published by Elsevier Inc. on behalf of National Stroke Association.

From the *The Graduate School of Peking Union Medical College, Beijing; and †Centre for Osteonecrosis and Joint-Preserving & Reconstruction, Department of Orthopedic Surgery, Beijing Key Laboratory of Arthritic and Rheumatic Diseases, China-Japan Friendship Hospital, National Health and Family Planning Commission of the People's Republic of China, Beijing, China.

Received March 25, 2017; revision received July 20, 2017; accepted August 13, 2017.

This study was supported by the Beijing Natural Science Foundation (7174346), National Natural Science Foundation of China (81372013, 81672236), and the Research Fund of China-Japan Friendship Hospital (2014-4-QN-29), China-Japan Friendship Hospital Youth Science and Technology Excellence Project (2014-QNYC-A-06).

Address correspondence to Wei Sun, MD, Department of Orthopedics, China-Japan Friendship Hospital, Beijing 100029, China. E-mail: 18901267995@163.com.

¹ Joint first authors.

1052-3057/\$ - see front matter

© 2017 Published by Elsevier Inc. on behalf of National Stroke Association.

<https://doi.org/10.1016/j.jstrokecerebrovasdis.2017.08.019>

Introduction

Spasticity is a neurological symptom frequently appearing in patients after a stroke and is defined as a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks due to the hyperexcitability of the stretch reflex.¹ The prevalence of spasticity was reported to be approximately 42%,² and permanent contracture and skeletal deformities may occur in a certain group of hypertonic muscles suffering from sustained contraction,³ leading to a tougher life and a higher medical fee for poststroke patients. Therefore, how to control and treat spasticity after a stroke has been an increasingly essential problem. There were various options for the management of spasticity including physical therapy (cryotherapy, thermotherapy, vibration, and electrical stimulation), oral antispastic drugs (baclofen, tizanidine, dantrolene, and benzodiazepines), chemical nerve blocks (phenol and alcohol), and botulinum toxin injections.⁴ But considerable adverse effects of the existing treatments have been found during or after the treatments. For example, systemically administered antispastic drugs may reduce the force of normal muscles and their value may diminish with prolonged use; nerve blocks like phenol often cause skin sensory loss and dysesthesia.⁵ In addition, repetitive injections of botulinum toxin may stimulate the formation of neutralizing antibodies, and the inappropriate dosage and incorrect injection sites also challenge the efficacy of this treatment.⁶ The previously reported physical modalities either needed a further evaluation of effectiveness or lacked long-term effects.^{7,8} Compared with these conventional treatments, extracorporeal shock wave therapy (ESWT) is considered as a safe, effective, practical, and noninvasive method for reducing spasticity. The shock wave is defined as a sequence of single sonic pulses characterized by high peak pressure (sometimes more than 100 MPa), fast pressure rise (<10 ns), and short duration (10 us) and is conveyed by an appropriate generator to a specific target area.⁹ ESWT was first introduced in the early 1980s to break up kidney stones.¹⁰ In recent years, shock wave therapy has been reported as the leading choice for the treatment of musculoskeletal diseases such as calcific tendinitis of the shoulder,¹¹ nonunion of a long-bone fracture,¹² plantar fasciitis,¹³ and lateral epicondylitis of the elbow.¹⁴

Several recent clinical trials have been performed to evaluate the effect of ESWT on muscle spasticity in poststroke patients. Those research adopted different clinical scales, and the follow-up time varied from each other, so it was insufficient to draw a definite conclusion on the effectiveness. Up to now, we only found 1 similar review article published in 2016 by Dymarek et al.¹⁵ This article arrived at the conclusion that the application of extracorporeal shock wave was safe and effective on upper- and lower-limb spasticity in poststroke patients, which

was also similar to our conclusion. However, they did not use a meta-analysis, and the date of retrieval was 1 year ago. Therefore, we conducted this meta-analysis to provide a more prudential and convincing conclusion, hoping to bring some evidence to the application of extracorporeal shock wave for spasticity in poststroke patients.

Material and Methods

This meta-analysis was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA)¹⁶ reporting guidelines for conducting a meta-analysis of intervention trials. In this meta-analysis, we unified the clinical grades using the Modified Ashworth Scale (MAS), which was extensively used and proved to have good validity when evaluating spasticity in patients with stroke.^{17,18} The MAS is a velocity-independent clinical method that manually assesses the resistance during a passive movement of a joint with varying degrees of velocity using a 6-grade scale, ranging from 0 (normal muscle tone) to 4 (limb rigid in flexion or extension).¹⁹

Data Sources and Searches

We searched PubMed, Embase, Web of Science, and Cochrane Library databases to identify relevant studies using ESWT to relieve spasticity in patients after a stroke. The following search terms were used for the initial literature search: (Hypertonia or Spasticity) and (Shock Wave or ESWT) and (Stroke). Articles were not limited to any particular study design, but the language of the publications was restricted to English. The study selection was then independently performed by 2 of the authors, and any different opinions were resolved through discussion. In addition, we checked the reference lists of the articles manually to identify other potentially relevant publications.

Study Selection

Studies were considered eligible if they met the following criteria: (1) the study enrolled patients with muscle spasticity because of ischemic stroke or hemorrhagic stroke; (2) the language of the publications was English; (3) the results of the study were at least evaluated by the MAS, and the baseline and post-treatment results were all provided by or could be transformed into a mean and standard deviation. Studies were excluded if they were animal studies or used ESWT in combination with any other treatments such as botulinum toxin injection and chemical nerve blocks. Articles that reported at least 1 outcome were included, and those without the outcome measures of interest were excluded. Letters, comments, editorials, and practice guidelines were excluded.

Data Extraction and Quality Assessment

Two authors independently reviewed all titles and abstracts of the studies identified by searches according to the eligibility criteria described above. Full texts of articles that met the inclusion criteria were reviewed thoroughly. Disagreements were resolved by discussion to reach consensus. Data on patient characteristics (age, sex, and other baseline characteristics), intervention, and outcomes were extracted in duplicate by 2 authors using a standardized form.

The methodological quality of randomized control trials (RCTs) was assessed using a modified version of the Jadad Scale (0 [“very poor”] to 7 [“rigorous”]). The modified Jadad Scale contained 2 questions each on randomization and masking, and 1 question on the reporting of dropouts and withdrawals.²⁰ The Newcastle–Ottawa Scale was used for nonrandomized control trials (nRCTs), assessing the population selection, comparability of the exposed and unexposed, and adequacy of outcome assessment (including ascertainment and attrition of outcome).²¹ Data extraction and quality assessment were undertaken independently by 2 of the authors.

Data Synthesis and Analysis

MAS grades were statistically analyzed, and the Stata 12.0 (Stata Corp, College Station, TX, USA) was used for the graphical representation of the pooled data. Statistical heterogeneity was assessed by both a Cochran’s chi-squared test (Q test) and an I-squared test. Continuous outcomes were expressed as the mean \pm standard deviation, and they were summarized using the standardized mean differences (SMDs) and 95% confidence intervals (CIs). A fixed effects model was used when there was no significant statistical heterogeneity ($P > .1$ and an I^2 value $< 50\%$). Otherwise, a random effects model statistical method was employed. And if there was still some huge statistical het-

erogeneities, a sensitivity analysis investigating the influence of each single study on the overall outcome estimate was conducted by omitting 1 study at each turn. The funnel plot, Begg’s tests,²² and Egger’s tests²³ were simultaneously used to assess potential publication biases.

Results

A total of 63 potentially relevant articles were initially searched from the PubMed, Embase, Web of Science, and Cochrane Library databases, and 32 duplicates were discharged by EndNote. Then 10 were excluded after scrutiny of their titles or abstracts, leaving 21 articles to be evaluated through full text scrutiny according to the selection criteria, and then 15 were excluded. Eventually, 6 studies^{24–29} consisting of 9 groups were included in this meta-analysis, and they were all nRCTs. Four groups conducted their studies on the plantar flexor; 1 tested the shoulder external rotator muscles, and the finger flexor and wrist flexor were evaluated by 2 groups. One study²⁸ examining the plantar flexor divided the subjects into 2 groups according to muscle echo intensity using the Heckmatt scale.³⁰ Figure 1 shows the progress and details of our search work. The basic characteristics of the included studies are listed in Table 1. The methodological quality of each component study was assessed with the Newcastle–Ottawa Scale, which is also shown in Table 1 by transforming the stars to scores. Sensitivity analysis was used to determine whether the modification of the inclusion criteria affected the final results. Considering the small number of the selected studies in our meta-analysis, we used Begg’s test and Egger’s test to evaluate the publication bias.

Immediately after ESWT

Five studies including 8 groups^{25–29} evaluated the effect of ESWT on the improvement of the MAS grades

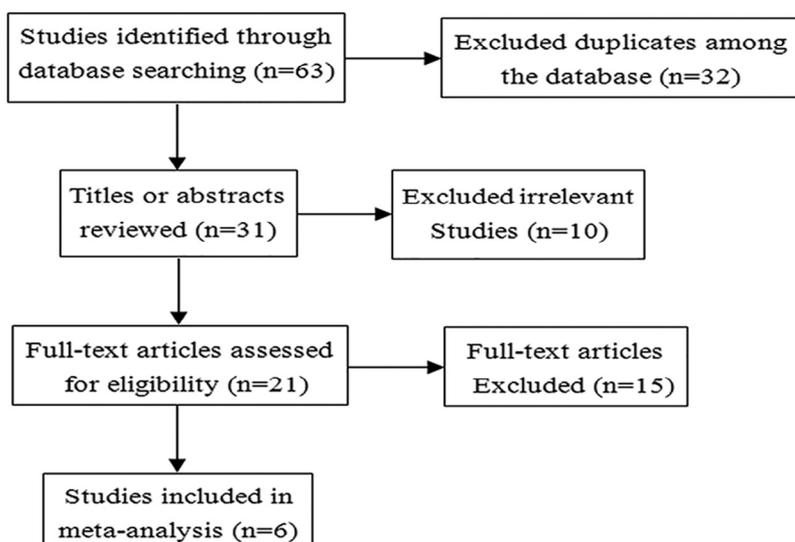


Figure 1. The selection process of this meta-analysis.

Table 1. Demographic characteristics of component studies

Author/ published year	Number of patients (n) (F/M)	Average age (year)	Average onset	Stroke subtype (n): ischemic/ hemorrhagic	Adverse effects	Quality score	Energy (mJ/mm ²)	Dosage and treated muscles	Tested muscles	Modified Ashworth Scale (mean ± SD)		
										Baseline	Immediately after ESWT	4 weeks after ESWT
Kim et al (2013) ²⁴	57 (24/33)	55.40	24.60 months	22/35	Pain, petechiae	7	.630	3000 shots for subscapularis	Shoulder external rotator muscles	2.70 ± .90	-	1.40 ± .60
Manganotti and Amelio (2005) ²⁵	20 (9/11)	63.00	≥9.00 months	15/5	No adverse effects	6	.030	1500 shots for forearm flexor, 3200 shots for hand interosseus muscles (800 for each muscle)	Wrist flexor (a) Finger flexor (b)	3.40 ± .70 3.20 ± .60	2.00 ± .9 .80 ± .40	2.30 ± .70 1.30 ± .40
Dymarek et al (2016) ²⁶	20 (7/13)	63.15	45.35 months	20/0	No adverse effects	5	.030	1500 shots for wrist flexor, the flexor carpi radialis, and flexor carpi ulnaris	Wrist flexor (a) Finger flexor (b)	1.60 ± .70 2.00 ± 1.00	1.30 ± .60 1.40 ± .80	- -
Moon et al (2013) ²⁷	30 (13/17)	52.60	80.50 days	16/14	Not mentioned	6	.089	1500 shots for gastrocnemius	Plantar flexor	2.50 ± .67	1.41 ± .67	1.75 ± .62
Santamato et al (2014) ²⁸	23 (8/15)	57.60	24.90 months	12/11	Pain, muscular weakness	7	.100	1500 shots for gastrocnemius and the soleus	Plantar flexor: Heckmatt grades I, II, and III (a) Plantar flexor: Heckmatt grade IV (b)	3.50 ± 1.00 4.70 ± .50	2.10 ± 1.10 3.30 ± 1.20	- -
Sohn et al (2011) ²⁹	10 (4/6)	44.90	53.40 months	2/8	Mild pain	6	.100	1500 shots for gastrocnemius	Plantar flexor	2.67 ± 1.15	1.22 ± 1.03	-

Abbreviations: ESWT, extracorporeal shock wave therapy; SD, standard deviation.

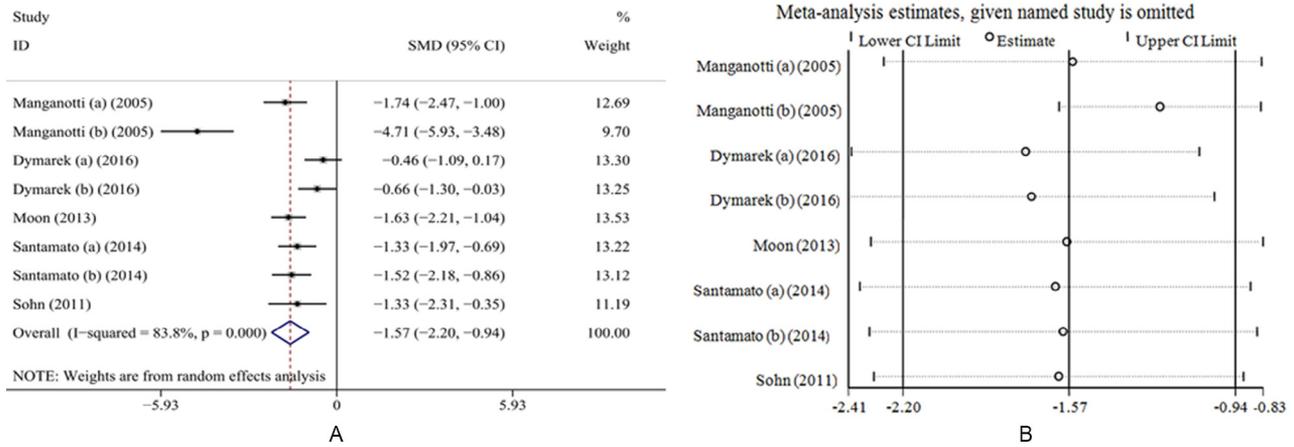


Figure 2. Forest plot of outcomes assessment (A) and sensitivity analyses plot of outcomes assessment (B) immediately after extracorporeal shock wave therapy. Abbreviations: CI, confidence interval; SMD, standardized mean difference.

immediately after the application of ESWT. A random effects model was used to analyze these data. The pooled estimate of effect size showed that ESWT induced statistically significant MAS grade reduction immediately compared with the baseline values (SMD = -1.57; 95% CI, -2.20, -0.94; $z = 4.88$; $P < .001$) (Fig 2, A). Considering a significant level of interstudy heterogeneity among the included trials (chi-square = 43.14; $df = 7$; $P < .001$; $I^2 = 83.8\%$), we conducted a sensitivity analysis. When omitting the second group provided by Manganotti and Amelio,²⁵ we found a relatively remarkable different outcome, but this result did not show materially a difference with that from the original analysis (Fig 2, B). The results of Begg’s test ($P = .108$) and Egger’s test ($P = .055$; 95% CI, -16.000, 2.149) suggested that publication bias was not evident in the included studies.

Four Weeks after ESWT

Data on 4 weeks after ESWT were provided by 3 of the selected papers (4 groups).^{24,25,27} A fixed effects model

was used to analyze these data. There was a significant difference in the improvement of the MAS grades between the results of baseline and 4 weeks after ESWT (SMD = -1.93; 95% CI, -2.71, -1.15; $z = 4.83$; $P < .001$), with a significant level of heterogeneity among the studies (chi-square = 18.33; $df = 3$; $P < .01$; $I^2 = 83.6\%$) (Fig 3, A). The sensitivity analysis also showed an outcome differing from the primary result when the data from the second group, from Manganotti and Amelio,²⁵ were neglected, but this result did not suggest a statistical difference (Fig 3, B). The publication bias was not found according to the results of Begg’s test ($P = .734$) or those of Egger’s test ($P = .321$; 95% CI, -20.983, 11.202).

Adverse Effects

Some patients in Kim et al’s study²⁴ complained of post-treatment pain, and 1 patient had petechiae that resolved spontaneously. Mild adverse effects were reported (injection site pain for 5 patients and lower-limb muscular weakness for 2 patients) by Santamato et al,²⁸ which were

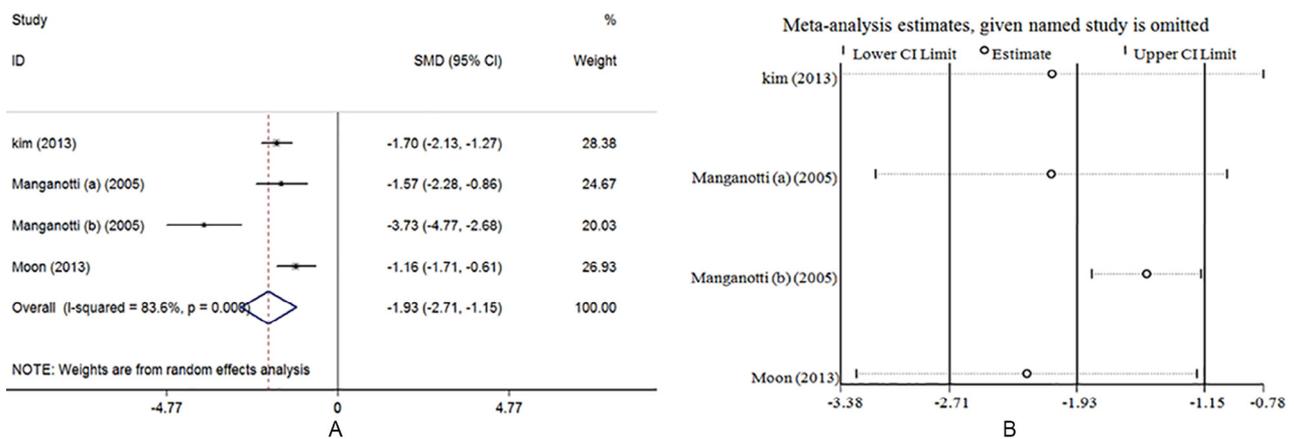


Figure 3. Forest plot of outcomes assessment (A) and sensitivity analyses plot of outcomes assessment (B) 4 weeks after extracorporeal shock wave therapy. Abbreviations: CI, confidence interval; SMD, standardized mean difference.

resolved in a few days. Mild pain during treatment from several cases was also reported by Sohn et al.²⁹ Two studies suggested no adverse effects,^{25,26} and the rest did not mention the adverse event.²⁷ So we regarded shock wave therapy as a safe management for spasticity in poststroke patients.

Discussion

As one of the major positive clinical signs after stroke, spasticity can also cause a series of clinical problems including increased muscle tone and excessive contraction of antagonist muscles, and it may postpone the rehabilitation of relative muscles.^{31,32} Due to the various adverse effects of previous treatments, the extracorporeal shock wave has been used for treating spasticity. The exact mechanism underlying the beneficial effect of ESWT on spasticity has not yet been clarified. And some previous studies hypothetically described the mechanism as the production of nitric oxides (NOs),³³ the effect on spinal cord excitability,^{25,34} and the affection on the golgi tendon organ. According to 2 of our selected studies,^{25,29} patients with improved MAS grades did not show a higher F-wave responses or Hmax/Mmax ratio after ESWT, which suggests that the ESWT did not result in an improvement of alpha motoneuron excitability. A direct effect of mechanical stimulation by shock waves on the golgi tendon organ to suppress motor nerve excitability can also be excluded.³⁵ NO, generated by shock wave, is involved in neurotransmission, memory formation, and synaptic plasticity in the central nervous system, and it plays an important role in the formation of neuromuscular junctions in the peripheral nervous system.³⁶ Therefore, NO was regarded by many scholars as a mostly possible factor in reducing spasticity and improving muscle stiffness.

Previous study has reported that the effectiveness of shock wave therapy was of dose dependence,³⁷ which is related to the energy reaching the target area. In these included articles, only 1 examined the high stimulation intensity (.630 mJ/mm²) of shock wave on shoulder external rotator muscles,²⁴ and it only examined the effectiveness in 4 weeks after ESWT. The rest of those literatures all employed the low intensity (.030-.100 mJ/mm²).²⁵⁻²⁹ Therefore, we were not able to identify the optimum energy. The dose dependence does not mean that the increase of treatment time and frequency will lead to a better therapeutic effect. A previous meta-analysis did report that the frequency of 3000 shots was better than 1500 or 2000 shots, but no significant improvement was found with the frequency increased further.³⁸ Further studies with high quality were needed to explore the appropriate stimulation of ESWT.

We aimed to conduct a meta-analysis to combine the results of previous studies to arrive at a summary conclusion. The pooled data immediately after ESWT and 4 weeks after ESWT compared with baseline date all sug-

gested that ESWT had a significant effect on relieving spasticity caused by stroke. But the differences in the subtype of shock wave, therapeutic energy, treatment sessions, and tested muscles might contributed to the varied effects reported in selected studies, leading to a significant level of heterogeneity among the studies.

According to the Cochrane review guidelines, the chi-squared test and the I-square test were performed to assess the impact of study heterogeneity on the results of the meta-analysis. And if severe heterogeneity was presented ($P > .10$ and $I^2 > 50\%$), the random effect model was chosen. Moreover, sensitivity analysis was conducted by deleting each study individually to evaluate the quality and consistency of the results. In our study, we used a random effects model, and the outcomes from sensitivity analysis indicated the stability of the results. Additionally, we did not find any obvious publication bias in our meta-analysis according to the results of Begg's tests or those of Egger's tests.

Generally speaking, various limitations presented in our review should not be ignored. First, the RCTs, which can optimize follow-up and data quality while minimizing the selection bias and other confounding factors, were the best choice for a meta-analysis. But due to the limited number of relevant research, only 6 nRCTs were included in our study. Second, the number of the included studies and the subjects in each of the study were not so sufficient: the smallest study only consisted of 10 patients. Therefore, potential publication bias was likely to exist, although no evidence was obtained from our statistical tests. Third, the language of the studies was limited to English, which may result in potential language bias.

Despite of the shortcomings mentioned above, the results of our study offered some useful information about the positive efficacy of ESWT toward spasticity in poststroke patients. This had great clinical significance, especially for patients who could not tolerate the adverse effects of the previous treatments. In addition, there were no severe adverse effects associated with ESWT.

Conclusions

This meta-analysis found positive treatment effects on poststroke patients immediately and 4 weeks after ESWT in passive movement measured by MAS grades. Moreover, no serious adverse effects were observed in any patients after shock wave therapy. Nevertheless, our current study with some limitations, such as the small sample size, only provided limited quality of evidence; confirmation from a further meta-analysis with large-scale, well-designed RCTs is required.

References

1. Lance J. Disordered muscle tone and movement. *Clin Exp Neurol* 1981;18:27-35.

2. Ryu JS, Lee JW, Lee SI, et al. Factors predictive of spasticity and their effects on motor recovery and functional outcomes in stroke patients. *Top Stroke Rehabil* 2010;17:380-388.
3. Farmer SE, James M. Contractures in orthopaedic and neurological conditions: a review of causes and treatment. *Disabil Rehabil* 2001;23:549-558.
4. Francisco GE, McGuire JR. Poststroke spasticity management. *Stroke* 2012;43:3132-3136.
5. Bakheit AMO, Pittock S, Moore AP, et al. A randomized, double-blind, placebo-controlled study of the efficacy and safety of botulinum toxin type A in upper limb spasticity in patients with stroke. *Eur J Neurol* 2001;8:559-565.
6. Siatkowski RM, Tyutyunikov A, Biglan AW, et al. Serum antibody production to botulinum a toxin. *Ophthalmology* 1993;100:1861-1866.
7. Gracies JM. Physical modalities other than stretch in spastic hypertonia. *Phys Med Rehabil Clin N Am* 2001;12:769-792.
8. Smania N, Picelli A, Munari D, et al. Rehabilitation procedures in the management of spasticity. *Eur J Phys Rehabil Med* 2010;46:423-438.
9. Wang CJ. An overview of shock wave therapy in musculoskeletal disorders. *Chang Gung Med J* 2003;26:220-232.
10. Sun XZ, Zhang ZW. Shock wave lithotripsy for uric acid stones. *Asian J Surg* 2006;29:36-39.
11. Spindler A, Berman A, Lucero E, Braier M. Extracorporeal shock wave treatment for chronic calcific tendinitis of the shoulder. *J Rheumatol* 1998;25:1161-1163.
12. Cacchio A, Giordano L, Colafarina O, et al. Extracorporeal shock-wave therapy compared with surgery for hypertrophic long-bone nonunions. *J Bone Joint Surg Am* 2009;91:2589-2597.
13. Boddeker R, Schafer H, Haake M. Extracorporeal shockwave therapy (ESWT) in the treatment of plantar fasciitis—a biometrical review. *Clin Rheumatol* 2001;20:324-330.
14. Buchbinder R, Green SE, Youd JM, et al. Systematic review of the efficacy and safety of shock wave therapy for lateral elbow pain. *J Rheumatol* 2006;33:1351-1363.
15. Dymarek R, Ptaszkowski K, Słupska L, Halski T, Taradaj J, Rosinczuk J. Effects of extracorporeal shock wave on upper and lower limb spasticity in post-stroke patients: a narrative review. *Top Stroke Rehabil* 2016;23:293-303.
16. Moher D, Liberati A, Tetzlaff J, et al. Preferred Reporting Items for Systematic Reviews and Meta-Analyses: the PRISMA statement. *J Chin Integr Med* 2009;7:889-896.
17. Fu-Mei L, Mohamed S. Correlation of spasticity with hyperactive stretch reflexes and motor dysfunction in hemiplegia. *Arch Phys Med Rehabil* 1999;80:526-530.
18. Gregson J, Leathley M, Moore AP, et al. Reliability of the tone assessment scale and the Modified Ashworth Scale as clinical tools for assessing poststroke spasticity. *Arch Phys Med Rehabil* 1999;80:1013-1016.
19. Charalambous CP. Interrater reliability of a Modified Ashworth Scale of muscle spasticity. *Phys Ther* 1987;67:206-207.
20. Jadad AR, Moore RA, Carroll D, et al. Assessing the quality of reports of randomized clinical trials: is blinding necessary? *Control Clin Trials* 1996;17:1-12.
21. O'Connell D. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomized studies in meta-analyses. *Appl Eng Agric* 2002;18:727-734.
22. Begg CB, Mazumdar M. Operating characteristics of a rank correlation test for publication bias. *Biometrics* 1995;50:1088-1101.
23. Egger M, Minder C. Bias in meta-analysis detected by a simple, graphical test. *BMJ* 1998;315:629-634.
24. Kim YW, Shin JC, Yoon JG, et al. Usefulness of radial extracorporeal shock wave therapy for the spasticity of the subscapularis in patients with stroke: a pilot study. *Chin Med J* 2013;126:4638-4643.
25. Manganotti P, Amelio E. Long-term effect of shock wave therapy on upper limb hypertonia in patients affected by stroke. *Stroke* 2005;36:1967-1971.
26. Dymarek R, Taradaj J, Rosinczuk J. Extracorporeal shock wave stimulation as alternative treatment modality for wrist and fingers spasticity in poststroke patients: a prospective, open-label, preliminary clinical trial. *Evid Based Complement Altern Med* 2016;2016:4648101.
27. Moon SW, Kim JH, Jung MJ, et al. The effect of extracorporeal shock wave therapy on lower limb spasticity in subacute stroke patients. *Ann Rehabil Med* 2013;37:461-470.
28. Santamato A, Micello MF, Panza F, et al. Extracorporeal shock wave therapy for the treatment of poststroke plantar-flexor muscles spasticity: a prospective open-label study. *Top Stroke Rehabil* 2014;21(Suppl 1):S17-S24.
29. Sohn MK, Cho KH, Kim YJ, et al. Spasticity and electrophysiologic changes after extracorporeal shock wave therapy on gastrocnemius. *Ann Rehabil Med* 2011;35:599-604.
30. Heckmatt JZ, Leeman S, Dubowitz V. Ultrasound imaging in the diagnosis of muscle disease. *J Pediatr* 1982;101:656-660.
31. Ward AB. Long-term modification of spasticity. *J Rehabil Med* 2003;60-65.
32. O'Dwyer NJ, Ada L, Neilson PD. Spasticity and muscle contracture following stroke. *Brain* 1996;119(Pt 5):1737-1749.
33. Mariotto S, Cavalieri E, Amelio E, et al. Extracorporeal shock waves: from lithotripsy to anti-inflammatory action by no production. *Nitric Oxide* 2005;12:89-96.
34. Leone JA, Kukulka CG. Effects of tendon pressure on alpha motoneuron excitability in patients with stroke. *Phys Ther* 1988;68:475-480.
35. Bae HS, Lee JM, Lee KH. The effects of extracorporeal shock wave therapy on spasticity in chronic stroke patients. *J Korean Academy Rehabil Med* 2010;34:663-669.
36. Mariotto S, de Prati AC, Cavalieri E, et al. Extracorporeal shock wave therapy in inflammatory diseases: molecular mechanism that triggers anti-inflammatory action. *Curr Med Chem* 2009;16:2366-2372.
37. Kim JH, Kim JY, Choi CM, et al. The dose-related effects of extracorporeal shock wave therapy for knee osteoarthritis. *Ann Rehabil Med* 2015;39:616-623.
38. Lu Z, Lin G, Reed-Maldonado A, et al. Low-intensity extracorporeal shock wave treatment improves erectile function: a systematic review and meta-analysis. *Eur Urol* 2017;71:223-233.