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CLINICAL CASE REPORT

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# Three Case Reports of Successful Vibration Therapy of the Plantar Fascia for Spasticity Due to Cerebral Palsy-Like Syndrome, Fetal-Type Minamata Disease

*Fusako Usuki, MD, PhD and Satsuki Tohyama, BHS*

**Abstract:** Fetal-type Minamata disease is caused by the exposure to high concentrations of methylmercury in the fetal period and shows cerebral palsy-like clinical features. Relief of spasticity is a major task of rehabilitation to improve their activities of daily living.

Here we report the effect of long-term vibration therapy on bilateral lower-limb spasticity in 3 patients with fetal-type Minamata disease. We used a simple, inexpensive, and noninvasive approach with hand-held vibration massagers, which were applied to the plantar fascia at 90 Hz for 15 minutes.

The effect was observed soon after the first treatment and resulted in better performance of the repetitive facilitation. Vibration therapy for 1 year improved Modified Ashworth Scale for the ankle flexors in 2 cases. The labored gait improved and gait speed increased in another case. Continued vibration therapy for another 1 year further improved Modified Ashworth Scale score and range of motion of ankle dorsiflexion in 1 case. This case showed the decreased amplitude of soleus H-reflex after the 15-minute vibration therapy, suggesting that α-motor neuron excitability was suppressed.

Vibration therapy using a hand-held vibration massager may offer safe and effective treatment for lower-limb spasticity in patients with chronic neurological disorders.

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**Abbreviations**: ADL = activities of daily living, A-ROM = active range of motion, FMD = fetal-type Minamata disease, MAS = Modified Ashworth Scale, MeHg = methylmercury, P-ROM = passive range of motion.

## INTRODUCTION

"Spasticity" is defined as a velocity-dependent increase in muscle tone that is usually caused by damage to the central nervous system, which controls voluntary movement. Disease conditions that cause spasticity include cerebral palsy, stroke, multiple sclerosis, traumatic brain injury, and so on. Continuous spasticity causes various signs and symptoms such as muscle stiffness and shortening, muscle fatigue, pain or tightness around the joints, abnormal posture, and joint contractures. Relief of spasticity is a major task of rehabilitation to improve patients' activities of daily living (ADL). Although botulinum-toxin injections[1] and neurosurgical intrathecal baclofen pump implantation[2] have been recently developed as effective approaches, both have some problems: the former is expensive and limited by the dose and number of injections that can be administered, and also the short shelf life, whereas the latter requires a specialized technique and is invasive. Therefore simpler, noninvasive, and widely available methods are needed to attenuate spasticity.

Fetal-type Minamata disease (FMD) is caused by methylmercury (MeHg) intoxication in fetal period in utero because MeHg accumulates in the brain of the fetus by crossing the placenta and the blood–brain barrier via amino acid transporters.[3] FMD shows cerebral palsy-like clinical features (delay of psychomotor development, motor dysfunction, abnormal muscle tone, and involuntary movement) with mental disturbance.[4] Recently, we have reported that vibratory stimulation of the plantar fascia using a hand-held vibration massager attenuated severe planter pain and lower-limb spasticity in 1 patient with FMD.[5] The spherical-shaped head of the frequency generator used in that study fitted the plantar curvature. The method was inexpensive, noninvasive, simple, and convenient. The vibratory stimulus has a history of influencing not only pain[6] but also muscle tone.[7] However, the vibration therapy has not been widely used clinically yet.

Here, we present 3 cases of FMD who received vibratory stimulation generated by a hand-held vibration massager to plantar fascia to draw lessons from the treatment experience. We demonstrated that long-term vibration therapy of the plantar fascia effectively provides a relief of lower-limb spasticity and improves motor function of patients with FMD.

## CLINICAL FINDINGS, INTERVENTIONS, AND OUTCOME

Three patients with FMD first received vibration therapy at 48, 53, or 54 years of age. Case 1 was diagnosed at 7 years of age by high MeHg concentration of his umbilical cord. Cases 2 and 3 were diagnosed as probably having FMD by clinical findings and epidemiological data, though MeHg concentration of their umbilical cords could not be estimated. All of them showed delay of psychomotor development and cerebral palsy-like neurological signs with bilateral lower-limb spasticity, although their severity and clinical features were different. Case 1 had the most severe clinical features with mild systemic dystonia. Detailed clinical features of case 1 were previously presented.[5] Case 2 showed mild clinical features without involuntary movement. Case 3 showed labored unstable gait on toes. He had difficulty in flexing his knee in walking and often fell down when walking. Although past interventions for spasticity included oral medications and conventional

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From the Department of Clinical Medicine, National Institute for Minamata Disease, Minamata, Kumamoto, Japan.
Correspondence: Fusako Usuki, Department of Clinical Medicine, National Institute for Minamata Disease, 4058-18 Hama, Minamata, Kumamoto 867-0008, Japan (e-mail: usuki@nimd.go.jp).
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**TABLE 1.** Changes in MAS Score, ROM, and Motor Function After Vibration Therapy

| Patient No. | | 1 | 2 | 3 |
|---|---|---|---|---|
| Age, sex | | 53 years, M | 48 years, M | 54 years, M |
| MAS | Before | R 3 L 3 | R 2 L 1+ | Undeterminable |
| P-ROM | Before | R -10° L 0° | R 0° L 20° | R 20° L 20° |
| A-ROM | Before | R -10° L 0° | R 0° L 20° | R 20° L 20° |
| Ambulation | | W/C | W/C due to deformed hip joint, R ankle brace | Labored Unstable gait, walking on toes |
| MAS | After (1Y) | R 2 L 2 | R 1+ L 1 | Undeterminable |
| | (2Y) | R 1+ L 1 | n.e. | n.e. |
| P-ROM | After (1Y) | R -10° L 0° | R 0° L 20° | R 20° L 20° |
| | (2Y) | R 10° L 20° | n.e. | n.e. |
| A-ROM | After (1Y) | R -10° L 0° | R 0° L 20° | R 20° L 20° |
| | (2Y) | R 10° L 20° | n.e. | n.e. |
| Change in motor function | | Standing and transfer steadiness | R ankle brace off | Improved gait unsteadiness |

A-ROM = active range of motion, L = left, MAS = Modified Ashworth Scale, n.e. = not examined, P-ROM = passive range of motion, R = right, W/C = wheel chair.

rehabilitation such as stretching and limb-positioning in case 1, spasticity and ADL progressively got worse. Cases 2 and 3 had no intervention for spasticity. Clinical features of 3 cases before vibration therapy were shown in Table 1. The ambulation difficulty of case 2 was due to his right deformed hip joint. He had a brace on his right ankle. Although case 3 showed spasticity, his Modified Ashworth Scale (MAS) score was undeterminable because the muscle tension was often induced by psychological reactions to external stimuli.

Patients were placed in a supine position to relax their muscle tone. The head of a hand-held vibration massager (Thrive MD-01; Thrive Co., Ltd, Osaka, Japan) was fixed with a Velcro tape on the planter fascia as shown in Figure 1. The frequency of the vibratory stimulus was 90 Hz and the amplitude was 1.0 mm peak to peak. The vibratory stimulus was performed for 15 minutes twice a week for case 1 and once a week for cases 2 and 3 depending on their rate of clinic visits. The repetitive facilitation exercise (Kawahira method)[8] of the lower limbs followed after vibration therapy for cases 1 and 2. For case 3, stretch exercises were performed. Kawahira method was not suitable for case 3 because of his psychological reactions to external stimuli. Case 1 continued to receive therapy for more than 2 years. The patients' medications were not changed during the study. We assessed the effects of vibration stimuli on lower-limb spasticity by the changes in the MAS score of the ankle flexor muscle, passive range of motion (P-ROM) and active range of motion (A-ROM) of ankle dorsiflexion, and functional activity. Clinical assessments were performed every 2 to 3 months. For case 3, 10-m straight forward free walking time was measured before and after the vibration therapy for 15 minutes.

The effect of vibratory stimulation on spasticity was observed soon after the first 15-minute treatment and resulted in better performance of the repetitive facilitation. Although spasticity rebound was observed, the duration of attenuation of spasticity became progressively longer. Table 1 shows the changes in the MAS scores, ROM, and motor function 1 or 2 years after vibration therapy. Vibration therapy for 1 year improved MAS score in cases 1 and 2. The right ankle brace was removed in case 2. The labored gait improved and gait speed increased in case 3 (Figure 2). After the intervention for 8 weeks, his free walking velocity became 12 s/10 m, which neared the velocity 10 s/10 m that most people can walk on a crosswalk before the light to change. He became able to flex his knee in walking, resulting in the improvement of gait unsteadiness and a decrease in falling down when walking.

Case 1 continued vibration therapy for more than 2 years and showed further improved MAS score and ROM. His motion behavior of standing and transferring became better balanced due to the improvement of lower-limb spasticity and ankle movement. None of them showed adverse and unanticipated effects caused by vibratory stimulation of the plantar fascia.

An electrophysiological study of the soleus H-reflex was performed in case 1. The right soleus H-reflex was evoked by a stimulus with a 1-millisecond duration and the intensity was set to elicit the maximum H-reflex using a Neuropack S1 Electromyography Unit (MEB-9402; Nihon Kohden Company, Tokyo, Japan). Sixteen H-waves were recorded and analyzed. The amplitude of soleus H-reflex decreased by 50% after the

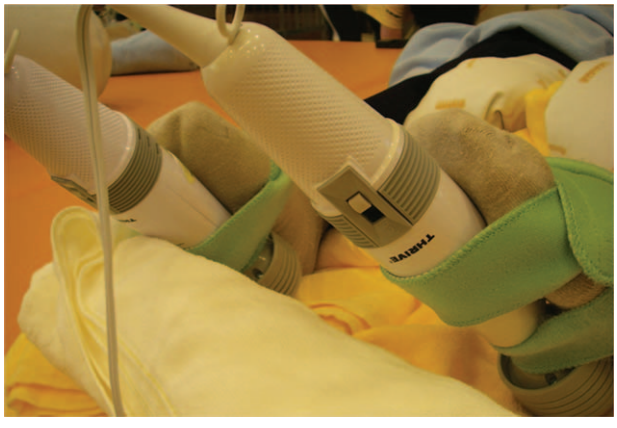

**FIGURE 1.** Vibration therapy of the plantar fascia. A patient was placed in the decubitus position and the head of a hand-held vibration massager was fixed to the planter fascia using Velcro tape.

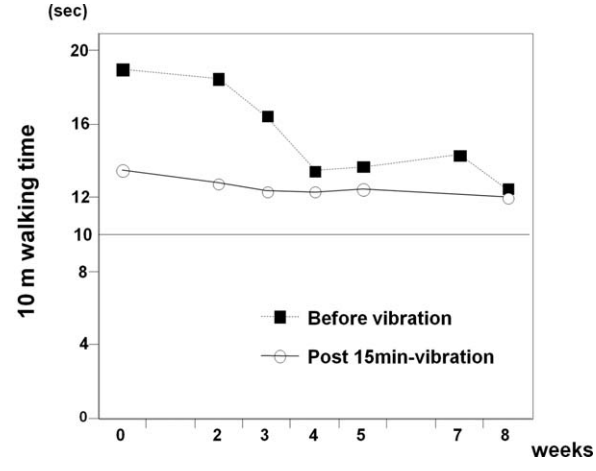


**FIGURE 2.** Change in 10-m straight forward free walking time after the intervention of vibration therapy in case 3.

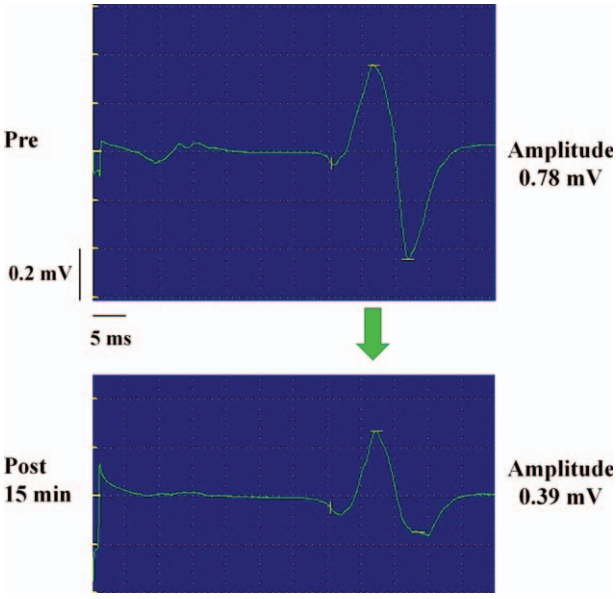


**FIGURE 3.** Attenuated amplitude of soleus H-reflex after vibration therapy of the plantar fascia at 90 Hz for 15 minutes in case 1. The averaged wave was demonstrated.

15-minute vibration therapy (Figure 3), indicating that α-motor neuron excitability was suppressed.

Informed consent for this study was obtained from the subjects according to the ethical guidelines of the National Institute for Minamata Disease.

## DISCUSSION

In this study, we demonstrated that long-term vibration therapy of the plantar fascia was effective for improving lower-limb spasticity and motor function of 3 patients with chronic FMD. The different improvements in 3 patients after vibration therapy observed in this study may be due to their different severity and clinical features. The device was a commercial hand-held vibration massager that could generate an effective frequency of 90 Hz. Since the method is inexpensive, noninvasive, and convenient, we believe that this approach could be widely used for lower-limb spasticity in patients with chronic neurological disorders.

The effects of vibration therapy of the plantar fascia were assessed using MAS scores of the ankle flexor muscle, and also P-ROM and A-ROM of ankle dorsiflexion. Ankle movements are essential for walking and weight-bearing. MAS score improved 1 year postintervention in 2 cases, and P-ROM and A-ROM were improved 2 years postintervention in 1 case who continued vibration therapy. We applied the repetitive facilitation exercise (Kawahira method) subsequently after vibration therapy to enhance voluntary ankle dorsiflexion. The improved A-ROM in case 1 suggests that vibration therapy enhanced the effect of the Kawahira method by suppressing spinal α-motor neuron excitability. Further, vibration therapy improved gait unsteadiness in case 3; labored gait improved and gait speed increased. The previous study demonstrated the importance of vibration frequency of 80 Hz to activate the cerebral cortex motor area.[9] As such, vibration stimulation of the plantar fascia with a frequency of 90 Hz may also work to coordinate motor function by stimulating the cerebral cortex and cerebellum. Further studies of the effect of vibratory stimulation on the central nervous system will uncover the mechanism.

Since the tonic vibration reflex was described,[7] vibratory stimulation has been traditionally applied to the antagonist of the spastic muscle. But the direct application to the spastic muscle using the same device as ours has been recently reported to be effective for short-term attenuation of spasticity in patients with stroke.[10] Here we applied vibratory stimulation to the plantar fascia because our previous FMD study demonstrated that the application of vibratory stimulation to the plantar fascia was more effective than that to the spastic muscle directly.[5] The mechanoreceptor reactive to vibration stimuli with a frequency >80 Hz has been known to be the Pacinian corpuscles. The Pacinian corpuscles, which are widely distributed in the mammalian body, were found in the dermis and subcutaneous tissues of the fingers, palm, and sole in addition to the muscles and tendons.[11–13] The effectiveness of vibration therapy may be dependent on the individual difference in the distribution of the Pacinian corpuscles.

In this study, we applied vibratory stimulation for 15 minutes. The duration of the vibration therapy of the plantar fascia for 10 minutes in case 1 induced an adverse increase in the amplitude of the H-reflex, an index of spinal motor neuron excitability (data not shown). Motor unit activity is determined by the integration of excitatory and inhibitory neural inputs to the α-motor neuron; activation of the cerebral cortex, Ib afferent from the Golgi tendon organ via the interneurons, and Ia afferent from the intrafusal muscle spindle that receives presynaptic inhibition.[14] Ia fibers have been found to be sensitive to vibratory stimulation,[15] and excitatory input to the α-motor neurons from Ia afferents is enhanced by brief vibration but suppressed by prolonged vibratory stimulation.[14] The decreased activities of the reflection circuit and motor neuron by prolonged vibratory stimulation are thought to be due to increased discharge thresholds of the Ia fibers,[16] presynaptic inhibition of the Ia terminals,[17] and transmitter depletion at the Ia terminals.[18] The previous autopsy study demonstrated a decreased number and morphological changes of the mechanoreceptors in the myotendinous junction of the spastic paralyzed human muscle.[19] Further, experimental study demonstrated that unloading hindlimb caused a decrease in calbindin-D28, a marker of mechanosensory afferent nerve terminals, in intrafusal fibers, and the impairment of muscle spindle.[20] The long duration of the vibratory stimulation for the suppression of H-reflex amplitude may be needed due to a decreased mechanoreceptor number and its impaired function in patients with chronic spasticity.

In conclusion, we demonstrated that long-term vibration therapy of the plantar fascia using a hand-held vibration massager at a frequency of 90 Hz relieved lower-limb spasticity and improved motor function in 3 patients with cerebral palsy-like syndrome, FMD. This approach is inexpensive, noninvasive, and available anywhere. It could be widely used for lower-limb spasticity in patients with chronic neurological disorders.

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