FUNCTIONAL ELECTRIC STIMULATION (FES) has been used to restore motor function in patients with upper motoneuron lesions caused by spinal cord injury (SCI) or stroke.1-4 In the first trial of FES, Liberson et al5 reported that hemiplegic patients improved their walking when the peroneal nerve was stimulated in synchronization with the swing phase of gait. Muscles paralyzed by upper motoneuron injury can be electrically activated through their surviving motor nerve supply, thus providing the possibility of improving impaired motor function by FES.

Many people with upper motoneuron lesions have difficulty making useful muscle contraction (eg, standing, walking) because their muscles are atrophied and weak.6 The forces exerted by any muscles on external objects may be limited either because input from higher brain centers to muscles motoneurons have been disrupted by the injury and/or because they have an impaired ability to activate the muscles motoneurons maximally during voluntary contraction. To prevent muscle atrophy, it is important to keep the muscle power when restoring paralyzed extremities with FES. To restore ambulatory function in paraplegic patients, therapeutic electric stimulation (TES) has been performed to restore muscle strength and to determine the tolerance of muscle fatigue before FES is applied.7 TES activates the neuromuscular junction either directly or by going through the peripheral nerves, and thus affects restoration of muscle strength.8 If muscle atrophy has occurred before TES is started, it will require a long period of time before the muscles return to near normal condition.

A few authors have reported on muscle atrophy9 in the acute phase of SCI10,11 Animal studies show that immobilization of the lower limbs for different periods results in muscle atrophy ranging from 15% to 70% of the original muscles, which mostly occurs in the first 7 days of immobilization.12-14 TES, to be helpful in protecting the muscle from atrophy after SCI, should be performed during the acute phase. However, to date, TES has been clinically used only in the chronic phase of SCI. Kagaya et al10 reported that the cross-sectional area of muscles and the muscles’ torque and force were increased at 25 weeks after TES in chronic paraplegic patients. According to their report, it was 6 months before the muscles were improved enough to permit FES. We believe that SCI patients can start FES earlier if muscle atrophy does not develop. The effect of TES on acute muscle atrophy needs to be further investigated.

The stimulation frequency for TES could be another important factor in reducing muscle atrophy. Clinically, low-frequency stimulation (20Hz) has also been applied for TES; Handa3 recommended a frequency of about 20Hz in clinical FES use. A suitable frequency for TES should be systematically analyzed because some muscle fatigue has been observed in clinical use.

When FES is clinically applied, low frequencies of about 20Hz are commonly used; however, Matsunaga et al12,13 suggested that high frequency had advantages in the closed-loop control used for reducing muscle fatigue. One advantage is a rapid response of muscle contraction, and a second is a strong contraction force. The closed-loop control system for restoration of the standing position is useful in minimizing muscle

The Effects of Therapeutic Electric Stimulation on Acute Muscle Atrophy in Rats After Spinal Cord Injury

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ABSTRACT. Misawa A, Shimada Y, Matsunaga T, Sato K. The effects of therapeutic electric stimulation on acute muscle atrophy in rats after spinal cord injury. Arch Phys Med Rehabil 2001;82:1596-603. Objectives: To evaluate the effects of electric stimulation in preventing acute muscle atrophy after spinal cord transection in rats. Design: A randomized experimental design. Setting: Animal facilities for experimental medicine. Animals: Fifty-six adult male Wistar rats assigned to control, low-frequency, and high-frequency groups. Interventions: The rats were implanted with a percutaneous intramuscular electrode in the vicinity of the peroneal nerve; then the spinal cord was transected at a T9 level. The stimulation frequency was low (20Hz) or high (100Hz). The stimulation cycle was 4 seconds of stimulation every 8 seconds. Main Outcome Measurements: The lesser fiber diameters from type 1, 2A, and 2B muscle fibers were measured. In another assessment, maximal contraction force was measured. The muscle force produced at 20 and 100Hz was expressed as increasing values in tetanic force. Results: Comparison between nonstimulated and stimulated tibialis anterior muscles found that atrophy of type 1 fibers (p < .01) and type 2B fibers (p < .05) at both stimulated levels and of type 2A fibers at 100-Hz level (p < .05) was prevented by therapeutic electric stimulation (TES). There were significant differences in the size of muscle fiber diameter between nonstimulated and stimulated muscles at 100Hz in type 2A and, markedly, in type 2B. The increasing value of muscle force was significantly greater at 100Hz than at 20Hz (p < .05). No significant histologic differences were observed between high- and low-frequency stimulated fibers of any of the 3 muscle types. Conclusions: Acute atrophy of muscle fibers was more effectively prevented by high-frequency stimulation (100Hz) than by no stimulation or low-frequency stimulation (20Hz). The increasing value of muscle force was significantly greater at high-frequency than low-frequency stimulation, suggesting that the clinical application of high-frequency stimulation in acute spinal cord injury should be studied. Key Words: Electric stimulation; Muscular atrophy; Rats; Wistar; Rehabilitation; Spinal cord injuries.

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fatigue caused by electric stimulation and in preventing knee buckling from the feed-back mechanism.\textsuperscript{16} During intermittent stimulation in Matsunaga’s study, muscle fatigue was greater at lower frequencies than at higher frequencies, meaning that higher frequency stimulation was more useful in preventing muscle fatigue.\textsuperscript{15} On the basis of these results, we expected that high-frequency stimulation could elicit muscle contraction with less fatigue. Therefore, intermittent and high-frequency therapeutic electric stimulation would be an effective way to minimize muscle atrophy. This study evaluated the effect of electric stimulation in preventing acute muscle atrophy after spinal cord transection in rats.

METHODS

Surgical Procedures

Forty adult male Wistar ST rats with an average body weight of 448g (range, 380–550g) were used in these experiments. The animals were assigned to 1 of 3 groups: the control group (C group, \( n = 5 \)); the 20-Hz stimulation (low-frequency) group (20Hz group, \( n = 15 \)); and the 100-Hz stimulation (high-frequency) group (100Hz group, \( n = 15 \)). Rats in the C group did not have the spinalization and their right hindlimb was studied. All animals were kept individually in metal cages and received standard rations of food and water.

For the spinalization, the rats were deeply anesthetized with an intraperitoneal injection of 40mg/kg of body weight of pentobarbital sodium. A percutaneous intramuscular electrode,\textsuperscript{7} which has been used in clinical FES, was implanted unilaterally (in the right leg) in the vicinity of the peroneal nerve to stimulate tibialis anterior and extensor digitorum longus. The left limb served as an unoperated control. The percutaneous electrode was a helical coil wound from a Teflon®-coated, 19-strand, stainless steel cable (SES114a).\textsuperscript{2,17} The cable consisted of SUS 316L-type hard-drawn stainless steel wires; the diameter of a single stainless steel wire was 25\( \mu \)m. The diameter of the cable was about 0.4\( \mu \)m. The electrode leads led under the skin and through an incision in the rat’s back, where they were attached to a miniature plug. The plug, attached to the proximal ends of the electrodes, was secured at incision site on the rat’s back and it did not interfere with its movements.\textsuperscript{18} The wounds on the rats’ backs were closed after the leads were implanted. The rats could be connected to a stimulator by a length of flexible lead so that their freedom of movement was not restricted. After the electrode was implanted, the rat was incised dorsally to expose the T9–10 vertebrae. The muscular insertions on the posterior and transverse processes were dissected and cut. A T9 laminectomy was done, and the spinal cord was transected; the lower limbs were then paralyzed\textsuperscript{19} (fig 1). The animal care protocol for this study was approved by our university’s institutional animal care and use committee and followed the guidelines of the US National Institutes of Health (NIH).

Stimulation Protocol

Electric stimulation was started 1 day after the spinalization. Tibialis anterior and extensor digitorum longus were stimulated for 60min/d, at frequencies of 20 or 100Hz, for 1 week. At either frequency, the pulses had negative square monophasic waveforms, pulse width was 0.2ms, and constant voltage was adjusted to give maximum contraction force. The stimulation cycle was 4 seconds on, 4 seconds off for 60 minutes.

Histochemical Methods

The day after the stimulation period ended, the rats were anesthetized, and the tibialis anterior and extensor digitorum longus muscles from both legs were surgically removed. Muscle samples were taken from the maximum circumference part of each muscle in a 10-mm thick cross-section. Subsequent samples were taken using a 90° cut cross-section of this sample. The muscles were then rapidly frozen in 2-methylbutane (isopentane) and cooled in liquid nitrogen. Each sample was stored at \(-80^\circ \text{C}\) until it was analyzed. Samples were then cut into 10-\( \mu \)m thick serial sections, with the cryostat maintained at \(-18^\circ \text{C}\). Transverse serial sections were stained using a histochemical method (adenosinetriphosphatase) with preincubation at pH 4.4. Type 1, type 2A, and type 2B fibers were identified according to the criteria of Brooke and Kaiser\textsuperscript{10} and Dubowitz and Brooke\textsuperscript{21} (fig 2), and the lesser fiber diameters of 300 fibers from each muscle fiber type were measured with an NIH image. We followed the distribution of the muscle types, and then we counted and measured muscle fibers.\textsuperscript{22-24} This measurement is designed to overcome the distortion that occurs when a muscle fiber is cut obliquely, producing an oval appearance.\textsuperscript{23} The data are reported as mean \( \pm \) standard deviation.
(SD). All data were analyzed statistically using a 2-factor factorial analysis of variance (ANOVA). Criterion for significance was \( p \) less than .01. The difference in the lesser diameters at 20 and 100Hz in each muscle type were statistically evaluated by Fisher’s protected least significant difference (Fisher’s PLSD). This criterion for significance was \( p \) less than .05.

**Physiologic Assessment**

Sixteen adult male Wistar ST rats with average body weight of 497g (range, 400–580g) were used in these experiments. We grouped the rats into a low-frequency group stimulated at 20Hz and a high-frequency group stimulated at 100Hz; each group had 8 rats. The procedure for implanting electrodes and spinalization was as described earlier. After the stimulation, the isometric force was measured for the tibialis anterior and extensor digitorum longus of both hindlimbs. These muscles were carefully exposed so as not to disturb their blood or nerve supply. Two hook electrodes, made from silver chloride, with a 5-mm distance between the poles, were attached to the common peroneal nerve about 1cm proximal from the neuromuscular junction. The distal tendon of the tibialis anterior or extensor digitorum longus was attached to an isometric force transducer. The tibia was secured to a rigid fixator to ensure no limb movement during testing. Throughout the experiment, the muscle was kept at the natural length using the resistance of 1N. The tibia was secured to a rigid fixator to ensure no limb movement during testing. Throughout the experiment, the muscle was kept at the natural length using the resistance of 1N. The tibia was secured to a rigid fixator to ensure no limb movement during testing. Throughout the experiment, the muscle was kept at the natural length using the resistance of 1N.

**RESULTS**

**The Lesser Fiber Diameter in Each Fiber Type**

**Tibialis anterior.** The mean population of type 1, type 2A, and type 2B muscle fibers \( \pm SD \) were, respectively, 4.7% \( \pm \) 0.7%, 43.1% \( \pm \) 2.7%, and 52.1% \( \pm \) 2.3% in the C group; 4.1% \( \pm \) 1.5%, 43.8 \( \pm \) 4.1%, and 52\% \( \pm \) 3.7% in the nonstimulated muscle of the 20Hz group; 4.1\% \( \pm \) 2.7%, 44.7\% \( \pm \) 5.0%, and 51.1\% \( \pm \) 5.0% in the stimulated muscle of the 20Hz group; 4.0\% \( \pm \) 2.6%, 44.4\% \( \pm \) 6.0%, and 51.1\% \( \pm \) 7.3% in the nonstimulated muscle of the 100Hz group; and 4.1\% \( \pm \) 1.0%, 42.6\% \( \pm \) 0.6%, and 53.2\% \( \pm \) 5.1% in the stimulated muscle of the 100Hz group. There was no significant change in the population of muscle fibers at each group (2-factor factorial ANOVA, \( p = .6930 \)).

The lesser diameters of type 1, type 2A, and type 2B muscle fibers were, respectively, 31.85 \( \pm \) 2.76\( \mu \)m, 35.00 \( \pm \) 2.11\( \mu \)m, and 43.73 \( \pm \) 3.81\( \mu \)m in the C group; 25.40 \( \pm \) 2.11\( \mu \)m, 32.17 \( \pm \) 6.94\( \mu \)m, and 37.30 \( \pm \) 3.54\( \mu \)m in the nonstimulated muscle of the 20Hz group; 28.07 \( \pm \) 3.73\( \mu \)m, 30.74 \( \pm \) 3.26\( \mu \)m, and 40.94 \( \pm \) 4.55\( \mu \)m in the stimulated muscle of the 20Hz group; 26.88 \( \pm \) 2.30\( \mu \)m, 29.71 \( \pm \) 2.63\( \mu \)m, and 39.50 \( \pm \) 4.06\( \mu \)m in the nonstimulated muscle of the 100Hz group; and 29.61 \( \pm \) 2.92\( \mu \)m, 35.00 \( \pm \) 4.70\( \mu \)m, and 49.44 \( \pm \) 5.59\( \mu \)m in the stimulated muscle of the 100Hz group (fig 4).

In a comparison between control muscles and experiment muscles, the nonstimulated muscles of both groups and the stimulated muscles of the 20Hz group in type 1 were smaller than those in the C group (2-factor factorial ANOVA, all comparisons \( p < .01 \)); however, there was no significant difference between the C group and the stimulated muscles in the 100Hz group. There was no significant difference between the C group and all of the muscles in type 2A. In type 2B, nonstimulated muscles in the 20Hz group were smaller than those in the C group (2-factor factorial ANOVA, \( p < .01 \)); however, stimulated muscles in the 100Hz group were larger than those in C group (2-factor factorial ANOVA, \( p < .01 \)).
and there were no significant differences between the other muscles in C group.

From the statistical calculation of the lesser fiber diameters, stimulation had a significant effect on muscle fiber size (2-factor factorial ANOVA, p < .0001); the frequency significantly affected muscle fiber size (2-factor factorial ANOVA, p < .0001). Therefore stimulation affected muscle fiber size independently. The stimulated muscles were significantly larger than the nonstimulated muscles of both groups in type 1 (Fisher’s PLSD, both p < .05). There was a significant difference in the size of muscle fiber diameter between the nonstimulated and stimulated muscles in the 100Hz group (Fisher’s PLSD, p < .05); however, there was no significant difference in the 20Hz group in type 2A. In addition, there was a significant difference in the stimulated muscles between the 2 frequencies (Fisher’s PLSD, p < .01) in type 2A. There was a significant difference in the size of muscle fiber diameter between nonstimulated and stimulated muscles of both groups (Fisher’s PLSD, 20Hz, p < .05; 100Hz, p < .01) and between the other muscles in the C group. In type 2B, nonstimulated muscles of both groups (2-factor factorial ANOVA, both p < .01) and stimulated muscles of the 20Hz group (2-factor factorial ANOVA, p < .01) were smaller than the C group muscles; however, there was no significant difference between the C group and the stimulated muscles in the 100Hz group.

From a statistical calculation of the lesser fiber diameters, stimulation had a significant effect on muscle fiber size (2-factor factorial ANOVA, p < .0001); the frequency significance did not affect muscle fiber size in extensor digitorum longus. The stimulation affected muscle fiber size independently. There was no significant difference in muscles in either group in type 1 muscle fiber. There was significant difference between stimulated and nonstimulated type 2A muscle fiber in the 100Hz group (Fisher’s PLSD, p < .01); however, there was no significant difference in the 20Hz group and between the 2 frequencies in type 2A. There was a significant difference between stimulated and nonstimulated type 2B muscle fiber in both groups (Fisher’s PLSD, both p < .01). There was no significant difference of stimulated muscles between the 2 frequencies in type 2B.

Stimulus Frequency

The force produced at different frequencies was expressed as a percentage of the maximum tetanic force and plotted in linear scales (figs 6A, B; 7A, B). The tension-frequency curve had a steep section where rate modulation of muscle force output was the most efficient (figs 6, 7), and this part did not move to the
right or the left or toward either of the imposed frequencies in the tibialis anterior and extensor digitorum longus.

Muscle Tension

At 20Hz, the increasing value of the tibialis anterior in the 20Hz group was 123.14% ± 45.33%, and of the extensor digitorum longus, it was 156.14% ± 111.94%. Also at 20Hz, the increasing value of the tibialis anterior in the 100Hz group was 179.15% ± 92.66%, and of the digitorum longus, it was 148.61% ± 22.24%. From the statistical calculation of increasing value, there was significant difference in tibialis anterior between 20 and 100Hz (Fisher’s PLSD, \( p < .05 \)); however, there was no significant difference in extensor digitorum longus (fig 8).

At 100Hz, the increasing value of the tibialis anterior in the 20Hz group was 118.25% ± 10.13%, and of the extensor digitorum longus, it was 124.68% ± 22.39%. Also at 100Hz, the increasing value of the tibialis anterior in the 100Hz group was 188.10% ± 103.45%, and of the extensor digitorum longus, it was 216.06% ± 24.91%. There was a significant difference in tibialis anterior and extensor digitorum longus between the 2 frequencies (Fisher’s PLSD, \( p < .05 \)) (fig 9).

DISCUSSION

In this study, we assessed muscle atrophy in each frequency group from the size of the muscle fiber and the muscle force. We considered being able to make assumptions with regard to translating the muscle fibers effects in rats to humans based on the muscle characters.\(^{12,20,27}\) There was no significant change in the population of type 1, type 2A, and type 2B muscle fibers at 20Hz (Fisher’s PLSD, \( p < .05 \)) however, there was no significant difference in extensor digitorum longus (fig 8).

At 100Hz, the increasing value of the tibialis anterior in the 20Hz group was 118.25% ± 10.13%, and of the extensor digitorum longus, it was 124.68% ± 22.39%. Also at 100Hz, the increasing value of the tibialis anterior in the 100Hz group was 188.10% ± 103.45%, and of the extensor digitorum longus, it was 216.06% ± 24.91%. There was a significant difference in tibialis anterior and extensor digitorum longus between the 2 frequencies (Fisher’s PLSD, \( p < .05 \)) (fig 9).

**Fig 5.** The mean data ± SD of the lesser diameters (y axis) of type 1, type 2A, and type 2B fibers from extensor digitorum longus in the groups of the control, 20Hz, and 100Hz. There was a significant difference between stimulated and nonstimulated type 2B muscle fibers at 20Hz. There was a significant difference in the size of muscle fiber diameter between nonstimulated and stimulated muscles at 100Hz in type 2A and type 2B. * \( p < .05 \); ** \( p < .01 \).

**Fig 6.** (A) The tension-frequency curve in tibialis anterior stimulated at 20Hz. (B) The tension-frequency curve in extensor digitorum longus stimulated at 20Hz. The force produced at different frequencies is expressed as a percentage of the maximum tetanic force (y axis). The x axis is the stimulated frequency. The tension-frequency curve for both muscles did not move to the right or the left. NOTE: Values are mean ± SD.
was prevented by TES. We suggest that the high-frequency stimulation of TES in the acute phase of SCI is more effective in reducing atrophy for type 2 fiber than is low-frequency stimulation. The increase value of muscle force was greater with high-frequency stimulation than low-frequency stimulation, suggesting that high-frequency stimulation of acute atrophied muscles is more effective in increasing muscle force.

In animal models of disuse, such as in spaceflight,28,29 after spinal cord transection,30 and hindlimb suspension,31 muscle atrophy occurs at an extremely high rate for the first several months.28,29,31 A recent study32 has shown that thigh girth decreases up to 50% within 3 weeks after SCI, suggesting that atrophy is virtually complete within the first month after SCI. Significant atrophy continues well into the ninth month post injury. Animal studies have also shown that muscle fiber cross-sectional areas can decrease by up to 45% in some rat muscles after 28 days of hindlimb suspension.30 Muscle atrophy in SCI patients has been demonstrated with a progressive decrease in the fiber diameter and changes in the fiber type distribution, with predominant type 2 atrophy in the early stages and type 1 atrophy in the later stages of the cord transection.11

Type 2B fibers have the largest twitch and tetanic tension, and they show signs of fatigue in repetitive stimulation. Type 2A fibers have intermediate-sized twitch and tetanic tensions are resistant to fatigue in repetitive stimulation, whereas type 1 muscles have the smallest twitch and tetanic tensions.27

To restore paralyzed muscles by FES, an increase in muscle power is required.6,33 It is important to maintain muscle power by increasing the size of muscle fiber. If muscle atrophy develops, recovery from it requires a much longer period of time. This is why muscles must be stimulated before atrophy develops. Physiologically, type 1 muscle fibers (slow muscle) were stimulated at low frequency (10–20Hz), and type 2 muscle fibers (fast muscle) were stimulated at high frequency (30–60Hz) by depending on the nerve supply.34 In our results, there were significant differences in muscle fiber diameters between nonstimulated and stimulated muscles at 100Hz in types 2A and 2B. Although there is no significant difference at 20 or 100Hz in type 1 muscle, the positive effects on type 2 muscle were shown in this study. In the acute phase, type 2 muscle fibers are smaller, but the atrophy is not progressing quickly during high-frequency stimulation.

Fig 7. (A) The tension-frequency curve in tibialis anterior stimulated at 100Hz. (B) The tension-frequency curve in extensor digitorum longus stimulated at 100Hz. The tension-frequency curve for both muscles did not move to the right or the left. NOTE. Values are mean ± SD.

Fig 8. Increasing values at 20Hz. Increasing values (%) = Ts/Tn × 100 where Ts is the tetanic force of stimulated muscle and Tn is the tetanic force of nonstimulated muscle. There was a significant difference in tibialis anterior between 20 and 100Hz. NOTE. Values are mean ± SD. Abbreviations: 20 TA, 20Hz tibialis anterior; 100 TA, 100Hz tibialis anterior; 20 EDL, 20Hz extensor digitorum longus; 100 EDL, 100Hz extensor digitorum longus. * p < .05.

Fig 9. Increasing values at 100Hz. There was a significant difference in tibialis anterior and extensor digitorum longus between the 2 frequencies. NOTE. Values are mean ± SD. * p < .05.
We measured muscle force stimulated at 1, 2, 5, 10, 20, and 100Hz and evaluated the effects on increasing muscle force in paraplegic rats between stimulation at 20 and 100Hz. To obtain the effect of stimulation, the value of the rate of the mean force produced by nonstimulated muscle and that of the stimulated muscle in each frequency at both 20 and 100Hz were compared. Interestingly, with a low frequency of stimulation applied over a large fraction of the day, Kernell et al.²⁸ found a decrease in force. Brief bursts of high-frequency activity, superimposed on the low-frequency pattern, could prevent this decline. In addition, Edwards et al.²⁶,²⁷ reported that a full tetanus was obtained when the nerves controlling the muscles were maximally stimulated at 50 to 100Hz. Stimulation at 20Hz generates only about 65% of the full force. The wide range of muscle contraction force produced by high-frequency stimulation has a great advantage in muscle exercise. Our results showed a marked change in muscle force when both muscles were stimulated at 100Hz; however, we also found a small increase in muscle force when stimulated at 20Hz, as compared to stimulated control muscles. High-frequency stimulation is more useful in increasing muscle strength.

The force produced at different frequencies is expressed as a percentage of maximum tetanic force and is plotted on linear scales. A logarithmic scale may also be used for frequency. In our study, there was no consistent difference in the muscles stimulated at each frequency. Gorza et al.²⁸ investigated slow-to-fast transformation of denervated rats soleus muscles by chronic high-frequency stimulation. They found that the tension-frequency curves for the stimulated muscles moved progressively to the right as compared with innervated and denervated control soleus muscles. When related to other results, they described how the tension-frequency characteristics of the muscles might be quickly adjusted with altered activity, through producing changes in excitation-contraction during the coupling processes. Our results showed that the tension-frequency characteristics of the muscles had not changed.

We adopted intermittent stimulation for the stimulation cycle. During intermittent stimulation, muscle fatigue was greater at lower frequencies than it was at higher frequencies.¹⁵ This is contrary to results obtained during continuous stimulation. In an earlier study,¹⁵ we suggested that due to the differences in physiologic mechanisms between low-frequency fatigue and high-frequency fatigue, the recovery time during high-frequency stimulation might have an effect on the reduction of muscle fatigue. The mechanism of intermittent stimulation is unclear, however, the condition of high-frequency and intermittent stimulation has a synergism. Edwards³⁷ compared the maintenance of force during prolonged stimulation of normal human quadriceps femoris at between 30 and 100Hz. They reported that during the first 18 seconds of continuous stimulation at 30Hz, the force decreased by 9.7% of the maximal force, whereas at 100Hz, 59.6% of the maximal force was reduced. This suggests that continuous, high-frequency stimulation is not appropriate for increasing muscle force. Type 2 muscle recovery in the acute phase of SCI outpaces fast muscle atrophy with intermittent high-frequency stimulation, and muscle strength increases, in comparison with the use of intermittent low frequency or no stimulation.

CONCLUSION

In the clinical use of TES or FES, low frequencies of about 20Hz are commonly adopted. Our results suggest that high-frequency stimulation for acute atrophied muscles is more effective in reducing muscle atrophy. We suggest that future studies examine the effects of the clinical application of high-frequency stimulation in acute SCI.

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