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# Effect of High Frequency Vibration on Nerve Excitability

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The effect of vibration on the median nerve excitability was studied in 18 healthy subjects by applying the vibrator on the skin overlying either the median nerve or the tendon of flexor digitorum superficialis. The excitability index (EI) was used to assess the median nerve excitability. This index was expressed as the ratio of the amplitude of submaximal evoked compound action potentials (SECAP) before vibration (baseline level) to that during or after the vibration. Comparison between the experimental and the control side showed that vibration over the median nerve did not induce any significant change of EI. However, vibration over the tendon of flexor digitorum superficialis elicited a significant increase of median nerve excitability. In conclusion, the nerve excitability was increased in response to the vibration applied over the tendon, but not over the nerve itself. Activation of the tonic vibration reflex was the probable mechanism. (J Rehab Med Assoc ROC 1999; 27(1): 7-14)

Key words: excitability, nerves, nerve conduction, vibration

#### INTRODUCTION

The effects of vibration on nerve function, such as pain relief <sup>[1,2]</sup>, motor functions <sup>[3-5]</sup>, and occupational hazards <sup>[6]</sup> have been studied previously. Lundeberg suggested that the pain relief obtained with vibration was not associated with release of endogenous opioids, but rather it was related to the gate control theory <sup>[1]</sup>. The vibration stimuli preferably activated large afferent fibers to inhibit smaller pain fibers in the dorsal horn of the spinal cord. The physiological basis of vibratory therapy

on motor dysfunction was related to the alteration of motoneuron excitability from vibratory stimulation <sup>[3-5]</sup>. However, the pathophysiological mechanism of demyelinating neuropathy in chain saw operators secondary to chronic vibration is still unclear <sup>[6]</sup>.

Ertekin et al <sup>[7]</sup> studied the effect of vibration on sural nerve and found that continuous vibration applied to the sural nerve innervated skin area produced an amplitude reduction of the sural nerve action potentials on both painless and painful stimulation conditions. The decrease in the amplitude of sensory nerve was attributed to an impulse blockage of the large diameter afferent

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nerve fibers.

Nordin and Hagbarth <sup>[8]</sup> applied vibration over the finger extensor tendons to study the influence of the tonic vibration reflex (TVR), a reflex elicited by vibratory stimulation of the muscle spindle primary endings resulting in the increase of motoneuron excitability <sup>[3-5, 9-12]</sup>. Although evidence showed that vibratory stimulation acted on sensory nerve terminals (mainly on the muscle afferent fibers, but may also on the cutaneous sensory fibers), whether there was an additional influence directly from the stimulation of motor nerves per se has not been well addressed.

Therefore, the purpose of this study was to determine whether there was any effect on motor excitability by the direct stimulation of nerve itself in addition to the stimulation on peripheral sensory nerves.

# **MATERIALS AND METHODS**

# Subjects

Eighteen healthy volunteer subjects (10 males and 8 females) were recruited for this study. Their age ranged from 24 to 36 years. All of them had no history of neuropathological problems involving the neck or the upper extremity, and free from neurological signs or symptoms at the time of this study.

# General Design

According to the sites of stimulation, the experiment was divided into two parts. One was the nerve vibration and the other was tendon vibration. The study of nerve vibration was to measure the changes in median nerve excitability by applying the vibrator on the skin overlying the median nerve. The other study was to measure the same changes by applying the vibrator on the tendons of flexor digitorum superficialis. All 18 subjects participated in the first part of nerve vibration experiment, while only 15 of them further participated in the second part of tendon vibration experiment due to difficulty in scheduling the subjects. The experiment of tendon vibration was conducted at least 24 hours after the experiment of nerve vibration. For either part of study, the limb of either right or left side was randomly selected experimental measurements (with vibratory

stimulation), and the other side was selected for control (without vibration).

#### Vibrator

The vibrator used in this study was a commercially available device in USA to provide home use for pain control (VIBRO-FLEX, Stoffrengen & Company, Anaheim, California). This vibration device generated a constant frequency of 100 Herz (Hz) and in an amplitude of approximately 0.5 mm. Velcro adhesive pads were attached to the vibrator to strap the forearm circumferentially with a firm and tight contact of the vibrator to the skin at a constant pressure in order to ensure constant vibratory amplitude and to provide the vibratory energy to spread into the underlying deep tissues. The surface of the vibrator is round in shape with a diameter of 3 cm.

# EMG Assessment of Motor Nerve Excitability

Subjects were in supine lying in a relaxed posture with approximately  $20^{\circ}$  of elbow flexion throughout the study <sup>[9]</sup>. An EMG machine was used for the measurement of nerve excitability. Room temperature was at  $24 \pm 2^{\circ}$ C and skin temperature was at  $32 \pm 1^{\circ}$ C.

The arrangement of the recording and stimulating electrodes is shown in Figure 1. For the experiment of nerve vibration, the active recording electrode was placed on the prominence of abductor pollicis brevis muscle and the reference recording electrode (R1 in Figure 1) was placed on the metacarpophalangeal joint of the thumb. The stimulating electrodes, a pair of bar electrodes, were fixed firmly with tape to the medial aspect of the antecubital space, just lateral to the brachial artery (S1 in Figure 1) to stimulate the median nerve. The vibrator (V1 in Figure 1) was applied on the median nerve about half way between stimulating and recording electrodes. With this placement, it might avoid the vibratory effect to the tendon of abductor pollicis brevis (if it was applied on the wrist). During the vibration, all subjects could feel a tingling or vibration sensation in the palmar surface of the thumb, index and middle fingers to ensure that the median nerve was vibrated.

For the experiment of tendon vibration, the active recording electrode (R2 in Figure 1) was placed on the middle portion of the flexor digitorum superficialis muscle and the reference recording electrode was placed 2-3 cm distal to the active recording electrode. The stimulating electrodes (S2 in Figure 1) were placed at the same sites as in the experiment of nerve vibration. The vibrator (V2 in Figure 1) was applied on the tendons of flexor digitorum superficialis about 5 cm above the wrist. The underlying median nerve might be vibrated. However, the distal median nerve (at the site of vibration) is not related to the flexor digitorum superficialis in either motor or sensory connection.

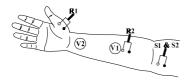


Figure 1. Sites of the stimulatory electrodes, recording electrodes, and vibrator.

Experiment I (nerve vibration):

V1 = site of vibration on the median nerve;

S1 = site of stimulation electrode:

R1= site of recording electrode.

Experiment II (tendon vibration):

V2= site of vibration on the flexor digitorum superficialis tendons;

S2= site of stimulation electrode;

R2= site of recording electrode.

For each experiment, the setting of control measurement was exactly the same as experimental one. The only difference was no vibratory output generated from the vibrator of the control setting (placebo nerve or tendon vibration).

For each experiment, the control measurements were done first. For the control measurements, the readings were recorded at one-minute interval for eight minutes. For the experimental measurement, baseline readings were recorded first at time zero before the vibrator was turned on. The vibrator was then turned on for five minute. The measurements were recorded at one-minute interval for five minutes during the vibration period. The vibrator was then turned off at the end of five minutes. post-vibratory measurements Subsequently, recorded at one-minute interval for another three minutes. The experimental measurements were performed at least fifteen minutes after the control measurements on the other side had been recorded to eliminate the electrical stimulating effect.

The measurement of the median nerve excitability was arranged similarly to the protocol described in other studies previously [13,14]. For each measurement of nerve excitability, the sub-maximal evoked compound muscle action potential (SECMAP) was recorded. The intensity of SECMAP stimulation was adjusted to 50% of supra-maximal stimulation. The stimulating intensity was maintained at a constant level throughout the whole course of each experiment. The amplitude of the SECMAP was measured from the baseline to the first negative peak for each recording and was a sensitive indicator in the change of excitation threshold when the stimuli were repeated at a constant intensity [13,14]. For each measurement in either the control or the experiment study, three SECMAPs were obtained for average.

# Data Analysis

The excitability index (EI) was then calculated by dividing the averaged SECMAP amplitude at each time point over the SECMAP amplitude at time zero (before the vibration). The nerve excitability was considered increased when the EI was greater than one, and vice versa.

Two-way analysis of variance (ANOVA) was used to determine the statistical significance in the changes of EI from vibration as compared to the controls. Statistical significance was considered when the p value was less than 0.05.

#### RESULTS

#### Control Data

The variations in the amplitudes of SECMAPs among 3 consecutive readings in each measurement, both in the control and the experimental group, were minimum. The EI values of the control measurements were found to be grouped around 1.00, ranging from 0.99 to 1.03 in the nerve vibration group and from 0.99 to 1.04 in the tendon vibration group (Table 1, Figure 2). Therefore, this assessment of nerve excitability can be considered reliable and valid.

#### Nerve Vibration

For the nerve vibration, the EI values were also grouped around 1.00, ranging from 0.99 to 1.02 during vibration and 1.00 to 1.02 after vibration (Table 1, Figure 2). Based on ANOVA, there were no significant changes in the median nerve excitability either during or after vibratory stimulation on the skin overlying the median nerve (p > .05) (Table 1).

#### Tendon Vibration

During tendon vibration, the mean EI value increased progressively from 1.22 (at the end of the first

minute) up to 1.40 (after 5 minutes of vibration). Then the mean EI value reduced gradually after cessation of vibration, but not returned the original value 3 minutes later (Table 1, Fig. 2). Based on ANOVA, there were significant changes in the median nerve excitability either during or after vibratory stimulation on the tendon of flexor digitorum superficialis (p < .001) (Table 1).



# **DISCUSSION**

From this study, it has been demonstrated that nerve excitability could be increased by vibratory stimulation on the tendon, but not on the nerve itself.

Table 1. The Effect of Vibration on Median Never Excitability Index

	Nerve Excitability Index (EI $^{a}$ ) (mean $\pm$ SD)		
	Control (no vibration)	Experiment (with vibration)	p value (ANOVA)
Study of Nerve Vibration (N = 18)			
Time zero	1.00	1.00	
1 minutes	$0.99 \pm 0.03$	$0.99 \pm 0.03$	P = 1.00
2 minutes	$1.00 \pm 0.03$	$1.01 \pm 0.05$	p = 0.98
3 minutes	$1.02 \pm 0.03$	$0.99 \pm 0.05$	p = 0.95
4 minutes	$1.03 \pm 0.05$	$0.99 \pm 0.05$	p = 0.93
5 minutes	$1.02 \pm 0.03$	$1.00 \pm 0.04$	p = 0.96
Post 1 minute	$1.02 \pm 0.04$	$1.02 \pm 0.08$	p = 1.00
Post 2 minutes	$1.03 \pm 0.06$	$1.00 \pm 0.05$	p = 0.95
Post 3 minutes	$1.02 \pm 0.03$	$1.02 \pm 0.08$	p = 1.00
Study of Tendon Vibration (N=15)			
Time zero	1.00	1.00	
1 minutes	$0.99 \pm 0.04$	$1.22 \pm 0.28$	<i>p</i> <0.001
2 minutes	$1.00 \pm 0.04$	$1.28 \pm 0.32$	<i>p</i> <0.001
3 minutes	$1.01 \pm 0.07$	$1.32 \pm 0.31$	<i>p</i> <0.001
4 minutes	$1.04 \pm 0.09$	$1.37 \pm 0.36$	<i>p</i> <0.001
5 minutes	$1.03 \pm 0.08$	$1.40 \pm 0.35$	<i>p</i> <0.001
Post 1 minute	$1.02 \pm 0.04$	$1.39 \pm 0.34$	<i>p</i> <0.001
Post 2 minutes	$1.03 \pm 0.05$	$1.33 \pm 0.30$	<i>p</i> <0.001
Post 3 minutes	$1.04 \pm 0.05$	$1.30 \pm 0.26$	P < 0.001

<sup>&</sup>lt;sup>a</sup>EI: The ratio of averaged amplitude of sub-maximal evoked compound muscle action potentials (SECMAP) at different time points (during or after vibration) over the SECMAP at time zero (before vibration) from the same intensity of vibratory stimulation.

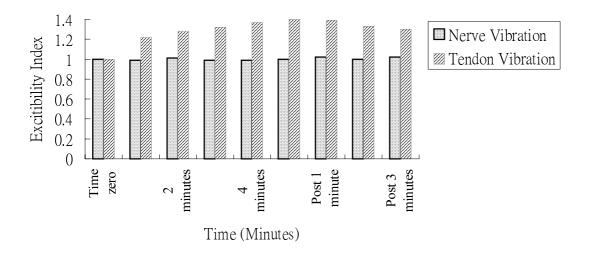


Figure 2. The effects of 100-Hz vibration on median nerve excitability.

#### Technical Issue

In this study, similar to previous studies on tonic vibration reflex [8-10,12,15], all tissues covered tendons or median nerve (including skin, subcutaneous tissue, cutaneous nerves, vessels, muscles) were vibrated simultaneously. Since no change in nerve excitability was found during nerve stimulation, the afferent effect of the vibration to the cutaneous nerve was not an important issue. Cutaneous nerves were also stimulated by vibration in both :nerve vibration" and "tendons vibration" studies. Therefore, it is reasonable to state that nerve excitability could be increased by tendon vibration, but not by nerve stimulation. One may speculate that if median nerve, which is deeply seated in the forearm, is actually vibrated during the study of nerve vibration. In our study, the vibratory surface could cover a 3-cm segment of the median nerve and the vibrator was fastened tightly to ensure that the vibratory energy could be transmitted into the deep nerve with limited amount of vibratory energy loss. In fact, all subjects could feel tingling or vibratory sensation during vibration on the forearm.

#### Nerve Vibration

Direct nerve vibration of the sural nerve has been previous studied by Ertekin et al. [7]. They concluded that the amplitude decrease in sensory nerve was attributed to an impulse blockage of the large diameter nerve as a consequence of the "busy line" phenomenon. However, we were unable to demonstrate any significant changes in motor nerve excitability when the nerve was vibrated directly. Perhaps vibration over the motor nerve itself was not as efficient as vibration on the sensory receptors (or nerve terminals) to induce any significant changes. It is likely that the receptors can perceive a certain type of energy form that is not perceivable by the main nerve fibers. Another possibility is that the nerve is beneath the skin and muscle and a portion of vibratory energy may be absorbed by these tissues.

#### Tendon Vibration

Vibration had been used as one of therapeutic modalities for the relief of pain and treatment of motor dysfunction [2,3,5]. The mechanisms for such therapeutic applications are related to spinal cord reflexes. Previous studies have confirmed that the increase of motoneuron excitability by tendon vibration (TVR) is a spinal cord reflex elicited by stimulation on muscle spindles [3-5, 9-12]. Constant muscle spindle activation can induce the tonic contraction of the correspondent muscle [5,10,15]. It had been shown that the TVR led to a depolarization of the alpha motoneuron and a consequent increase in motoneuron excitability [11]. A study on the decerebrate cats suggested that supra-spinal centers were involved in facilitating the efferent impulses needed for tonic contraction [16].

It is likely that the increase in nerve excitability by tendon vibration in our study was related to the increase of motoneuron excitability following the same pathway of TVR. Thus, the change of excitability observed in this study of tendon vibration was not a direct response of nerve excitability, but an influence from the changes of motoneuron excitability as a consequence of tendon vibration (i.e. TVR)

As shown in Figure 2, there was a slowly rising trend of EI during tendon vibration as observed in the TVR. This further supports the possibility that the change in nerve excitability due to tendon vibration is related to the TVR mechanism. The explanation of this change was based on a progressive increase in muscle spindle sensitivity due to the gradually increase of stretch from the tonic contraction [5,9]. The increased sensitivity therefore led to an increase in afferent impulses even though the vibration intensity remained unchanged. There was also a slowly decreasing trend after the vibration being turned off. The decreasing trend was due to a progressive decrease in muscle spindle sensitivity after cessation of vibration. It was probably due to a gradual decrease in muscle spindle sensitivity after removal of vibratory stimulation

## Mechanism of Pain Relief from Vibration

As already mentioned, vibration has been used for pain relief with success [1,2]. Lundeberg and his associates had suggested that peripheral sensory receptors, either deep or superficial, preferentially elicited impulses through the large afferent fibers led to a central mechanism for pain relief [1,2]. The gate control theory still remains as one of the most likely mechanisms for the centrally mediated pain relief [17]. This study provides more evidence for excluding a peripheral mechanism for pain relief via a direct action on peripheral nerve axons. Specifically, the results of nerve vibration in this study suggested that a vibration-induced excitability change in either small pain fibers or larger afferent fibers, proximal to the source of pain, probably does not exist. Therefore, a possible enhancement of larger afferent fiber transmission or a blunting of small pain fiber transmission by proximal nerve vibration was not a likely

mechanism for pain relief. On the other hand, distal tendon vibration could increase the activity of large afferent fibers via the same pathway of TVR, therefore, subsequently inhibited small fiber activities at the spinal cord level.

# **CONCLUSION**

In conclusion, this study has demonstrated that vibratory stimulation may cause an increase in motor nerve excitability when the vibration is applied to the tendon, but not to the nerve itself. A well-documented pathway, TVR, is probably the route for the increase in nerve excitability by its facilitation to the alpha motoneuron pool.

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# 高頻振動引發神經興奮之效應

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本研究對 18 位健康成年人施子高頻振動,以了解振動對正中神經興奮性的效應。受試者被隨機選取一側手臂為實驗側,另一側則為控制側。振動器分别置於正中神經上之皮膚或淺指屈曲肌腱上之皮膚做刺激,實驗側接受振動刺激,控制側則無接受刺激。正中神經興奮性以興奮指數(Excitability Index)表示;所謂興奮指數係指振動前、中、後的次極大誘發複合動作電位(submaximal evoked compound action potential)之強度對基準電位之比值。比較實驗側與控制側的結果發現,直接對正中神經施予刺激並不能引發興奮指數的改變具有統計上的差異。然而,對淺指屈曲肌腱施予振動,則可誘發正中神經興奮性,其升高具有統計上的意義。本研究結果顯示,神經興奮性的升高來自於對肌腱振動,其機轉可能來自於張力性振動反射(tonic vibration reflex)。(中華復健醫誌 1999; 27(1): 7-14)

關鍵詞:興奮性(excitability),神經(nerves),神經傳導(nerve conduction),振動(vibration)

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