NEUROMUSCULAR INHIBITION THROUGH LINEAR VIBRATION

(Pilot Study)

Abstract

Although vibration has long been considered one of the *tools of the trade* in the physical therapy armory it does not generally appear in the clinical setting as a mode of treatment for pathogenic neuromuscular disorders, or for the relief of chronic or acute myogenic pain in the otherwise normal patient. However, research has shown that vibration does affect muscle activity. The purpose of the Pilot Study was to explore and further define this effect; the four Case Studies included indicate some of the ways in which it might be beneficially exploited as a treatment mode in the clinical setting.

Review

The earliest investigators of the effects of vibration on muscle tissue demonstrated that vibration of a muscle's tendon could produce an involuntary contraction in the muscle, which they labeled as a stretch reflex.[3] They showed that the force of the contraction would increase as the amplitude of vibration was increased. They also showed that the contraction would occur whether skin covered the muscle or not, thus demonstrating that the responding elements lay within the muscle and did not result from tactile sensory nerve stimulation.

Kuffler et al. established in 1951 that the organ in the muscle that was responsive to vibration was the muscle spindle.[4] They showed that a single muscle spindle afferent nerve fiber would discharge at a frequency rate corresponding to the frequency of the vibration up to several hundred hertz (Hz) or cycles per second (c/s). Bianconi and Van der Meulen postulated, from their investigation, that while the nuclear bag produced a phasic response to vibration, the myotube and central portion of the nuclear chain were responsible for the slow developing contraction resulting from vibration.[5]

Eklund and Hagbarth labeled the slow developing contraction resulting from vibration (a function of the tonic stretch reflex) as the *tonic vibration response* (TVR).[6] The TVR was found to be present in all skeletal muscles except the facial muscles and the tongue. They determined that the TVR could also be produced by vibrating the muscle belly, but only at higher amplitudes. They also found that vibration of a muscle's tendon at frequencies of from 50 to 100 c/s gradually increased the muscle's electromyographic (EMG or EMM) activity and strength of contraction while decreasing the activity of its antagonist (muscle

response to vibration was generally not observed for longer than 30 seconds after vibration of the muscle ceased). Ekland and Hagbarth also demonstrated that even though the TVR is involuntary the subject could stop the response at will. This finding, coupled with the demonstration by deGail et al. that the force of the contraction produced by vibration decreases markedly when the muscle belly is infiltrated with procaine and that muscles innervated distally to a complete transection of the spinal cord will not respond to vibration with a contraction, led to the conclusion that the contraction resulting from vibration depends on supraspinal centers. Investigators showed that vibration depresses the phasic stretch reflex while augmenting the TVR.[7,8,9] In other words, as the muscle spindle changes its tonic biasing in response to vibration, there is a commensurate negation of the phasic stretch reflex as the mechanism resets to accommodate a new spindle length.

Two main conclusions may be drawn from the above studies. (A) Vibration of muscle tissue (tendon or muscle belly) affects the muscle spindle to input proprioceptive activity to the central nervous system causing supraspinal structures to produce a TVR (slow-developing muscle contraction) in the muscle vibrated through the tonic stretch reflex mechanism. (B) The muscle's antagonist experiences a reciprocal decrease in muscle spindle tension and a concurrent decrease in extrafusal muscle tension because of its tonic stretch reflex mechanism. Vibration produces these effects at frequencies of from 50 to several hundred Hz. Higher amplitudes are needed to produce the same amount of change from vibration of the muscle belly.

When the agonist is compelled to produce a TVR by vibration, its antagonist is reciprocally compelled to decrease its activity, while the phasic stretch reflexes of both muscles are repressed or suspended as the phasic stretch mechanisms reset themselves to accommodate the new spindle lengths. However, when afferent and efferent tracts in the spinal cord are interrupted, the muscles innervated below the site of lesion will fail to produce a TVR in response to vibration but the phasic stretch reflex will still be suppressed during the procedure.

The Pilot Study

To help explore the use of vibration as a treatment modality in a clinical setting, a series of tests was performed to obtain additional data regarding the effects of vibration. Here are the issues that were addressed in the Pilot Study:

1. The duration of the after effects of vibration on myoelectric activity from the muscle vibrated, when applied to normal muscle tissue (i.e., the patient being without any recognized pathology of muscle, or central or peripheral nervous system tissues).

2. The comparative after effects of vibration, over time, on the myoelectric activity from antagonistic muscles, when vibration is applied to (a) the hypertonic (tight) muscle or (b) to its antagonist.

3. The comparative myoelectric after effects of vibration applied at 30 Hz and at 60 Hz, to antagonistic musculature.

4. The post-vibration myoelectric response of stimulated muscle tissue compared with that of post-electrical stimulation myoelectric response of the same muscular tissue.

Method

The subject of these pilot studies was a 23-year old woman with a history of chronically *tight* shoulder musculature (right upper trapezius greater than left). Two Model 502A electromyometers (EMMs) produced by the Biofeedback Research Institute* were used to provide simultaneous myoelectric activity data from the muscles monitored. The data was recorded as microvolts indicated by meters on each machine. The machines were matched calibrated. The EMMs were attached to the muscles monitored by electrode cables, which were clipped to silver EKG electrodes. The electrodes were affixed to the skin by adhesive electrode collars. The vibrator used was a McShirley# linear vibrator, with variable frequency and intensity. For the purposes of these studies, the linear vibrator was set to vibrate at 30 Hz for Tests 1 and 2, and at 60 Hz for Tests 3 and 4, with a stroke force of 3.5 pounds per square inch (psi). During the vibration procedure, this instrument was hand-held in position, with moderate pressure, by one of the investigators. For Tests 5 and 6 an electro galvanic stimulator (EGS), produced by Electro-Med Health Industries, Inc.! was used to electrically stimulate the various muscles at approximately 25 Hz with approximately 110-volts and less than 0.2-amperes. The stimulator was set to surge on for five seconds, with five-second intervals between surges.

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Procedure

The subject was asked to sit in an armless straight-backed chair in a relaxed upright position with her head facing forward. A pair of active (pickup) electrodes was attached to corresponding sites on her skin over each of the upper trapezius muscles, along a hypothetical line from the spinous process of the C7 vertebra to the distal head of the clavicle. They were placed approximately 2.5 and 3 inches from the C7 vertebra, respectively, along that line. The ground electrode for each electrode set was placed on a site equidistant from the two pickup electrodes. The electrodes were connected by electrode cables to the two EMMs placed out of the visual field of the subject, with the audio feedback mode turned off.

In Test 1, initial simultaneous recordings were made of the myoelectric activities from each of the upper trapezius muscles. Successive recordings were taken every 2 minutes for six minutes. The suboccipital fibers of the right upper trapezius muscles were then vibrated with a linear vibration at 30 Hz applied for 60 seconds just lateral to the C1 and C2 vertebrae. Simultaneous recordings were then taken from each upper trapezius every 2 minutes for 14 minutes. In Test 2, the procedure performed in Test 1 was applied to the suboccipital fibers of the left upper trapezius muscle. In Tests 3 and 4, the procedure performed in Tests 1 and 2 was repeated with linear vibration applied at 60 Hz.

In Test 5, initial simultaneous recordings were made of the myoelectric activities of each of the upper trapezius muscles, as in Tests 1 through 4. Successive recordings were taken every 2 minutes for six minutes. The patient was then placed in a reclining chair and reclined back to approximately 45°. An electrical stimulation pad (1.75 $inch^2$) was placed over the suboccipital cervical fibers of the right upper trapezius muscles and another pad (3 $inch^2$) was placed over the fibers of the same upper trapezius muscle in the supraspinatus area (to act as a ground for the smaller pad). Initially, the smaller pad was the cathode (negative) electrode and the large was the anode (positive) electrode. The electrical stimulation was first applied for 10 minutes with a negative polarity and then for five minutes with a positive polarity (the poles reversed). The subject was then returned to her previous upright position in the straight-backed chair.

Simultaneous recordings were then taken from each upper trapezius muscle every 2 minutes for 14 minutes. In Test 6, the procedure performed in Test 5 was applied to the left upper trapezius muscle (the antagonist of the tense muscle). Collection of data was performed by one of the investigators, in a position out of the visual field of the subject, with a clear and un-obscured view of the EMM meters. No coaching or instruction was given to the subject beyond the instruction to remain sitting and to relax. Each of the tests was carried out on a different day.

Results

In Tests 1 and 3, vibration of the tense muscle, in each case, ultimately resulted in little change in the *average* of the *previbration resting levels* (PRL), as reflected by the final myoelectric recordings from each of the antagonists at the end of the 14-minute period, following cessation of vibration.

In Tests 2 and 4, however, vibration of the antagonist of the tense muscle resulted in myoelectric activity levels from the tense muscle falling 71% and 59%, respectively, below the *average* of the PRL readings in 14 minutes. Tests 1 through 4 show substantial increases in myoelectric activity from all antagonists above *final* PRL readings just after vibration had ceased; this effect appears to be increased when vibration is applied at the higher frequency of 60 Hz.

In Test 5, EGS applied to the tense muscle caused an initial decrease in myoelectric activity in both muscles to below the average PRL readings, followed by a jump in myoelectric activity from both muscles 10 minutes after vibration had ceased (up to 82% above the average PRL in the right and 627% in the left). Final activity levels very nearly matched the average PRL readings. In Test 6, EGS applied to the antagonist of the tense muscle caused an overall rise in myoelectric activity from both muscles to finish with activity levels well above the average PRL readings (223% above the average PRL from the right and 76% from the left).

Study Discussion

The results of Tests 1, 2, 3 and 4 take us past earlier observations of the short-lived TVR response to vibration and lead us to the dramatic inhibitory effects of linear vibration. These four tests indicate that vibration inhibits myoelectric activity in the antagonist of the muscle vibrated, whether the muscle vibrated is the hyperactive (tense) muscle of the pair or not. Tests 1 and 3 may be interpreted as an indication that vibration of a hyperactive muscle increases its activity and therefore might, by itself, be contraindicated as a treatment modality.

Linear vibration does not appear to produce appreciably greater benefits at 60 Hz than vibration at 30 Hz when the results of Test 1 and 2 are compared with Tests 3 and 4; contrarily, 30 Hz would appear to have a superior inhibitory effect when applied to the antagonist of a tense muscle.

Tests 5 and 6 indicate that electrical stimulation (ES) has a much different effect on muscle activity than linear vibration. The long-term effects would seem to be facilitory of myoelectric activity after cessation of stimulation (for at least 10 minutes) in both muscles when one of the muscles is stimulated. As in Tests 1-4, the greatest myoelectric facilitory response seems to come from the antagonist of the muscle stimulated, especially when the antagonist is the hyperactive muscle, as in Test 6.

A direct comparison of the myoelectric response to linear vibration with the myoelectric response to ES may be possible by comparing the representative graphs. The results of this comparison are surprising. The post-cessation effect of linear vibration on all the muscles was generally inhibitory, while the ultimate

effect of electrical stimulation was facilitory (for at least the first 10 minutes after cessation of stimulation). Apparently, vibration and ES seem to have a very different effect on myoelectric activities after cessation of either type of stimulation. This finding led us to consider combining both modalities in a treatment plan designed to control muscle hyperactivity in cases of muscle tension, spasm, or referred pain trigger point syndromes.

Case Studies

The patient in **Case Study #1** was a 30-year old female who had been diagnosed as suffering from athetoid spastic cerebral palsy. The patient's neuromuscular system was dominated by nearly all the developmental reflexes including the flexor withdrawal, extensor thrust, crossed extension (1 and 2), labyrinthine supine and prone, the homolateral and contralateral associative reactions, and an extremely strong asymmetric tonic neck reflex. Neuromuscular involvement in these reflexes was so great that they prevented her from accomplishing almost any voluntary skeletal action, including walking, standing, crawling, creeping, sitting without support, feeding herself, or talking clearly.

A home exercise program using electromyometric (EMM) feedback, with her mother acting as technician, was initially instituted to help decrease hip adduction, throat, and jaw muscle over-activity, and to reduce dominance by the asymmetric tonic neck reflex in the upper extremities. Over time, her mother reported that hip adduction had been reduced to a degree, she had been able to get her right hand to her mouth while eating, and she spoke with improved clarity.

Several years later, the patient returned to have linear vibration assessed as a modality to augment her rehabilitation program. Her mother reported that she had continued to improve, especially in her ability to feed herself, talk, and had begun standing with support. However, the athetoid spastic motion was still a prevailing problem and her mother was interested in getting further help in controlling the spastic wrist and finger flexor muscles, hip adductor muscles, and bladder sphincters (which had required a regular program of catheterization).

To test the effect of vibration, the muscles of her right forearm were selected as the test sites. The muscles of the finger flexors and the extensor antagonistic muscles were simultaneously monitored by electromyometry. Initial readings indicated involuntary hyperactive finger and wrist flexor muscle groups, with a marked tonic and phasic stretch reflex response to voluntary activity of the finger and wrist extensor muscles. Vibration was applied to the homolateral extensor tendons with a McShirley linear vibrator, set to deliver a linear force of 3.5 psi at 30 Hz for one minute. Resting electromyometric flexor activity decreased to less than 50% of the initial electromyometric levels within a few minutes after cessation of vibration. Attempts to voluntarily increase extensor myoelectric activity above resting myoelectric activity were more successful, with a much more depressed flexor myoelectric activity response than was present before vibration. This experiment was repeated on muscles of the left hip.

The left hip adductor and internal rotator muscles and the antagonistic left hip adductor and external rotator muscles were simultaneously monitored by electromyometry. Linear vibration was applied for one-minute at 3.5 psi at 30 Hz over the medial origins of the gluteus medius, piriformis, obtuerator, and gemellus muscles.

This site was coincidentally over the area of the left sacral peripheral nerve roots. The results were much like those demonstrated by the muscular responses in the forearm. There was a marked decrease in the resting activity of the hip adductors as well as an improvement in the ability of the patient to increase voluntary myoelectric activity from the hip adductor and external reactor muscles.

The patient's mother commented that even after the short-term vibration there appeared to be a spontaneous improvement in the patient's functional ability to extend her right wrist and to spread her legs, even without the EMM being used as an aid. Consequently, the patient's mother acquired a vibrator of the type used clinically and agreed to use it as instructed in their home program in conjunction with the EMM training. The patient's mother reported the following week that her daughter had continued to increase functional control of her wrist and hip. Additionally, her daughter had experienced an unexpected increase in her ability to voluntarily control urinary elimination, noting that the need for catheterization had been absent for two or three days after a single application of vibration in the hip external rotator area. Follow up reports indicated that linear vibration of the left sacral roots needed to be repeated only once every three days for the patient to maintain almost complete control of voluntary urinary elimination (excessive emotional stress being the only over-riding factor).

The patient in **Case Study #2** was a victim of head trauma sustained in an automobile accident three years before our initial evaluation. His brain had been bruised when the left superior aspect of the skull had been crushed downward. This trauma produce *hemiplegia* of the right side, marked by neuromuscular spastic flexion synergies involving the right upper extremity and spastic extension synergies in the right lower extremity. Neuromuscular reeducation was instituted using two EMMs, providing the patient with an opportunity to increase voluntary myoelectric activity in one muscle while decreasing myoelectric activity in a homolateral antagonist; thus, allowing the patient to begin reprogramming fine motor control of his involved extremities, and to increase the possibility of his redeveloping functional use of them. The patient's therapy resulted in an improvement in ambulation as well as improved shoulder, elbow, and hand neuromuscular control, but the patient was still having difficulty with voluntary finger extension and inhibition of spastic finger flexors. It was decided to appraise the use of linear vibration as a tool for resetting muscle lengths and decreasing the phasic stretch reflex in the hyperactive muscles before EMM training.

The patient's hand was taped to a splint board with full extension of the wrist and fingers, with the thumb extended and abducted to a 90° angle with the index finger. The right finger extensor tendons were then

vibrated for a full minute with a hand-held McShirley linear vibrator set to deliver a linear vibrator force of 3.5 psi at 30 Hz. This caused the finger extensor muscles to immediately shorten and the finger flexor muscles to lengthen reciprocally. In other words, the wrist and finger flexor muscles relaxed enough that the wrist no longer pushed up against the tape restraining it, and the finger tips no longer dug into the supporting splint. After cessation of vibration, EMM, activity from the finger flexors dropped within 2 minutes to 50% of previous resting levels. The patient was then instructed to attempt to voluntarily increase finger extensor myoelectric activity, as reflected by the EMMs. After a one-hour session, the patient **voluntarily** extended his index finger several degrees for the very first time.

The patient in **Case Study #3** was first seen for evaluation of an acute back and leg pain syndrome that he feared was sciatica. The evaluation demonstrated that he was not suffering from peripheral (sciatic) nerve impingement, but was, instead, suffering from trigger point formation referred pain patterns originating from the left gluteus medius, minimus, and gastrocnemius muscles. The trigger point formations were treated with an electro galvanic stimulator (EGS), set at a frequency of 28 Hz and at the amplitude of 120 volts (0.20 amperes). The negative EGS pads were placed over the trigger point formations found in the left gluteus medius, minimus, and gastrocnemius muscles, and the positive (ground) pad placed in the posterior lower thoracic area. Polarity was reversed from negative to positive at the end of 10 minutes, and the stimulation continued for five minutes more.

The patient reported immediate relief from the pain in the left hip and thigh but still complained of pain in the left gastrocnemius area. Linear vibration was applied for one minute over the muscle bellies of the left long toe extensor, peroneus longus and peroneus brevis muscles with a hand held McShirley linear vibrator set to deliver 3.5 psi at 30 Hz. The patient reported an immediate cessation of pain in left gastrocnemius. The patient was then instructed in isometric exercises for the hip abductor, external and internal rotator muscles, as well as for the ankle dorsi and plantar flexors of the calf.

The following day, the patient returned complaining of pain radiating down the anterior surface of the left calf, but without any of the previous pain symptomology. Evaluation demonstrated a trigger point formation to be present in the long toe extensor muscle group at the site vibrated the previous day. Consequently, the trigger point formation was treated with EGS, as had been the trigger points discovered the previous day. After the treatment, some pain remained in the anterior calf area, so vibration was applied over the tendon *insertion* of the left gastrocnemius muscle. The patient stated that the calf pain was immediately relieved. The patient did not need to return for any further treatment.

The patient in **Case Study #4** was first seen for injuries of the soft tissues of the neck, shoulders, and low back sustained in an auto-versus-auto accident three weeks before initial evaluation. It was established that the patient had inflammation of the left cervical neck and bilateral lumbosacral areas, as well as

trigger points in the left upper trapezius, lower trapezius, upper longissimus, bilateral scaleni, splenius capitis, gluteus minimus, and gluteus medius muscles.

The treatment plan included EGS, phonophoresis of anti-inflammatories, transcutaneous nerve stimulation and isometric exercise (the latter to be performed at home).

The patient returned the following day complaining of a heightening of the right low back and hip pain, with pain radiating down into the right upper thigh. It was established that a trigger point formation in the right gluteus minimus muscle (hip internal rotator) was expressing itself and producing the pain pattern described. It was again treated with the EGS. The patient reported an initial sharp decrease in pain, but after a few minutes, the pain returned. The origins of the right hip external rotators (piriformis, gemellus, and obturator muscles) were subsequently vibrated for one minute with a hand-held McShirley linear vibrator set to deliver a linear force of 3.5 psi at 30 Hz. The patient initially reported no change in the pain pattern or intensity level, but after a rest period of approximately six minutes, she reported that the pain level was markedly decreased. In approximately nine additional minutes, she reported that the pain pattern had completely disappeared.

Observations

1. For the best inhibitory response, linear vibration should be applied to an antagonistic muscle's origin, insertion, or tendon.

2. Vibration of the muscle belly of a trigger point prone muscle will promote a trigger point formation with its resultant referred pain pattern.

3. Vibration of a homolateral antagonist is as effective as vibration of a contralateral antagonist as an inhibitor of myoelectric activity from a hyperactive muscle.

4. The inhibitory influences of vibration on the antagonist of the muscle vibrated may be successfully utilized in the treatment of referred pain originating from a trigger point formation.

5. Vibration can be effectively used in a program of rehabilitation for victims of cerebral vascular accident (CVA) or other types of central nervous system damage, when it is used to

mechanically reset antagonistic muscle lengths across a joint, and to decrease the phasic stretch reflex in hyperactive (spastic) musculature before electromyometric neuromuscular reeducation. The object of neuromuscular reeducation with electromyometry is to reprogram fine motor control as it relates to the tonic stretch reflex mechanism of the muscle spindle. Consequently, if the phasic stretch reflex can be suppressed or eliminated, so that the patient is only required to deal with tonic stretch reflex directly through electromyometry, the speed of learning may increase.

6. Post CVA and other types of central nervous system damaged patients respond more quickly to the post-cessation inhibitory effects of vibration than do patients without pathology.

Conclusion

Vibration appears to be a valuable tool for the treatment of hyperactive neuromuscular conditions, whether the source of hyperactivity is due to central nervous system damage or some other source of normal muscle hypertension, spasm, or trigger point formations.

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