Scand J Rehab Med 1 : 26–34, 1969

THE MUSCLE VIBRATOR—A USEFUL TOOL IN NEUROLOGICAL THERAPEUTIC WORK

K.-E. Hagbarth and G. Eklund

From the Department of Clinical Neurophysiology, University Hospital, Uppsala, Sweden

The aim of the present review is firstly to present to clinicians and physiotherapists some modern neurophysiological concepts concerning the participation of the stretch reflex in motor control functions, and secondly to describe how artificially induced tonic stretch reflexes can be used in the clinic for both therapeutic and diagnostic purposes.

Therapists actively engaged in motor habilitation or reeducation of patients with central motor disorders are confronted with many different schools of thought and techniques of treatment. Our intention is not to add just another item to the long list of therapeutic techniques but rather to emphasize the importance of adjusting the techniques to the constantly increasing knowledge of basic neural mechanisms involved in motor control. It has long been known that proprioceptive stimuli and various postural reflexes can be used for therapeutic purposes to change the strength and distribution of spasticity and to reinforce appropriate patterns of willed movements. There are diverging opinions, however, as regards the therapeutic value of using the "simple" spinal stretch reflex for such purposes. Before returning to this issue some facts about the stretch reflex must be made clear. The vast amount of literature on this reflex has been extensively covered in several books and reviews (13, 18, 33, 42, 43). In particular, a recent monograph by Granit (19) provides essential background data for the very short and simplified outline which follows.

THE STRETCH REFLEX IN NORMAL MOTOR CONTROL

It is important to make a clear distinction between the *phasic* (*or dynamic*) *stretch reflex*, in the clinic commonly recognized as the tendon jerk, and the *tonic* (*or static*) *stretch reflex*, which—even though it plays a major role in motor control—is not easy to demonstrate in healthy subjects since it is "turned-off" when the subject relaxes and operates only when the muscles are active.

The receptors responsible for both the phasic and the tonic stretch reflex are the primary endings of the muscle spindles. These endorgans are length receptors which—suspended by contractile intrafusal muscle fibres—lie in parallel with the main extrafusal fibres of the muscle. The anatomical arrangement is such that the receptors can be activated both "externally" by muscle stretch and "internally" by intrafusal contraction. The intrafusal muscle fibres have a nerve supply of their own, the fusimotor (or gamma) nerve fibres which are now believed to be a functionally heterogenous fibre group providing independent control of the static and dynamic properties of the receptors.

The spindle endings probably require little, if any, fusimotor support to respond to such fast stretches as those resulting from tendon taps, but they do require continuous central support to respond effectively to sustained muscle stretch. During normal motor relaxation there is not sufficient fusimotor outflow to make the tonic stretch reflex operate; the muscles are therefore slack and do not automatically resist passive joint movements. However, when the subject actively contracts his muscles to resist or overcome some external load it is not only his "ordinary" alpha motoneurones which start firing to contract the extrafusal fibres; he automatically co-activates also his fusimotor fibres to these muscles so that the spindles and

thereby the tonic stretch reflex starts operating. Direct evidence for such central co-activation of alpha and gamma motoneurones (alpha-gamma linkage) has recently been obtained in microelectrode recordings from human muscle nerves (26, 27). When the muscles are active, their tonic stretch reflexes serve to adjust the strength of the contractions to the external loads. As the load of a contracting muscle increases the afferent spindle signals run through mono- and polysynaptic spinal paths to the alpha ventral horn cells (not to gamma motoneurones), causing a motor pattern which involves not only autogenetic excitation but also reciprocal inhibition of antagonist motoneurones. To understand the functional significance of these events one has to realize that in their task of maintaining the upright posture or of executing willed movements, the individual muscles acting on the skeleton are met by complex changes of external forces and loads, which must be properly counteracted or balanced by fast automatic changes of active muscle tension.

THE STRETCH REFLEX IN SPASTICITY

It is well known that central nervous lesions tend to cause not only primary deficiency symptoms, but also various types of neural release phenomena, often manifested in abnormal exaltation of certain reflexes or postural automatisms. In fact, Denny Brown (6) ascribes all types of central muscular hypertonias to release phenomena resulting in "disequilibrium" between competing tonic reflexes or motor control mechanisms operating at various levels of the neuraxis. All specific tonic postural reflexes (tonic neck-, vestibular reflexes etc.) involve excitation of certain muscle groups and inhibition of others. Consequently, certain muscle groups tend to become hypertonic whereas others tend to become inhibited (or weak) as a result of tonic reflex exaltation. However, the lesion may also upset the normal close coordination between the central paths involved in the control of the alpha and gamma motoneurones, so that an exaggerated motor pattern chiefly engages either one of these two systems. The neurophysiologist differentiates between alpha- and gamma types of muscular hypertonus whereas the clinician talks about spasticity, rigidity and dystonia depending on the type of resistance he feels when he stretches the hypertonic muscles. There is now much evidence to support the notion that fusimotor hyperactivity is the main cause of the increased stretch reflexes in spastic muscles and there is also a growing conviction that spastic states in man are a heterogenous group where more or less hypertonus of the "direct" alpha type is combined with different degrees of hyperactive phasic and tonic stretch reflexes. Since the tonic stretch reflex involves a component of reciprocal inhibition, spastic muscles will tend to impose additional inhibition upon the weak antagonists.

Denny Brown emphazises that all central hypertonic states are more or less dynamic and unstable. Firstly, the strength and distribution of hypertonus (and weakness) may change according to which specific tonic reflex pattern happens to dominate at a given moment. It is well known, for instance, how a change from the supine to the prone position in cerebral palsy patients tends to reduce flexor and enhance extensor spasticity. Secondly, "suprasegmental mechanisms in powerful conflict" may result in co-contraction of antagonistic muscle groups or the conflict may be "resolved by alternating rhythmical actions, ranging from rapid tremor to more ample spontaneous clawing or progression movement" (6). It is also well-known how unspecific arousal stimuli, emotional strains or strong motor efforts of any kind may cause a general enhancement of muscular hypertonus. In this connection it should be noted that *unspecific*, non-reciprocal, motor arousal reactions may well give rise to *specific* motor patterns, determined by the pre-existing level of excitability in the various motoneurone pools. Such phenomena are possibly involved in those severe cases of spasticity where all voluntary attempts to move a paretic limb merely results in an exaggeration of the initial abnormal posture (7).

The stretch reflex release may cause secondary functional changes in the spinal reflex arc, resulting in a further exaltation of the reflex. Post-tetanic potentiation (PTP) is a long-lasting presynaptic facilitation of transmission across a synapse after a period of increased activity. As pointed out by Granit (17), this phenomenon may contribute to the production of hyperactive stretch reflexes in spasticity. It has also been claimed that motoneurones, partially deprived of presynaptic terminals may become hyper-responsive to signals in remaining afferent paths (41).

In severe states of spasticity there is usually also an abnormal "irradiation" of the stretch reflexes. In mild cases, stretch of any one muscle group has little obvious effect on other muscles of the limb but in more severe cases the stretch responses spread to involve functionally allied muscle groups at other joints. This fact explains the common clinical finding that a tenotomy or an intramuscular phenol or alcohol injection may relieve spasticity not only locally but also in distant muscle groups of the limb.

When a spastic muscle is forcefully extended, the resistance usually increases up to a certain point and then it melts away. This relaxing effect of overloading, known as the clasp-knife reaction, is probably related to the further stimulation of Golgi tendon organs and secondary spindle endings, which inhibit the alpha neurones of the muscle in which they lie (in the cat the autogenetic inhibition from the Golgi organs concerns extensor muscles only). The reflex status of a spastic muscle is thus determined by the relative strength of the competing autogenetic facilitatory and inhibitory effects.

PRINCIPLES OF TREATMENT

Treatment according to the "Bobath School" (2, 3, 4), is based on the principle that "the teaching of normal movements and the correction of postures is impossible as long as muscle tone is abnormal and released tonic reflexes are present" (2). Consequently, a primary goal of the treatment is to inhibit the released tonic reflex activity, which strives to "lock" the patient in an abnormal "postural set". This can often be done by passively bringing the patient into "reflex inhibiting postures", which are total or, more often, partial reversals of the patient's initial abnormal posture. The gradual reduction of spasticity as the patient adjusts to the new posture, probably depends on stretch-induced tonic, autogenetic inhibition from Golgi and secondary spindle endings—reactions which at a certain degree of muscle stretch tend to get command in competition with the tonic stretch reflex. According to the *law of reciprocal innervation* such a change of reflex dominance will also result in a facilitation (or disinhibition) of the antagonists to the spastic muscle groups—an effect which helps to "unlock" the abnormal postural set and thereby improves the patient's ability to move. The Golgi tendon organs in spastic muscles can also be stimulated by electrically induced muscle contractions and, according to Hufschmidt (32), repetitive contractions of this kind may be effective in reducing spasticity and activating weak antagonists.

The techniques of Kabat and Knott (34, 35, 36) aim more directly at proprioceptive facilitation of those weak muscle groups which act in movement patterns opposing the spastic posture. In particular, they recommend training of the weak muscles in active movement patterns carried out against resistance given by the physiotherapist. It is important to realize that even though this manoeuvre does not involve extension of the weak muscles, it will tend to activate the tonic stretch reflexes in these muscle groups: the voluntary effort to move will cause fusimotor activation of the muscle spindles and, providing these organs are "loaded" (by an external resistance), the primary spindle endings and the tonic stretch reflex start operating (26, 27). As is to be expected, maximal autogenetic facilitatory (or disinhibitory) effects will be obtained if the weak muscles are all stretched into their lengthened position at the commencement of the movement. The manoeuvre will help the patient to break away from the abnormal postural set, especially since the spindle inflow from the muscles concerned also imposes reciprocal inhibition on the motoneurones supplying the antagonistic spastic muscle groups. One of the effects resulting from tetanic stimulation of a human muscle nerve is an activation of the stretch reflex afferents, and Gračanin and Dimitrijević (16) recommend the use of such stimulation to activate, for instance the weak peroneal muscles in spastic patients.

As emphazised by Levitt, in her description and discussion of various physiotherapeutic approaches (39), "no one thechnique is the answer for all cases". Many techniques should be tried in attempts to compensate for the neural release phenomena, and the physiotherapist should constantly search for and try to expose the patient to such stimulus situations which are apt to cause a more normal balance between competing subcortical motor control mechanisms. Daily excercises of this sort can probably induce long-term benificial modifications of the reflex status—an underlying causal factor possibly being the PTP-phenomenon. Providing the primary deficiency

symptoms are not too severe, the daily exercise also helps the patient to regain cortical control of the reflexes and to utilize and develop compensatory central pathways for purposive motion.

NORMAL RESPONSE TO MUSCLE VIBRATION

A potent artificial way of stimulating the human muscle spindles and eliciting the tonic stretch reflex has recently been described (5, 9, 10, 22, 27, 44, 45).

When a mechanical vibrator (oscillating at 100–200 Hz with an amplitude of 1–2 mm) is applied over a relaxed human skeletal muscle or its tendon it causes a sustained neural discharge in the muscle afferents—a discharge which gains in strength with increasing initial muscle length. The relative weakness of the afferent vibration response at short muscle lengths can be compensated for by a weak voluntary contraction, which by itself tends to induce a sustained afferent discharge from the muscle (26, 27).

The strength of the motor response to vibration (the tonic vibration reflex, TVR) varies in a similar way with the passive muscle length and the degree of initial contraction, and it also varies with the instructions given to the subject during the test (10, 22). If the limb is initially relaxed and the subject instructed to remain passive the vibration usually results in a very slowly rising, involuntary contraction of the muscle vibrated. As an example, the effect of vibration on the elbow flexors in a relaxed, freely hanging arm may be mentioned. Soon after vibration starts an involuntary elbow flexion movement begins; the forearm feels light and seems to float upwards until it reaches a certain position where it then stays for as long as vibration continues and the subject remains passive. When vibration suddenly stops it feels as if the weight of the limb slowly returns and the forearm gradually sinks down to its original position. If the eyes are closed and the subject at the commencement of the test actively maintains a posture of slight elbow flexion, the effect of vibration becomes more apparent. Flexor vibration then induces a more rapid and distinct involuntary flexion movement whereas vibration on the elbow extensors inhibits the activity in the elbow flexors so that the arm extends.

The motor effects of vibration appear distinctly also when the test is performed "isometrically". The subject may for instance be instructed to maintain a certain constant effort in pushing his forearm upwards against resistance. In this situation flexor vibration rapidly raises the active tension to a higher level whereas extensor vibration has the opposite effect. Also, a subject repeatedly beating his first against a fixed object by rapid elbow flexions, unintentionally increases the beat force when vibration is applied on the biceps tendon, and again, extensor vibration has the reverse effect.

Such vibration-induced sustained effects of autogenetic excitation and reciprocal inhibition can normally be effectively counteracted or compensated for by voluntary efforts. Maximal voluntary power is, for instance, not affected by vibration and a subject allowed to watch the position of his arm or the signal from the tensionmeter can easily adjust the strength of his muscle contractions so as to keep a constant position or pressure during the test. When the test is performed isometrically, however, he cannot compensate fast enough to avoid the initial rise of tension as vibration starts and neither can he avoid a short period of overcompensation when vibration suddenly stops (10). Vibration on the knee flexors during walking causes a feeling as if "somebody was pushing on the legs from behind" and the subject tends to walk faster with sligthly flexed knees. By voluntary effort, however, he can compensate for this phenomenon and regain a normal walking pattern.

Animal experiments have shown that high frequency vibration of low amplitude is an efficient way of stimulating the primary endings of the muscle spindles, the Golgi and secondary spindle endings being less sensitive and requiring higher amplitudes of vibration (1, 40). There is indeed strong reason to believe that the tonic vibration reflex which has been demonstrated and studied also in the calf muscles of decerebrate cats (31, 40), originates from the muscle spindles and is homologous to the classical tonic stretch reflex. This deduction is supported also by the finding of Lang and Vallbo (38), that weak, electric, tetanic stimulation of a peripheral muscle nerve in man, just sufficient to excite only the large (I*a*) afferent fibres from the muscle spindles, tends to elicite an autogenetic reflex quite similar to the TVR. We thus arrive at the conclusion that vibratory

muscle stimulation in man imitates the effect of fusimotor activation of the primary spindle endings—a type of activation which may result from abnormal fusimotor release in spasticity, but which normally does not occur unless the subject actively contracts the muscle.

Of course, the vibratory stimulation in man excites not only the primary spindle endings but also a large number of other sense organs, both in the skin and the deeper tissues. Reflexes from such receptors can certainly interfere with the tonic stretch reflex response (TVR). One should, for instance, be aware that even though the vibration is not painful, it occasionally evokes protective reflexes, appearing as quick withdrawal movements away from the vibrator and this withdrawal pattern usually involves a contraction of the muscle underlying the vibrator (21). It is also quite plausible that autogenetic inhibitory reflexes from Golgi and secondary spindle endings to some extent operate during vibration and thus tend to reduce the strength of the vibration-induced stretch reflex. Furthermore, supraspinal reflex centres may be affected by the vibration. Thus, it has recently been shown how—in the standing position when supraspinal equilibrium reactions operate—vibration on the leg muscles induces specific tilting movements tending to bring the subject out of balance; reactions which should probably be explained in terms of vibration-induced "false", proprioceptive messages arriving at the equilibrium centres (8).

Since muscle afferents are considered to play such an important role in motor control functions, it may seem surprising that muscle vibration does not more seriously affect the subject's ability to move. Apparently the vibration "sets" motoneurone excitability at a "false" level but at the same time the healthy subject is aware of the "falseness" and able to compensate for it in his execution of skilled movements. As we shall see, patients with central motor disorders do not always have this ability.

There are interesting similarities between the motor response to muscle vibration and the phenomenon called *after-contraction*, which is the basis of certain parlor tricks (15, 46). For instance, at the end of a period of pressing the arms outwards against resistance there is often an involuntary raising of the arms. If the eyes are closed, the subject may feel that someone is pushing his arms upwards, a feeling quite similar to that induced by vibration on the deltoid muscles. Like the post-vibratory slow decline of the autogenetic excitatory effect, the after-contraction is probably related to central PTP-phenomena (cf. 20, 30).

The tonic vibration reflexes can be demonstrated in all skeletal muscles in healthy adults, flexors as well as extensors, and on the whole, the strength of the TVR in different muscles and individuals varies independently from the strength of the tendon jerks. It should also be mentioned that similar to the way that vestibular- and tonic neck-reflexes can change the strength and distribution of spasticity in paretic limbs, so can these reflexes affect the strength of the motor responses to vibration in healthy subjects remaining voluntary passive during the tests (10).

EFFECTS OF MUSCLE VIBRATION IN SPASTICITY

Therapeutic aspects

Vibration applied to appropriate muscle groups can effectively help a spastic patient to break away from his abnormal postural set. In typical cases of spastic hemiplegia, for instance, vibration on the weak extensor muscles in the arm helps the patient to activate these muscles and to overcome his flexor spasticity, and in a similar way, vibration of the weak flexor muscles in the leg helps him to overcome the extensor hypertonus and flex the limb (11, 23, 24, 25).

The tonus changes induced by vibration can also be demonstrated when such a patient tries to relax and remain passive during the test. Then, as clearly seen in EMG-recordings, vibration of spastic muscles usually enhances the hypertonus not only in the muscle vibrated but also in functionally allied spastic muscle groups at other joints. Vibration of a weak muscle, on the other hand, may produce little or no autogenetic contraction but an apparent reciprocal inhibition of the hypertonus in spastic antagonists. A third way to demonstrate the vibration-induced tonus changes is to measure the muscle resistance to passive movements. By imposing well-controlled, machine-driven movements at various speeds during continuous recording of muscle resistance, it is possible to get an objective measure of the forces normally felt by the neurologist when he moves a spastic limb. When using such a measuring device on hemi-

plegic patients, vibration of spastic muscles is usually found to cause a marked increase of their spastic resistance to stretch, whereas vibration of antagonistic weak muscle groups reduces this spastic resistance by reciprocal inhibition and often uncovers or adds some spastic resistance in the weak muscles themselves.

As might be expected, vibration applied on the most spastic muscles in hemiplegic patients reduces their ability to move. EMG-recordings show that such a vibration not only enhances the spasm in the muscles vibrated, but also inhibits voluntarily maintained activity in the weak antagonists. Depending on the severity of his motor handicap, the patient may be more or less successful in his attempts to overcome these motor effects of vibration. Patients, initially quite able to perform movements against the resisting spastic muscle groups, may—during vibration of these antagonists—become completely unable to move in the desired direction. Indeed, it frequently happens that during vibration their efforts result in movements opposite to those intended (25).

The post-vibratory slow decline of the autogenetic facilitatory effect, described above for normal subjects, can also be demonstrated in hemiplegic patients. Thus, the autogenetic potentiation of the voluntary ability to contract a paretic muscle remains for a while after the end of vibration. The duration of this period of improved motor control is variable and seems to depend upon how the patient "utilizes" his newly-won ability to move. Many patients report that after a few minutes of training with vibration-supported active movements the spasticity feels less severe for 30 minutes or more, providing they keep moving during the post-vibratory period. If they relax, on the other hand, spasticity and weakness return more quickly.

It is probably safe to conclude that vibration of weak muscles in hemiplegic patients is an effective way of restoring a more normal balance between conflicting tonic stretch reflexes from antagonistic muscle groups. In those patients where spasticity and weakness is about equally pronounced in flexor and extensor muscles, vibration is usually as effective in facilitating active movements belonging to the TVR-pattern as it is in preventing movements in the opposite direction. In such cases, the active range of movement can be considerably increased if the effort to move in a certain direction is automatically supported by vibration of the prime movers. Practical tests are being made with "electrophysiological bandages" consisting of a pair of small, portable vibrators, attached over antagonistic muscle groups and controlled by EMG-signals from these muscles (28).

There are also certain groups of spastic patients, however, in which the specific TVR-pattern is lost and replaced by vibration-induced widespread responses of co-contraction in antagonistic muscle groups. In patients with severe chronic spasticity it may even be observed how vibration induces a motor pattern opposite to that of the TVR, i.e. a pattern involving autogenetic inhibition and reciprocal excitation (25). Most findings of this sort are probably due to the fact that the TVR is overridden either by some specific, conflicting reflex or by more unspecific mass-reflexes or motor arousal phenomena. Obviously, the TVR cannot be used for therapeutic purposes in patients who react in this way to vibration.

It is probably in those patients where the motor handicap primarily depends on released tonic stretch reflexes (with accompanying reciprocal inhibition), that vibration of weak muscles is particularly effective in potentiating voluntary power and active range of movement. It should be emphazised that in such cases the vibration-induced potentiation of voluntary power is not a matter of simple addition in the sense that the motor effect of vibration, as seen during voluntary relaxation, now comes "on top" of the voluntary contraction. We have in EMG and strength recordings from spastic patients seen many examples of how the combined effects of vibration and maximal voluntary effort to move results in muscle contractions which are 10 or 20 times as strong as those induced by either of the two manoeuvres alone. Particularly strong contractions in the weak muscles are usually obtained when TVR support is given to voluntary attempts to move against resistance or, in other words, when "Kabat-manoeuvres" are combined with vibration of the prime movers. It may become still easier for the patient to break away from his abnormal postural set if during these manoeuvres he is placed in a "reflex inhibiting posture". There is no valid reason to make any principle discrimination between methods which "serve to strengthen weak muscle groups" and methods which "aim at an inhibition of released tonic reflexes". All the techniques de-

scribed may probably be regarded as different examples of how the tonic stretch reflex and its main segmental opponents (the autogenetic inhibitory reflexes) can be used to relieve certain motoneurone pools from excess excitation and other from excess inhibition.

Besides the fact that it is possible to use EMG-controlled vibrators as "physiological bandages" to give immediate stretch reflex support to voluntary movements, similar long-term therapeutic effects to those that may result from the Kabat and the Bobath methods are also to be excepted from daily sessions of vibration therapy. As emphazised by Bobath, an important factor in motor reeducation is that during the training sessions the patient gets a chance to perceive how it feels when his own efforts to move are successful (2). Such reafference (29) is probably essential in helping the patient to regain cortical control of his movements. There is also evidence that in experimental animals, constantly confined to a certain postural set, structural changes may develop in the muscular proprioceptors themselves (12). Daily training of voluntary movements with optimal reflex support may well help to prevent such changes as well as such disabling central changes which are likely to result from abnormal synaptic disuse or hyperactivity (PTP).

Small muscle vibrators, easy to apply on appropriate weak muscle groups have been constructed and delivered to various rehabilitation centres in order to evaluate their usefulness in motor reeducation of spastic patients.[1] Usually the vibrators are controlled by manual switches handled by the therapist or the patient, but EMG-controlled vibrators and such operated by heel-switches during walking are also tested. After three years of experience with muscle vibration treatment of spastic adults and cerebral palsy children, we are now fully convinced about the long-term therapeutic value of daily sessions during which TVR-support is given to those active motion patterns which should be trained (11). Observations made during these sessions indicate that vibration can also improve motor performance in other ways than by relieving the patient from disabling release phenomena. Thus, it has been noted, how in children with perceptual impairments involving spatial orientation and body image functions, vibration may improve their ability to recognize the affected limbs and strengthen their urge to move. In this connexion reference should be made to the recent experimental findings by Evarts (14), that during purposive movements in monkeys an increase of muscle load potentiates the output not only from the lower motoneurones but also from the pyramidal tract neurones. It is not unlikely that such proprioceptive influence upon upper motoneurone excitability is in part responsible for vibration-induced effects on voluntary motor performance.

Diagnostic aspects of muscle vibration

Some of the effects of muscle vibration, which are unwanted from the therapeutic point of view, are of considerable diagnostic and basic neurophysiological interest (25, 37). Thus, an abnormal, irradiation-induced "paresis" of antagonistic muscle groups may be an early diagnostic sign of an upper motoneurone lesion. The vibration can also serve to reveal a latent clonus in a limb affected by such a lesion. Vibration-induced rhythmic contractions of a clonic type occasionally appear in the calf muscles of normal subjects, but overt, sustained clonus, often outlasting the vibration, preferentially occurs in muscles with exaggerated tendon jerks.

As a rule, no therapeutic effects of muscle vibration can be obtained in patients with motor handicaps mainly due to cerebellar or extrapyramidal disorders. Some of these patients seem to react in a more or less "normal" way to vibration, but it frequently happens that vibration aggravates the motor handicap or induces TVR motor patterns which the patient cannot overcome by voluntary effort. Thus, it has often been observed how choreo-athetotic movements, parkinsonian and intention tremors increase during vibration of the muscles involved, and a more general and severe motor incoordination may also appear. For instance, a patient with very mild signs of an incipient Huntington's Chorea, responded to vibration on the right elbow flexors with strong, widespread choreatic movements and a complete inability to use his hand in writing. In some patients with parkinsonian or cerebellar syndromes it has also been observed how vibration on the palmar side of the wrist cause a distinct pronation movement, impossible to overcome by voluntary effort.

[1] The TVR-vibrators are manifactured by Firma Keydon, Box 322, 751 05 Uppsala 1, Sweden.

Phenomena of this sort may not only help to reveal a central motor dysfunction; when subjected to further analysis they may also help towards a better understanding of the neural mechanisms underlying such dysfunctions.

REFERENCES

1. Bianconi, R. & Van Der Meulen, J. P. 1963. The response to vibration of the end organs of mammalian muscle spindles. *J. Neurophysiol 26,* 177.
2. Bobath, B. 1955. The treatment of motor disorders of pyramidal and extra-pyramidal origin by reflex inhibition and by facilitation of movements. *Physiotherapy, 41,* 146.
3. — 1959. Observations on adult hemiplegia and suggestions for treatment. *Physiotherapy 45,* 279.
4. — 1959. The neuropathology of cerebral palsy and its importance in treatment and diagnosis. *Cerebral Palsy Bulletin 8,* 13.
5. de Gail, P., Lance, J. W. & Neilson, P. D. 1966. Differential effects on tonic and phasic reflex mechanisms produced by vibration of muscles in man. *J Neurol Neurosurg Psychiat 29,* 1.
6. Denny-Brown, D. 1966. *The cerebral control of movement.* University of Liverpool Press.
7. Dimitrijević, M. R. & Nathan, P. W. 1967. Studies of spasticity in man. 1. Some features of spasticity. *Brain 90,* 1.
8. Eklund, G. 1969. Influence of muscle vibration on balance in man. *Acta Soc Med Upsal* (in press).
9. Eklund, G. & Hagbarth, K.-E. 1965. Motor effects of vibratory muscle stimuli in man. *Electroenceph clin Neurophysiol 19,* 619.
10. — 1966. Normal variability of tonic vibration reflexes in man. *Exptl Neurol 16,* 80.
11. Eklund, G. & Steen, M. 1969. Muscle vibration therapy in children with cerebral palsy. *Scand J Rehab Med 1,* 35–37.
12. Eldred, E., Bridgman, C. F., Kano, M., Sasaki, Y. & Yellin, H. 1967. Changes in muscle spindle morphology and discharge with alterations in muscle status. In Yahr, M. D. & Purpura, D. P. (eds.): *Neurophysiological basis of normal and abnormal motor activities,* pp. 35–60. Raven Press, New York.
13. Eldred, E. & Buchwald, J. 1967. Central nervous system: motor mechanisms. *Ann Rev Physiol 29,* 573.
14. Evarts, E. V. 1967. Representation of movements and muscles by pyramidal tract neurones of the precentral motor cortex. In Yahr, M. D. & Purpura, D. P. (eds.): *Neurophysiological basis of normal and abnormal motor activities.* Raven Press, Ney York.
15. Forbes, A., Baird, P. C. & Hopkins, A. McH. 1926. The involuntary contraction following isometric contraction of skeletal muscle in man. *Amer J Physiol 78,* 81.
16. Gračanin, F. & Dimitrijević, M. R. 1966. Application of functional electrical stimulation in rehabilitation of neurological patients. First Symposium of Neurological Rehabilitation, Prague.
17. Granit, R. 1956. Reflex rebound by post-tetanic potentiation. Temporal summation-spasticity. *J Physiol Lond 131,* 32.
18. Granit, R. (ed.) 1966. *Muscular afferents and motor control. Nobel symposium 1.* Almqvist & Wiksell, Stockholm.
19. Granit, R. 1970. Basis of motor control. Academic Press, London. (Read in manuscript.)
20. Hagbarth, K.-E. 1962. Post-tetanic potentiation of myotatic reflexes in man. *J Neurol Neurosurg Psychiat 25,* 1.
21. Hagbarth, K.-E. & Finer, B. 1963. The plasticity of human withdrawal reflexes to noxious skin stimuli in lower limbs. In Moruzzi, G., Fessard, A. & Jasper, H. H. (eds.): *Progress in Brain Research,* vol. 1, Brain mechanisms, pp. 65–78. Elsevier, Amsterdam.
22. Hagbarth, K.-E. & Eklund, G. 1966. Motor effects of vibratory muscle stimuli in man. In Granit, R. (ed.): *Muscular afferents and motor control. Nobel Symposium 1,* pp. 177–186. Almqvist & Wiksell, Stockholm.
23. — 1966. Muscle vibration as a therapeutical aid in spasticity. First Symposium of Neurological Rehabilitation, Prague.
24. —-1966. Tonic vibration reflexes (TVR) in spasticity. *Brain Res 2,* 201.
25. — 1968. The effects of muscle vibration in spasticity, rigidity and cerebellar disorders. *J Neurol Neurosurg Psychiat 31,* 207.
26. Hagbarth, K.-E. & Vallbo, Å. B. 1968. Discharge characteristics of human muscle afferents during muscle stretch and contraction. *Exptl Neurol 22,* 674.
27. — 1969. Single unit recordings from muscle nerves in human subjects. *Acta Physiol Scand* (in press).
28. Hedberg, Å., Oldberg, B. & Tove, P. A. 1967. EMG-controlled muscle vibrators to aid mobility in spastic paresis. Seventh International conference on medical and biological engineering, Stockholm. 197.
29. Holst, E. von. 1954. Relations between the central nervous system and the peripheral organs. *Brit J Anim Behav 2,* 89.
30. Homma, S. & Kano, M. 1962. Electrical properties of the tonic reflex arc in the human proprioceptive reflex. In Barker, D. (ed.): *Symposium on muscle receptors,* pp. 167–174. Hong Kong Univ. Press, Hong Kong.
31. Homma, S., Ishikawa, K. & Watanabe, S. 1967. Optimal frequency of muscle vibration for motoneurone firing. *J Chiba Med Soc 43,* 190.
32. Hufschmidt, H.-J. 1968. Theoretische Überlegungen zu einer neuen Therapie. *Der Nervenarzt 39,* 2.
33. Jansen, J. K. S. 1967. On the functional properties of stretch receptors of mammalian skeletal muscles. In de Reuk, A. V. S. and Knikht, J. (eds.): Ciba Foundation symposium on myotatic, kinesthetic and vestibular mechanisms, pp. 20–34. J. & A. Churchill, London.
34. Kabat, H. 1950. Studies on neuromuscular dysfunction, XII. *Permanente Foundation Med Bull 8,* 9.
35. Kabat, H. & Knott, M. 1954. Proprioceptive facilitation therapy for paralysis. *Physiotherapy 40,* 171.

36. Knott, M. 1952. Specialized neuromuscular techniques in the treatment of cerebral palsy. *The Physical Therapy Review 32,* 73.
37. Lance, J. W., de Gail, P. & Neilson, P. D. 1966. Tonic and phasic spinal cord mechanisms in man. *J Neurol Neurosurg Psychiat 29,* 535.
38. Lang, A. H. & Vallbo, Å. B. 1967. Motoneurone activation by low intensity tetanic stimulation of muscle afferents in man. *Exptl Neurol 18,* 383.
39. Lewitt, S. 1962. *Physiotherapy in cerebral palsy.* Thomas, Springfield, USA.
40. Matthews, P. B. C. 1967. Vibration and the stretch reflex. In de Reuck, A. V. S. and Knight, J. (eds.): Ciba Foundation symposium on myotatic kinesthetic and vestibular mechanisms, pp. 20–34. Churchill, London.
41. McCouch, G. P., Austin, G. M., Lin, C. N. & Lin, C. Y. 1958. Sprouting as a cause of spasticity. *J Neurophysiol 21,* 205.
42. Roberts, T. D. M. 1967. *Neurophysiology of postural mechanisms.* Butterworths, London.
43. Rushworth, G. 1964. Some aspects of the pathophysiology of spasticity and rigidity. *Clin Pharmacol Therap 5,* 828.
44. — 1966. The effect of vibration on tonic and phasic reflexes in man. *J Physiol* (London) *185,* 63 P.
45. Yamanaka, T. 1964. Effect of high frequency vibration on muscle spindle in the human body. *J Chiba Med Soc 40,* 338.
46. Zigler, M. J. 1944. Neurophysiology of post-contraction. *Psychol Rev 51,* 315.

Key words: Reflexotherapy, vibration, spasm, paralysis, tendon, reflex, neuromuscular spindles.

Adress for reprints:
K.-E. Hagbarth, M. D. Ass. professor
Klinisk neurofysiologisk avd.
Akademiska sjukhuset
750 14 *Uppsala,* Sweden