SCAN ME

VIBRATIONS AND SPASTICITY

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ABSTRACT

This experimental study aims to evaluate and validate the effectiveness of the proposed application method in the use of "focused mechanical vibrations" for the treatment of muscle spasticity.
The basic concept of this study is that in spasticity, by specifically stimulating some clearly identifiable trigger points of the body, better results are obtained; in fact, with the localized mechanical-sound vibrations, by positioning the handpiece for the treatment on those specific points, immediate generalized relaxation of the entire spastic muscle is obtained through reflex pathways.
To evaluate this operative reality we treated 5 subjects affected by neurological pathology with spasticity and at T0 time (before treatment) and at T1 time (immediately after treatment), they were assessed with Ashworth Scale, Passive joint evaluation, Evaluation of active motility, Test of Bahkta (for non-functioning hand), surface electromyography The subjects were treated with a 120Hz vibratory therapy handpiece for a total time of 15 minutes
The data showed us a clear improvement of the parameters lost in the exam, in the less serious patients and a good improvement also in the most serious subjects.

INTRODUCTION

The "muscle tone" can be defined as the level of tension of a muscle at rest maintained so that at every motor command, voluntary or reflex, it is able to respond promptly and precisely.
Although the muscles are endowed with reflex activity that is independent of the command of the cortex, they actually constitute a unique and inseparable system. Not surprisingly, damage to the central nervous system seriously alters the motor skills of the individual.
In fact, it must be remembered that the peculiar characteristic of cortical activity is mainly of an inhibitory nature. The simple motor act of grasping an object translates, on a cerebral level, not so much with the excitement of the muscles delegated to perform that given movement, but with the inhibition of those that would hinder its harmony and fluidity (Bucholz, 1994).
The meaning of spasticity, as a consequence of damage to the nervous system, cannot be exhausted in simple "hypertonicity".
Spasticity is a motor disorder in which the increase in muscle tone represents one of the many components. The subjects affected by spasticity lose, first of all, the inhibiting capacity which prevents them from carrying out "voluntarily" the simplest and most common movements of daily life.
It is therefore necessary to know and understand the pathophysiological mechanisms underlying spasticity before being able to set up an adequate rehabilitation protocol. It is for this reason that, in the realization of this work, we are committed to deepening on the one hand the anatomy and physiology of the skeletal muscle to understand its functioning, but on the other, above all the neurological mechanisms that make both voluntary movements possible than those reflected (Glenn, 1994).
The starting point of our experimental study is precisely the analysis of the muscles that were most affected in case of spasticity.
The rehabilitation protocol presented as an experimental study is expressed through the application of mechanical sound vibrations at the frequency of 120 Hz which initially, used in a non-specific way, determined muscle relaxation in limited areas of a muscle, and increased spastic hypertonus in others of the same.
Our study, therefore, focused on the search for specific "key trigger points", which represent areas of hyper irritability and greater dysfunctional tissue characteristics which, if properly stimulated, allow a reduction in the tone of the entire muscle under examination.
The mechanical sound vibrations stimulate the mechanoreceptors present at the muscle level.
The signals they transmit are integrated into the nervous system. Without them, our brain is unable to generate the outputs necessary for carrying out motor activities.
What our study proposes is not the simple reduction of muscle tone as an end in itself, but the identification of the aforementioned "key trigger points",

whose specific stimulation allows to modify those nervous circuits that govern the reflex subcortical response and therefore spasticity, such as the reflection of the switchblade knife, the phenomenon of mutual inhibition, spastic hyperreflexia, spasms, synergies (Hagbarth, 1979; Kasdon, 1986; Penn et al., 1990).

MATERIALS AND METHODS

Presentation of the experimental study

Our work has focused on identifying specific "key points" that have proven to be useful in the treatment of spasticity with the ViSS® system (ViSS®, Vissman S.r.l., Roma, Italia).

This term refers to the identification of specific anatomical areas that represent the starting point of our treatment, expression of the maximum peripheral tissue dysfunction mediated by subcortical and medullary reflex responses and adaptations (Barassi et al., 2018).

In fact, as soon as these points were stimulated with focused mechanical sound vibration, it was possible to obtain an immediate elongation of the spastic muscle and an inhibition, after about 4 seconds, of the muscle tissue immediately adjacent to the treated area. The mechanism of neurophysiological interaction of focused vibration is expressed at the peripheral and subcortical receptor level (Barassi et al., 2019).

Our experimental study was subjected to:

- Five patients with spasticity.
- Evaluated at T0 with: Ashworth scale, Passive joint evaluation, Evaluation of active motility through MRC scales, Bahkta test (for non-functioning hand), Surface electromyography.
- The sample group was then treated with a mechanical sound system or ViSS® - Vibration Sound System.
- The subjects were re-evaluated at the seventh session (T1) and at the end of the treatment (T2).

Sample group

Five spastic female subjects, aged between 30 and 75, affected by the following pathologies: Multiple sclerosis, post ischemic hemiparesis, post neonatal quadriparesis.

The inclusion criteria were the diagnosis of a spastic neurological pathology, the presence of at least 1 point on the Ashworth scale and spasms.

The exclusion criteria include any classic therapeutic approach to spasticity, including the administration of antispasmodic drugs and infiltration with botulinum toxin.

All participants signed the informed consent for the experimental procedure, which complies with the latest revision of the Helsinki Declaration and with the procedures defined by the ISO 9OO1 standards for "Research and Experimentation"; this procedure also protects the privacy of subjects participating in biomedical research.

Means of evaluation

For the selection of the sample group, we adopted the Ashworth Scale and Spasms, establishing the need for at least one point as a necessary criterion for inclusion in our study.

Before the start of treatment (T0), the subjects were assessed through the following clinical scales: Ashworth scale, passive joint assessment, active motility assessment, Bahkta test (for non-functioning hand); in addition, they underwent an instrumental examination, i.e. surface electromyography.

Our study includes a therapeutic protocol of fifteen sessions with the Viss ® - Vibration Sound System, lasting fifteen minutes each.

The sample group was subsequently evaluated with clinical scales only exactly at the seventh session (T1). Upon completion of the rehabilitation protocol, i.e. the fifteenth session (T2), the subjects were finally re-evaluated with the same scales and the addition, this time, of surface electromyography.

The clinical scales adopted by us also take into consideration the proximal and distal joints with respect to the one / s treated. This choice reflects the need to assess whether our treatment focused on specific areas is capable of inducing changes even in the structures remote from those stimulated by us.

Surface electromyography

Patients were evaluated with surface electromyography at T0 and T2.

The instrument used for the acquisition and processing of the surface electromyographic signal is the NM4 Neuromuscular Meter (experimental device). Electromyography (EMG) is the recording of muscle electrical activity. The surface EMG uses for recording, instead of needle electrodes, surface electrodes and is therefore a non-invasive detection technique (Figures 1a, 1b and 1c).

The muscles we selected to undergo surface electromyography were:

Patient (Pt) A: - Posterior Tibial (right) - Peronieri (right) - Plantar fascia (right). Pt B: - Paravertebral (right). - Posterior cords (right) Pt C and Pt D (right), Pt E (left): - Biceps brachialis - Flexor of the carpus - Abductor of the thumb.

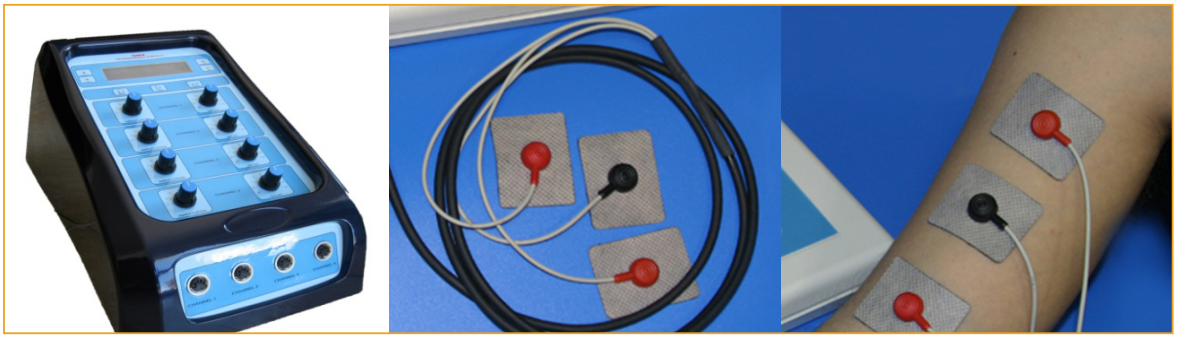

Fig. 1a, 1b, 1c - Neuromuscular measurement device NM4, superficial electrodes and example of placement of electrodes.

Fig. 2 - Sound vibrations transducer applied to be applied on the point requiring the treatment.

The NM4 software displays the electromyographic signal acquired over time, providing important indications on the resting tone and on the maximum value of the effort.

Therapeutic Device

The mechanical and sound vibration is a mechanical vibration that uses pressureless air cones to hit the skin and send a central stimulus causing deformation of the high-threshold mechanical receptors. The focused vibration of mechanical sound (Figure 2) is applied to individual muscle groups which, at appropriate frequencies, stimulate the activation of the neuromuscular spindles (300 Hz), the organs of the Golgi tendon (80 Hz) and type III and IV muscle mechanoreceptors (120 Hz). Therefore, with a signal of lowering the pressure produced by the sound with an adequate mechanical intensity, both mechanical receptors are induced by inducing muscle relaxation and the Vater-Pacini corpuscles deputies to tactile stimulation through the spinal gate control system, determine the inhibition of the passage of the nociceptive impulse. The percussions are produced by air cones without pressure which in our system follow the "all or nothing" technique thanks to the wave used, a square mechanical wave.

A fundamental element of the therapeutic effect or of the training is the number of activated receptors, therefore the increase of the interface, that is the portion of the muscular surface and of the tendon muscle subject to vibrations. It should be noted that the activation of a receptor is different from its discharge capacity, i.e. the intensity of the signal that each individual receptor emits and the number of activated receptors.

The quantity of activated receptors increases with increasing interface (contact surface); the intensity of discharge increases with the amplitude of the vibratory signal, that is with the increase in the amplitude of the vibration, that is with the greater "deformation" of the receptors.

The higher the intensity of the afferent signal, the more important the efferent response will be. Based on the above, it is clear that the amplitude of the signal and the interface with the mechanoreceptors is fundamental. (Di Pancrazio et al., 2013).

Therapeutic protocol

Our therapeutic protocol included a total of fifteen sessions lasting 15 minutes each, with a frequency of once a week. In each session, the sample group was subjected to a treatment with the Viss ® system, ie focused mechanical and sound vibrations, at a frequency of 120 Hz.

Specifically in our study, we considered it appropriate to use the "pen handpiece" (Fig.3)instead of the self-static transducers, as it lent itself better to the possibility of varying the points to stimulate during the session.

Starting from the assumption that, as is known from recent studies, the application of mechanical sound vibrations at the frequency of 120 Hz selectively stimulates type III and IV mechanoreceptors inducing a reduction in muscle tone, we have tried to identify "deductively and experimentally" some anatomical areas that, more than others, were sensitive to this stimulation. We therefore hypothesized that these areas were more densely rich than the aforementioned mechanoreceptors and, for this reason, they were classified by us as "key points" (Figure 3a).

In the proposed treatment, the key trigger points were stimulated according to a distal-proximal or caudal-cranial spatial criterion, since we assumed that, since the muscle origin is a fixed point, the areas contiguous to the tendon insertion were more rich in free nerve

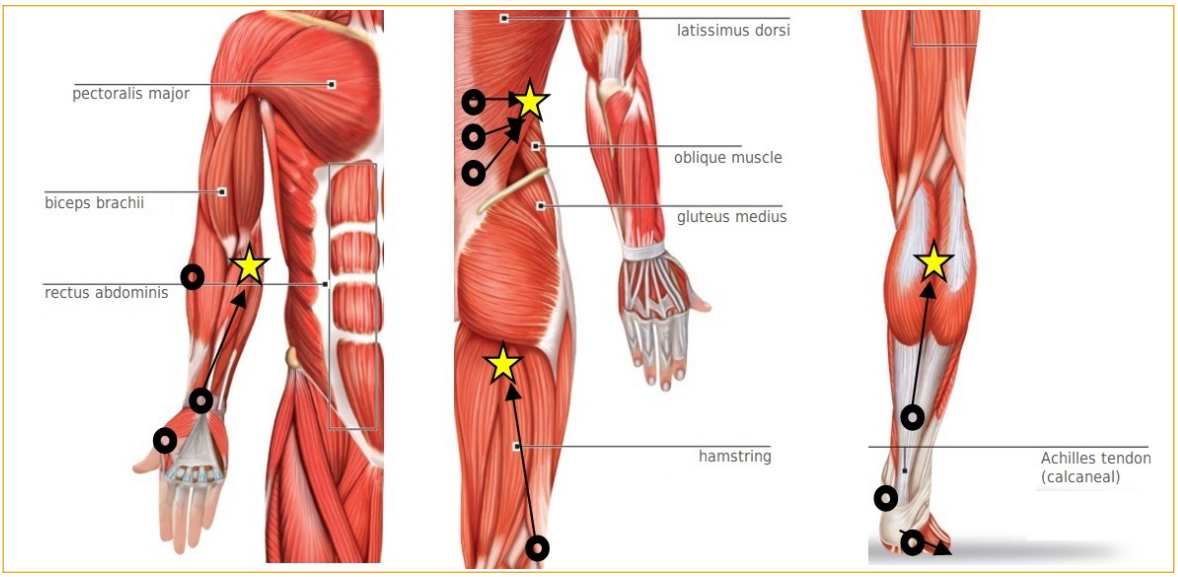

Fig. 3a - The dark circles correspond to the anatomical areas that we classify as key points. The arrows show the direction in which you can move the pen handpiece after successfully stimulating the key point in question. The yellow stars indicate the area where the treatment of the stimulated region can be concluded.

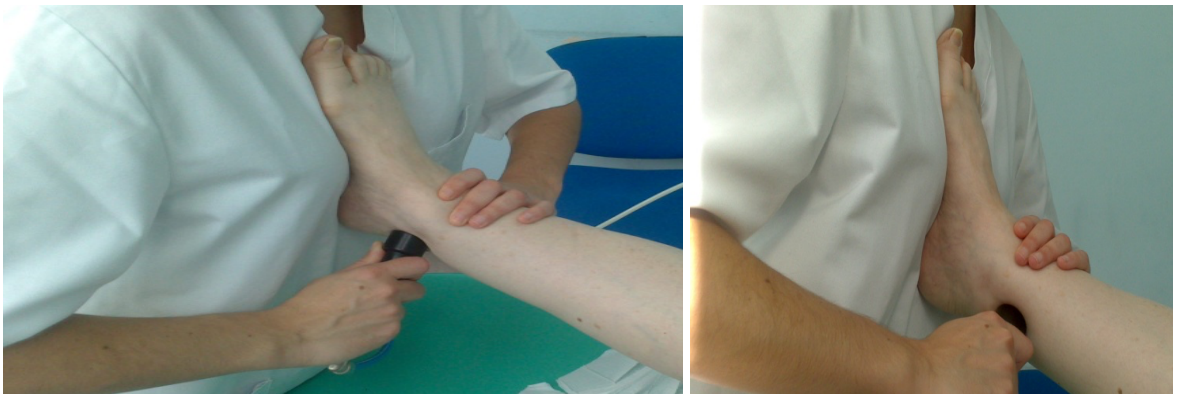

Fig. 4a, 4b - Medially to the Achilles tendon.

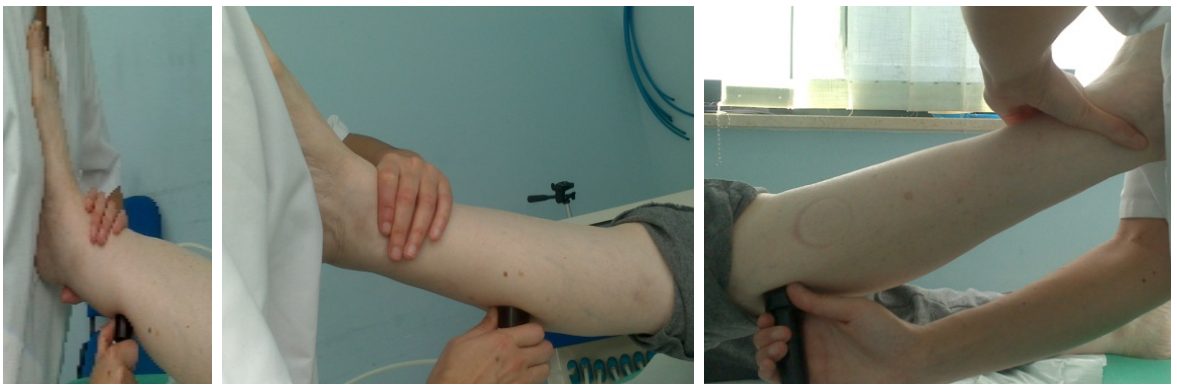

Fig. 4c, 4d, 4e - *A*t the level of the lower middle third of the leg, between the tibia and fibula, in correspondence with the crural interosseous membrane, following its course.

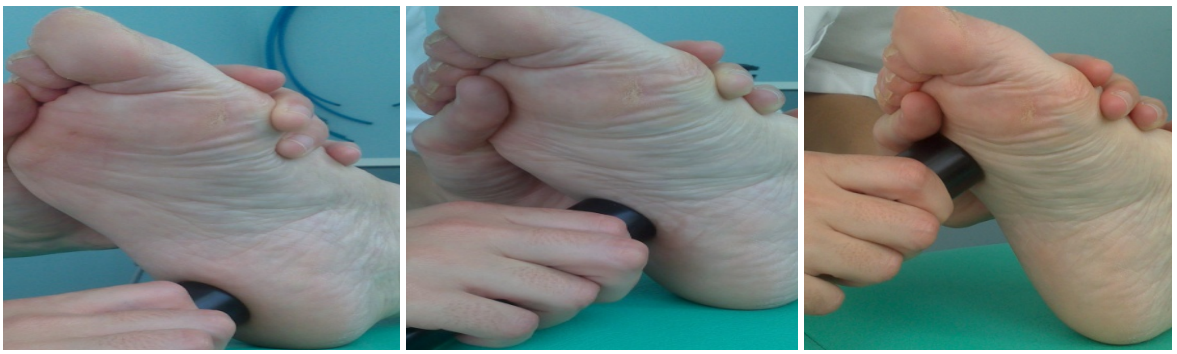

Fig. 4f, 4g, 4h - Laterally to the heel, at the insertion level of the long peroneal muscle at the level of the lateral bundle of plantar aponevrosis (or metatarsal calcaneal ligament), following its course.

endings . Furthermore, we have experienced that only after having adequately inhibited a key point was it possible to continue with the immediately adjacent structures, taking care not to deviate too much from the point just treated to avoid a spastic hypertonic reaction. The success of the stimulation of a key point was evidenced by the sudden relaxation of the muscle under examination, just as happens in the reflex of the switchblade knife: this allowed us to lengthen the affected muscular district and to mobilize the joints with extreme ease and in the absence of pain for the patient. The failure of the stimulation, however, caused an increase in hypertonicity and not infrequently the onset of muscle spasms. This could happen for variables dependent on the patient (from irritating spasticity spines) or the operator (error in positioning the handpiece, inadequate stimulation time). In these cases it was necessary to place the pen handpiece again on the first key point of that muscular district.

The stimulation time was on average 4-5 seconds, based on the severity of spasticity.

Finally, it was possible to repeat the procedure within fifteen minutes of the session, until the maximum reduction in tone allowed was achieved.

Directions of the treatment

Below we illustrate the detailed dircetions followed with the handpiece during the treatment (Figures 4a-4x).

RESULTS

The quantitative analysis of the collected data was performed using Office Excel software (Microsoft®, Redmond, Washington, USA), through which it was possible to create a graphic representation of the average variation of the values taken into consideration, through the calculation of the mean, standard deviation, percentage improvement and Student's T Test, to

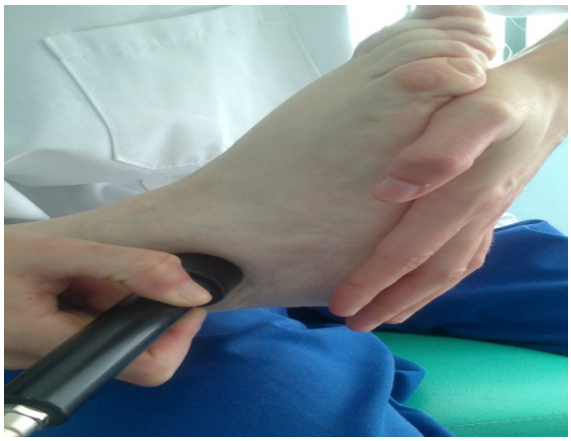

Fig. 4i - At the level of the lateral malleolus, near the common sheath of the tendons of the peroneal muscles.

evaluate their statistical significance.
Abuot passive ROMs, there is a clear improvement in joint mobility in all districts, both treated and neighboring, with particular statistical significance in the treatment of the upper limbs (Figure 5).
Positive were also the data on muscle strength assessed by the scale of muscle strength / weakness (MRC) as shown in the tables and graphs below (Figure 6).
Also with regard to the Ashworth scale, the results were very positive (Figure 7).
Statistically significant was the result of the Bahkta test (Figure 8).
And even more relevant the results obtained with surface electromyography detected at rest (Figure 9).

CONCLUSIONS

The regulation of muscle tone helps to maintain a normal posture and to facilitate movements (Sinkjær et al., 1999).
When a muscle stretches, the neuromuscular system can respond by automatically altering muscle tone. This modulation of the stretch reflex is important in controlling movement and maintaining balance.
Spasticity is manifested by an increased stretch reflex that intensifies with the speed of movement (Lance, 1980).
This results in excessive and inappropriate muscle activation which can contribute to muscle hypertonia. Spasticity is a known impairment following an upper motor neuron injury, such as cerebral palsy. Spasticity is often considered the most common motor impairment. However, there are many uncertainties regarding the contribution of spasticity to hypertonia and, in particular, its contribution to the observed gait anomalies (Sanger et al.. 2003).
What is clear from the data shown is certainly the validity of the focal application of mechanical sound vibrations in the treatment of spasticity.
All the patients included in our experimental protocol showed, although with different degrees, an improvement of all the evaluation parameters taken into consideration, except for the increase, even if modest, of the minimum value of the basal tone in some muscles in two patients .
This is justified by the fact that basal muscle tone, especially in patients with spasticity, is variable in relation to factors both external and internal to the subject.
Certainly the most interesting results emerge especially in those subjects with mild / moderate T0 spastic-

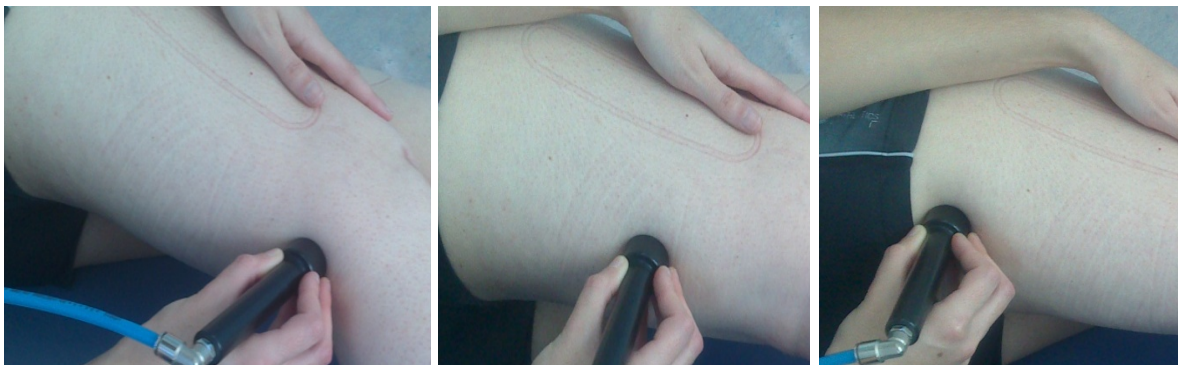

Fig. 4j, 4k, 4l - At the level of the lateral epicondyle of the femur, posteriorly, near the tendon of the biceps femoral muscle, following the course of the muscular abdomen in the cranial caudal direction, and at the level of the ischial tuberosity, the site of origin of the hamstring and semi-tendinous muscle.

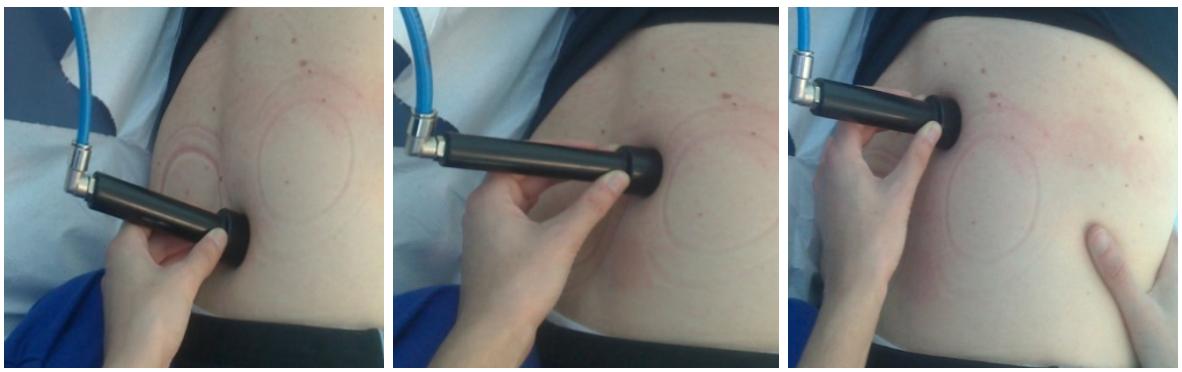

Fig. 4m, 4n, 4o - Laterally to the spinous apophysis of the lumbar spine, along the course of the lumbosacral fascia, on three points: L3, L4, T12.

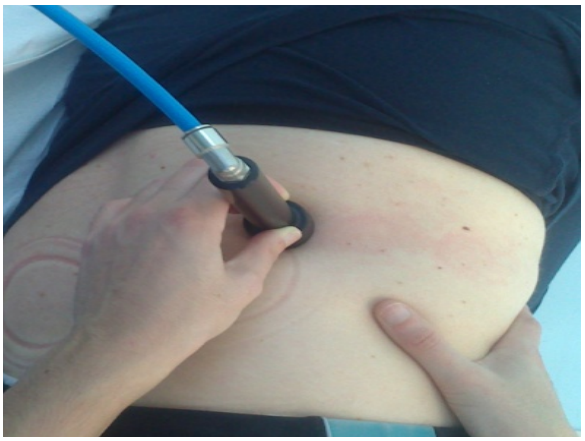
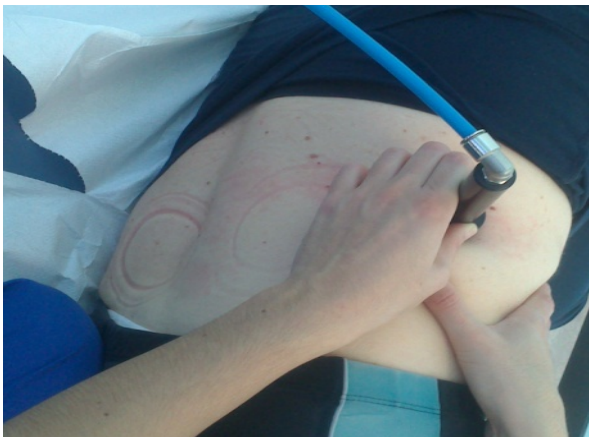

Fig. 4p , 4q - Laterally to the spinous apophysis of the lumbar spine, along the course of the lumbosacral fascia, on three points: L3, L4, T12.

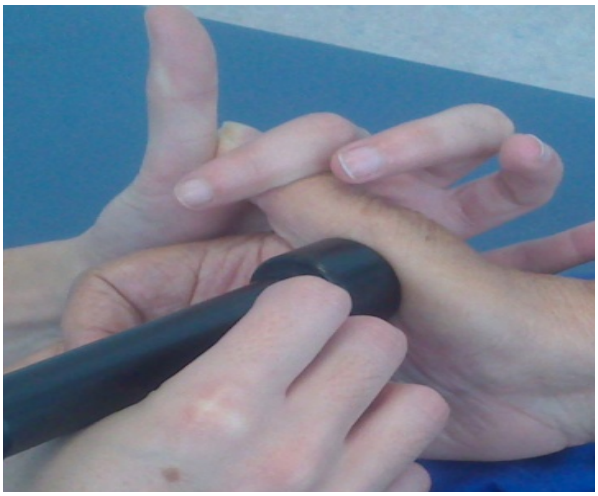
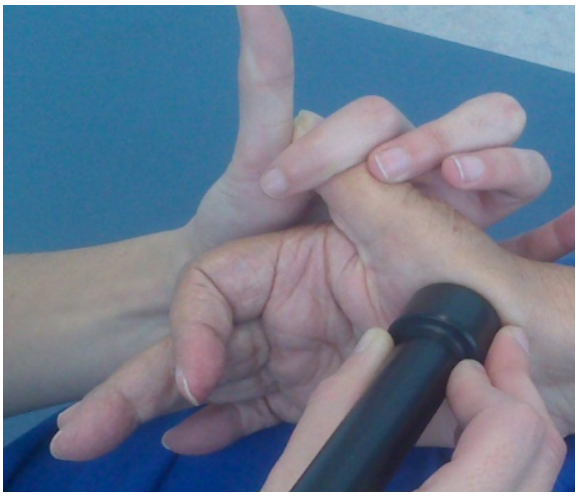

Fig. 4r , 4s - *A*t the level of the tender eminence of the hand, near the tendons of the abductor muscles short, short and long flexor, opponent of the thumb.

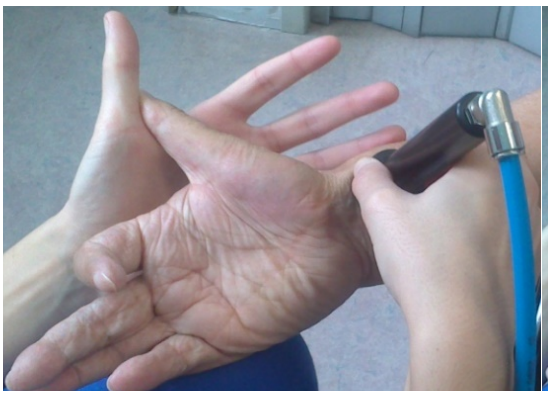
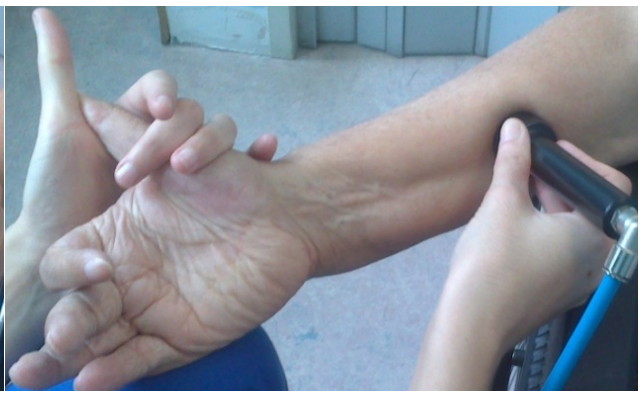

Fig. 4t , 4u - At the level of the tendons of the abductor muscles of the long thumb and radial flexor of the carpus, near the flexor retinaculum.

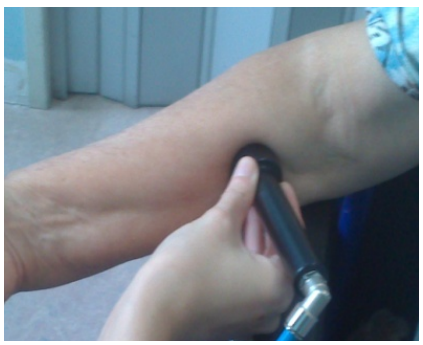
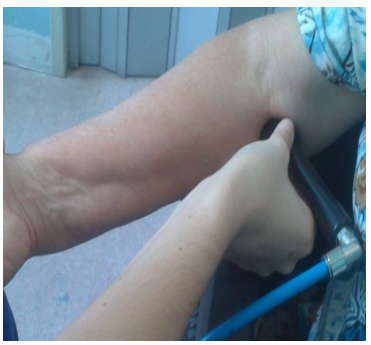
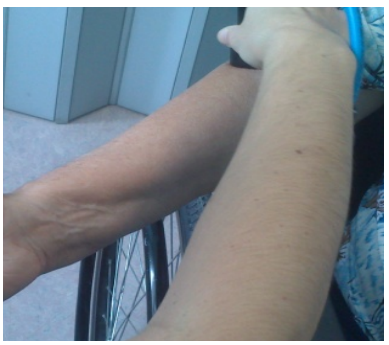

Fig. 4v, 4w, 4x - At the level of the half of the lateral face of the radius and, subsequently, of the medial epicondyle of the humerus, respectively insertion and origin of the pronator teres muscle and at the level of the radial tuberosity, where the

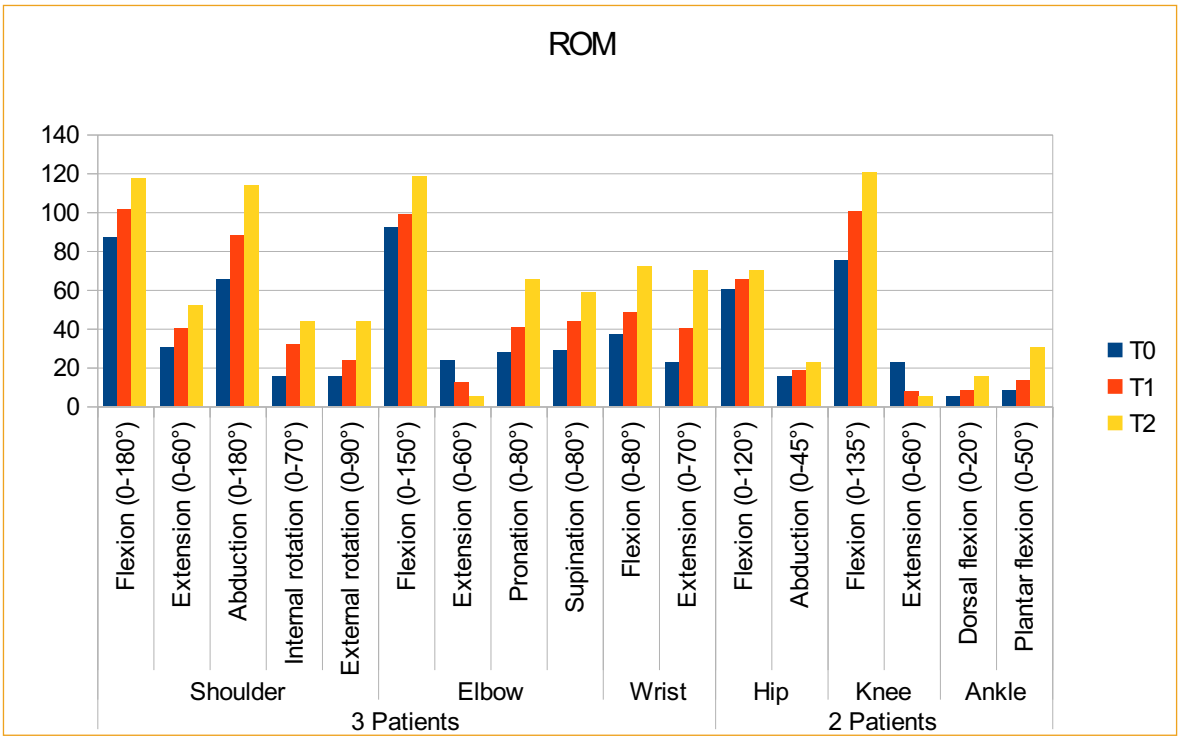

Fig. 5 - Variations of ROM.

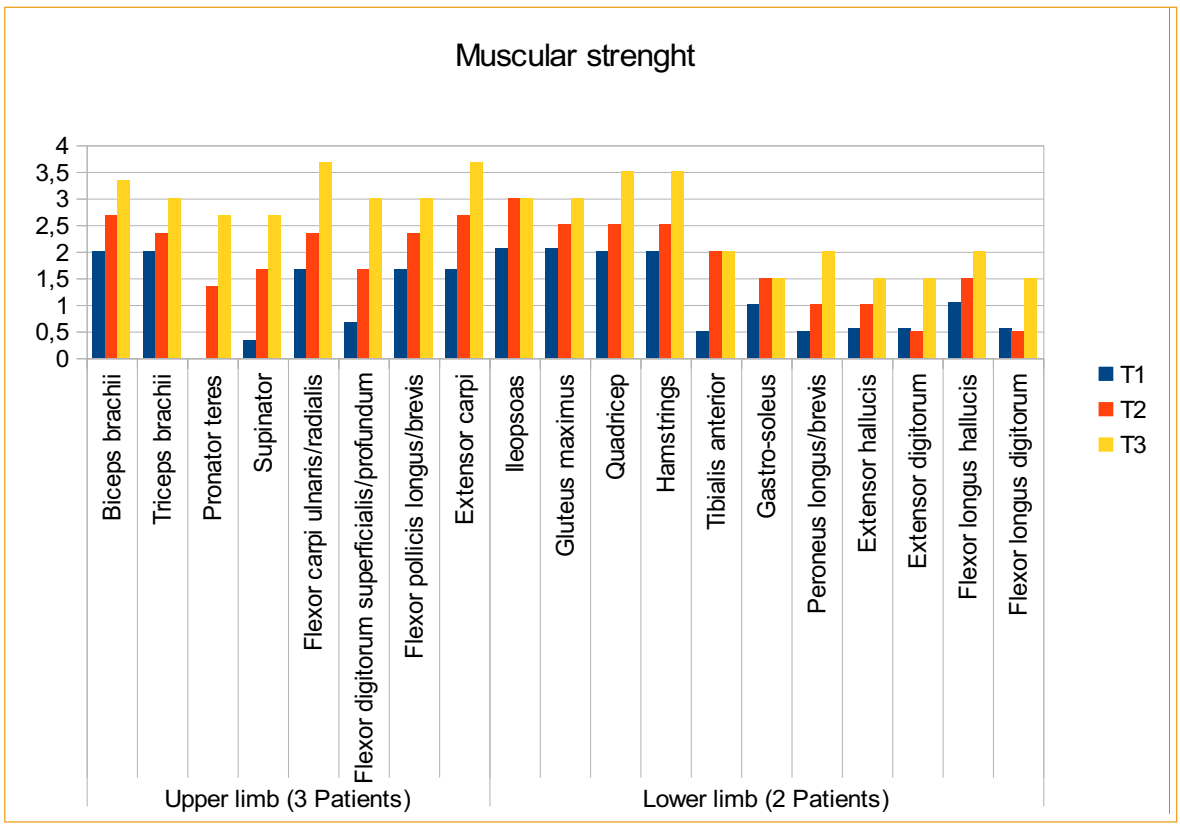

Fig. 6 - Variations of muscular strenght.

ity, compared to the only case of severe spasticity, in which the improvements, even if present, are minimal and perhaps irrelevant from a clinical point of view. .
The key points we have identified "experimentally" are all located near structures of a fascial, connective, tendinous and ligamentous nature. This is consistent with the "interstitial" location of the type III and IV mechanoreceptors, involved in the switchblade knife phenomenon.

Much of the uncertainty in the treatment of spasticity is related to bad communication relating to the definition and evaluation of spasticity.
In clinical terms, hypertonia is assessed as "passive stretch resistance while the patient maintains a relaxed state of muscle activity".
With spasticity-related hypertonia, the lack of modulation of the stretch reflex causes premature and / or exaggerated muscle contraction that can resist passive

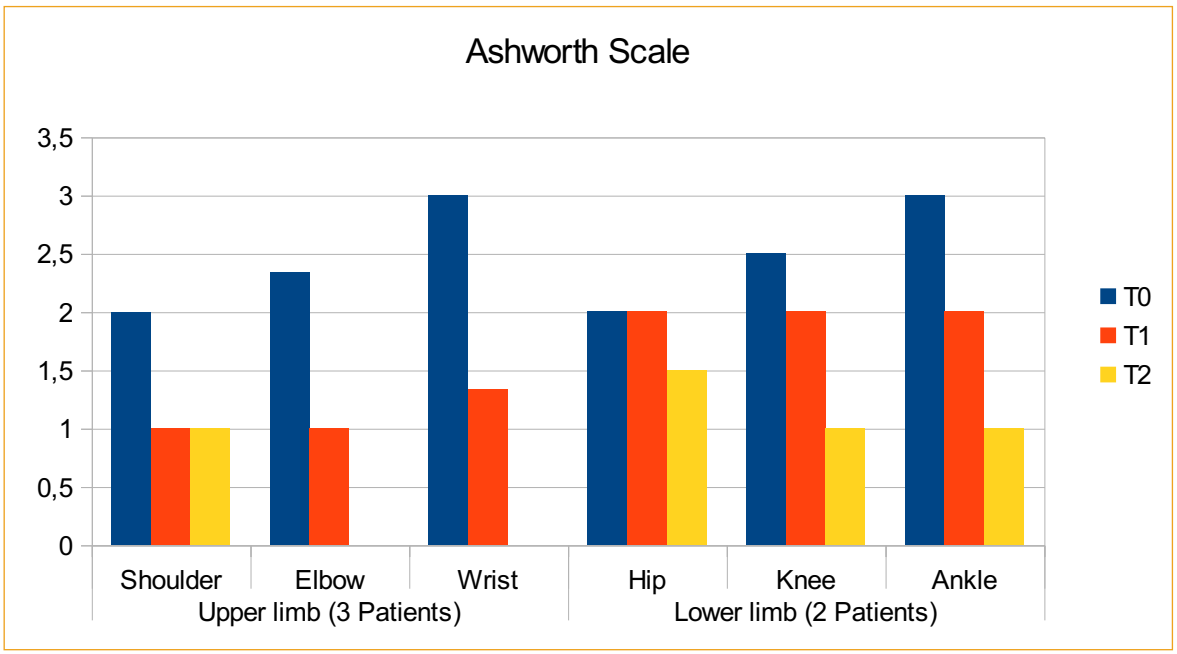

Fig. 7 - Variations of Ashworth Scale.

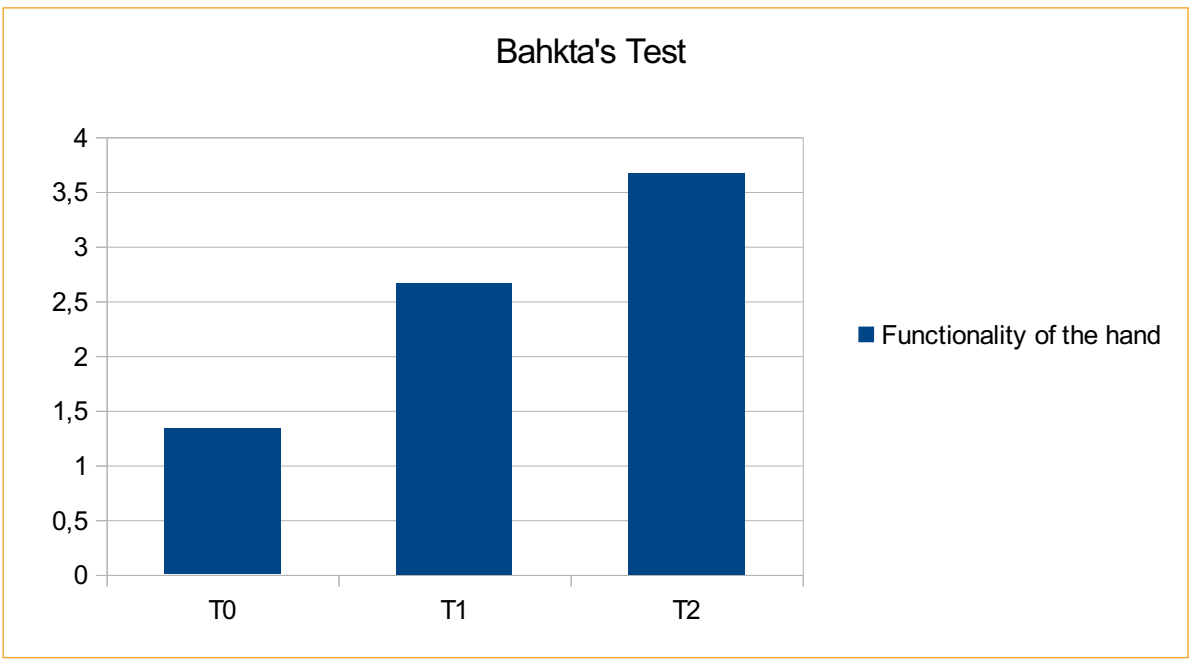

Fig. 8 - Bahkta's test.

stretching (Sanger et al.. 2003).
First, it depends on the dependence of an examiner's subjective interpretation; secondly, the speed of stretching and the level of relaxation of the muscle are uncontrolled; and thirdly, it does not allow the differentiation between the contributions of neural and non-neuronal components to the overall resistance perceived during muscle elongation. The non-neurological mechanical muscle properties such as stiffness and viscosity are often altered in the neurological alterations that cause spasticity and can also contribute to the sensation of greater resistance to passive movement (Lieber, 2010).
From the foregoing it can be concluded that hypertonia should be considered multifactorial and that equating all resistance to passive movement with spasticity is wrong.
Spasticity contributes only to this resistance and the extent of the contribution probably differs between muscles and individuals.
In this regard, the main objective should be to adequately analyze the components of hypertonia.
Recent developments in the instrumental treatment of spasticity provide a solid methodological basis on which to develop a more specific treatment of hypertonia in its components.
Given the small size of this study sample, and the limitations resulting from this small study, further clinical research is needed to validate and improve our treatment modalities and reduce the effect of hypertonicity on movement.

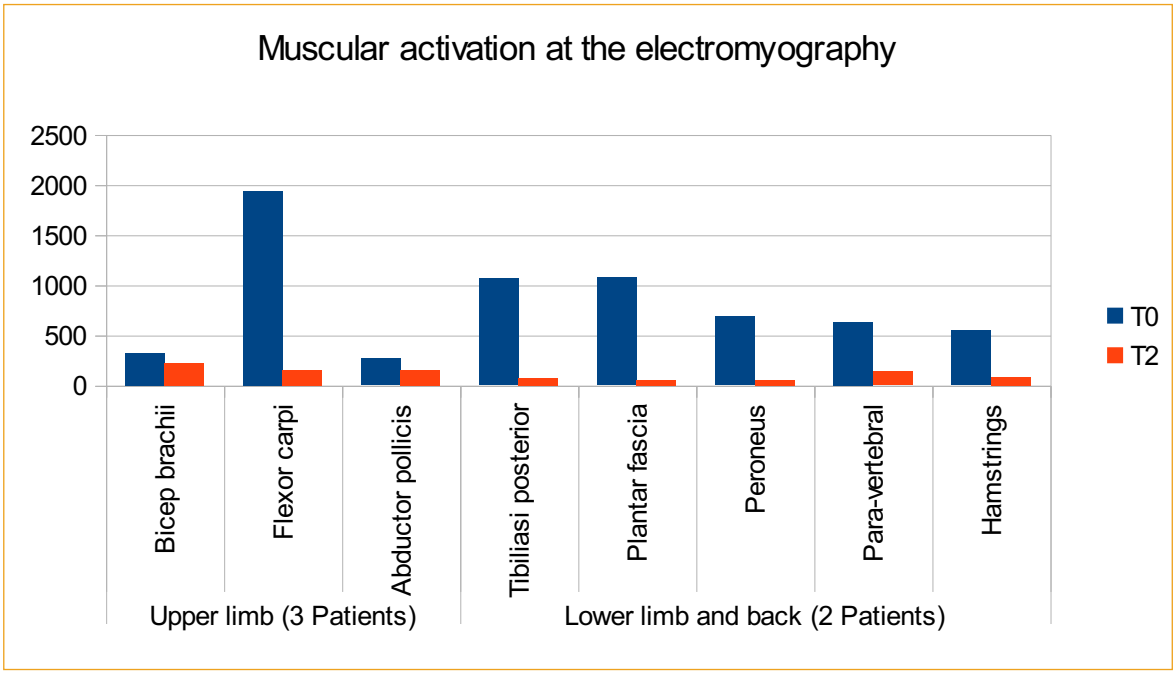

Fig. 9 - Values of musculary activation at the surface electromyography.

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