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To Study the H-Reflex in Spastic Hemiplegics Before and after Passive Movements on CPM Device

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Abstract: **Rehabilitation after stroke remains a challenge. Passive Movements are commonly used to help the stroke survivor recover from Physiologic impairments caused by the lesion of the CNS. When spastic Hypertonia is identified, joint mobilisations and muscle stretching are considered an integral part of the patient's daily management, continuous passive motion is one of them. The present study sought to assess the effects of continuous passive motion on spasticity in stroke patients.**

Objectives of the study:

1. **To study the H-Reflex parameters and H/M ratio in spastic hemiplegics both on the affected and normal side.**
2. **To study the association of H/M Ratio to clinical grades by MAS.**
3. **To study the effect of continuous passive motion on H-reflex, H/M Ratio and spasticity.**

Study Design: Interventional cross-sectional.

Methodology: The total study Sample was 21 participants – 13 Males and 8 Females. All the participants were the spastic hemiplegic patients in the study group and the normal lower limb served as the control. A detailed Neurological examination was done. The H-reflex study and the clinical grades by MAS (Modified Ashworth Scale) was done before and after passive movements on CPM device. Passive movements were given for 20 Minutes.

Main outcome measures: MAS, Soleus H-Reflex, H_{max}:M_{max} ratio.

Results: The spastic hemiplegics by MAS were in the range of 1+ to 2 before the CPM. Also, the H amplitude and H/M ratio studied, was mean 6.00 +4.81, SE 1.05 and 0.51 + 0.5, SE 0.10 before CPM respectively. Following CPM of 20 minutes, MAS were in the range of 1 to 1+. Also, the H amplitude and H/M ratio were 4.18+3.37, SE 0.73 (P-value 0.001) and 0.28+0.21, SE 0.04(p-value 0.019) respectively. Comparison of MAS and H/M ratio was also studied and showed there was strong relationship between the spasticity by MAS and H/M ratio post CPM (p-value 0.001), However Pre-CPM it was not significant (0.17).

Conclusion: The study indicates that continuous passive movement brought about significant alterations in spasticity post CPM. Subjectively all the patients reported significant benefits from 20 minutes of CPM in the form of reduced heaviness of the affected limb with improved strength and easy walking. The measures used, MAS and the H/M ratio used to grade spasticity were significantly reduced and correlated post CPM.

Keywords: **H-reflex, Spastic hemiplegia, MAS, H_{max}:M_{max} ratio, CPM device.**

1. INTRODUCTION

"Spasticity is a motor disorder that is characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome"

Lance described spasticity as a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes.

The term "Velocity dependent" means "the faster the passive movement of the limb through its available range, the greater the increase in muscle tone." This widely- accepted definition was broadened by Young to include other signs like exaggerated deep tendon reflexes, clonus, flexor/extensor spasm, the Babinski response (Babinski's sign), exaggerated phasic stretch reflexes, hyperactive cutaneous reflexes, increased autonomic reflexes, and abnormal postures. All of these signs describe manifestations of excessive involuntary motor activity.

The Medical literatures supports the notion that insufficient descending inhibition results in structural and physiologic reorganization of segmental circuits following injury or dysfunction within the UMN pathways, such as alterations of intrinsic and extrinsic properties of motor neurons and interneurons.

There is no single pathophysiologic mechanism to account for all the observable aspects of spasticity. Dysfunction with in the central nervous system of descending pathways to and within the spinal cord causes a UMN syndrome that is often associated with exaggerated reflexes and spasticity, which includes velocity dependent increased muscle tone.

Although the spinal alpha motor neuron is considered to be the final common pathway for expression of spasticity, one should consider the more complex motor pathway involved in the disorder movements of spastic brain injured patients. Spastic hypertonia encompasses a variety of conditions, including dystonia, rigidity, myoclonus, muscle spasm, posturing, and/or spasticity.

The Physiologic and pathophysiologic mechanism of spasticity includes:

1. The Monosynaptic reflex
2. The inverse stretch reflex
3. Elevated reflex activity
4. Multisynaptic segmental connections

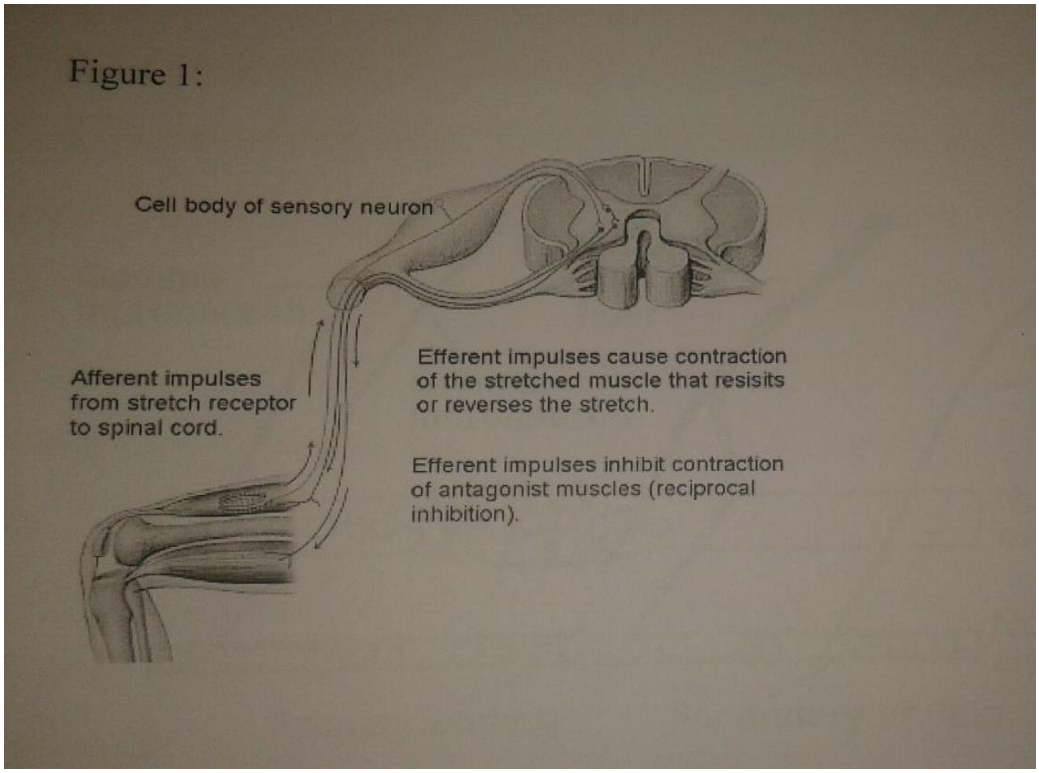


Figure 1: Spinal stretch reflex

The physiologic components involved **in the spinal stretch reflex response (Monosynaptic Reflex)** include the muscle spindle stretch receptor, the myelinated sensory neuron, the synapse, the homonymous motor neuron, and the muscle it innervates. This stretch reflex has two components: a brisk, short-acting phasic component that responds to the initial dynamic change in length, and a weaker, longer acting tonic component that responds to the steady stretch of the muscle at a new length.

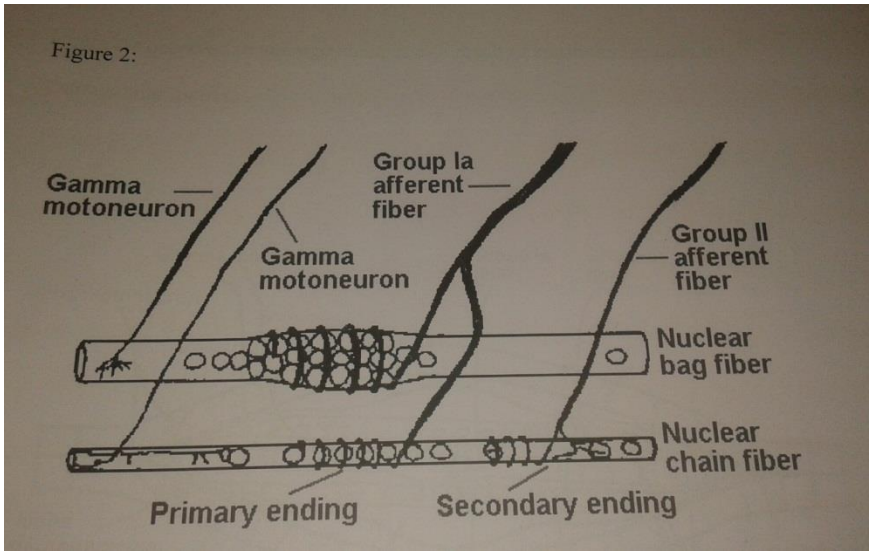


Figure 2: Intrafusal receptor organ

A change in muscle length can evoke a stretch reflex. Modified muscle fibers (intrafusal receptor organs) that detect changes in muscle length are called muscle spindles. Nuclear bag fibers are further divided in to dynamic and static nuclear bag fibers. Dynamic nuclear bag fibers are highly sensitive to the rate of change, providing velocity sensitivity to muscle stretch. Static nuclear bag fibers and nuclear chain fibers are more sensitive to the steady state, static or tonic, muscle length(figure 2).

The structural differences between these fibers are responsible for the physiologic differences in their sensitivities and for the two different components, phasic and tonic, of the stretch reflex, Intracellular muscle fibers are observed to undergo changes as a result of spasticity, as does the extracellular matrix.

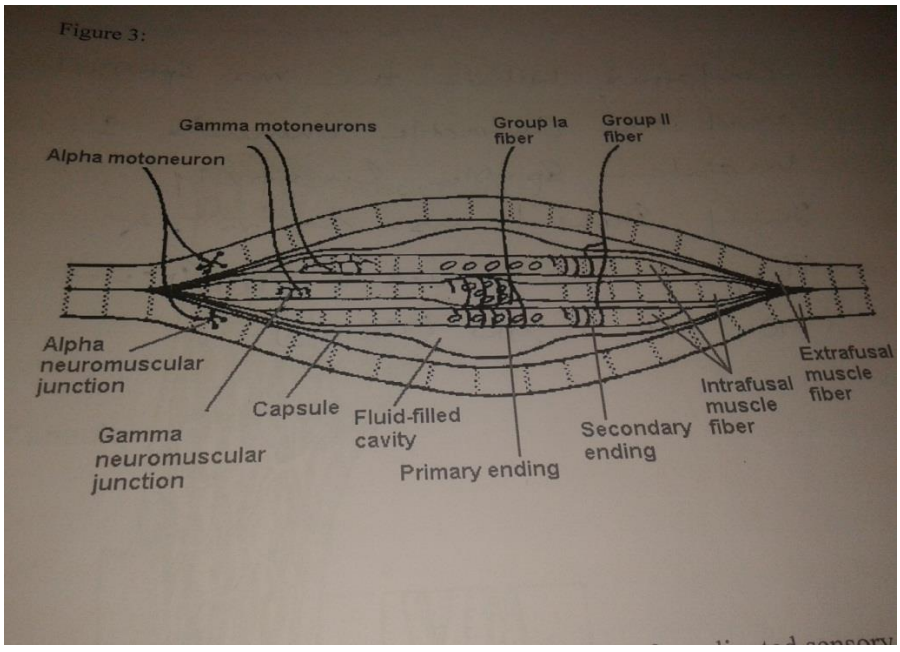


Figure 3:

Group Ia and group 2 fibers are two types of myelinated sensory afferent fibers that innervate intrafusal fibers. Group Ia, or primary sensory afferent (17 µm in diameter) is excited by both the nuclear bag intrafusal fibers and the nuclear chain fibers. Conversely, Group 2 or the secondary ending (8µm in diameter) fibers is usually excited only by the nuclear chain fibers (figure 3). Contained with the muscle spindle unit are contractile elements that stiffen the region of the nuclear bag fibers. These contractile elements maintain spindle sensitivity during skeletal muscle contraction. They are innervated by special motor neurons known as the γ motor neurons (5µm in diameter).

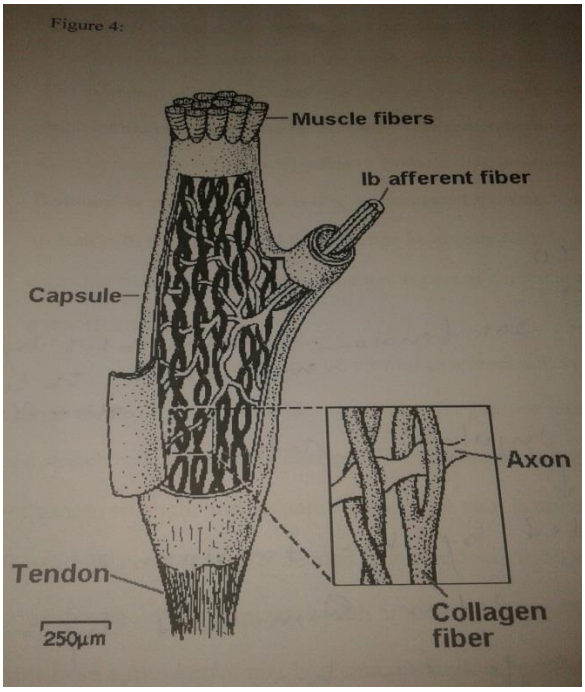


Figure 4:

The golgi tendon organ (GTO) is sensitive to intramuscular tension and is innervated by myelinated IB sensory afferents (16µm in diameter). The golgi tendon organ is particularly sensitive to muscle tension created by active muscle contraction, but has a high threshold for detecting passive stretch. Stimulation of Ib afferents leads to inhibition of the homonymous motor neuron and its synergists. The excitation of tis antagonistic motor neurons also stimulates Ib afferents. This behavior has called **the inverse myotatic reflex,** because its actions oppose those of the stretch (myotatic) reflex. It is also called Ib non-reciprocal inhibition. **It should be noted that this reflex is stimulated by muscle tension, whereas the stretch reflex is stimulated by a change in muscle length.** The golgi tendon organ has been hypothesized to function as part of a muscle tension feedback system (figure 4).

The stretch reflex can be viewed as a feedback system with muscle length as the regulated variable. Normally, the gain, or input-output relationship, of the stretch reflex to a given change in muscle length is kept low by descending influences when the individual is at rest. The gain , or input-output relationship, of the stretch reflex to a given change in muscle length is kept low by descending influences when the individual is at rest. The gain is enhanced when physical demand for performance is needed. Hyperreflexia is an example of segmental reflex dysregulation associated with an UMNL. Theoretically, hyperreflexia can result from a number of mechanisms, including decreased spinal inhibitory mechanisms from brain centres, hyperexcitability of α motor neurons, peripheral nerve sprouting, and increased γ fiber activity.

Long term reductions in inhibition can contribute to hyperreflexia. Examples of inhibition types are as follows: recurrent Renshaw inhibition, reciprocal Ia inhibition, presynaptic inhibition, non-reciprocal Ib inhibition, and inhibition from group 2 afferents. Various lines of research have supported deficient presynaptic and non-reciprocal inhibition as significant contributors to spasticity. The supportive evidence for it being due to deficient group 2 afferent related and Renshaw inhibition is lacking. Presynaptic inhibition is mediated via a GABAergic mechanism that decreases the efficacy of Ia transmitter release. Interneurons involved in presynaptic inhibition are modulated by desecending pathways.Thus the loss or reduction of rostral lesion control can reduce tonic levels of descending facilitation on inhibitory interneurons, leading to increased α motor response to normal Ia input.

The Ia inhibitory interneurons are normally controlled by descending excitatory pathways. Reciprocal Ia inhibition decreases the chance for co-contraction of antagonistic and agonistic muscles during voluntary movement. There is evidence for decreased excitability of Ia inhibitory neurons after rostral lesions of the CNS. This dysfunction could lead to an increased co-contraction and weakness of voluntary movement. Non-reciprocal Ib inhibition has been found to be decreased or even replaced by facilitation in patients with spastic paresis and spastic dystonia, in this case both stroke and SCI subjects, but not in subjects without spastic dystonia.

Renshaw cells are inhibitory neurons that are stimulated by collateral axons from α motor neurons(14μm in diameter). When an α motor neuron fires, it stimulates a Renshaw cell that, in turn, inhibits the initiating motor neuron fires, it stimulates a Renshaw cell that, in turn, inhibits the initiating motor neurons and its synergists. The Renshaw cell also inhibits the Ia inhibitory interneuron associated with the initiating motor neuron. Because the Renshaw cell inhibits Ia inhibitory interneurons as well as agonist α motor neurons might contribute to spasticity by decreasing reciprocal Ia inhibition.

Hyperexcitability of α motor neurons might contribute to spasticity. Examples of primary changes in membrane properties that would be expected to produce increased α motor neuron discharge include a reduction in the area of dendritic membranes, deafferentation dendritic hyperexcitability, and an increase in the number of excitatory synaptic inputs due to sprouting.

The majority of spinal segmental connections are polysynaptic. Interposed interneurons connect sensory afferents and antagonistic motor neurons to opposing muscle groups, resulting in a polysynaptic connection, Interneurons also receive excitatory and inhibitory signals from descending pathways.

Supraspinal centers can control joint stiffness through the modulation of excitatory and inhibitory input to segmental interneurons and interneuronal networks.

The interneurons that mediate Ib non-reciprocal inhibition connect inhibitory agonist and excitatory antagonistic motor neurons. At rest, Ib non reciprocal inhibition opposes the actions of the stretch reflex. Convergent input from Ia spindle afferents is received by Ib interneurons, along with low threshold cutaneous afferents and joint afferents excitatory and inhibitory inputs from descending pathways.

The afferents for Golgi tendon organs make polysynaptic connections, via interneurons, to homonymous motor neurons, synergist and antagonist motor neurons. Because of golgi tendon organ sensitivity to active muscle tension and the short-latency convergent input from Ia spindle afferents that Ib interneurons receive, cutaneous afferents, joint afferents, and modulating descending pathways, spinal interneuronal networks are likely to play and important role in exploratory movements of the limbs. A functional example of this network organization would be the reduction of muscle contraction if a limb encountered an unexpected obstacle. The interneuron receiving Ib afferent information would mediate the inhibition of the agonist, which would reduce the force against the impediment. Also, Ib inhibition could function to decrease muscle contraction at the extreme range of joint motion. The net effect of Ib inhibition during volitional activity depends on inputs from multiple sources. Recurrent Renshaw inhibition takes place via polysynaptic connections to α motor neurons via Renshaw cells and Ia inhibitory interneurons.

The majority of type 2 afferent connections are polysynaptic and involve several classes of interneuron. These interneurons typically arise from muscle spindles, but some afferents originate as free nerve endings or in other types of receptors. Their activation tends to activate flexor synergistic muscles and inhibits physiologic extensors. When unopposed, group 2 mediated activity produces tonic activation of physiologic limb flexors. Group 2 mediated activity produces tonic activation of physiologic limb flexors. Group 2 and 4 affer ents originate from deep muscle and cutaneous receptors. Group 3 fibers are thinly myelinated. Group 4 fibers are small diameter afferents, often unmyelinated, and originate as free nerve endings serving nociceptive and thermoregulatory functions. Both types of fiber convey impulses generated by extreme pressure, heat and cold. Similar to type 2 responses, the reflex responses to these stimuli are bilateral flexion predominantly, and are typically proportionate to the stimulus intensity.[3]

In stroke, an increase occurs in both tonic and phasic reflexes. Loss of upper motor neuron control causes disinhibited α and γ motor neuron activity and heightened sensitivity to class Ia and 2 muscle spindle afferents.

Consequently, monosynaptic and multisynaptic spinal reflexes become hyperactive. Spasticity develops shortly after completed stroke, and is initially manifested as an increased phasic response to tendon tap and a slight catch with passive ranging. Later, ranging can become difficult, and the patient might show tonic positioning in flexion or extension, often, as voluntary motor activity returns, a reduction in tone and reflex response is noted, but if recovery is incomplete, spasticity usually remains.[5]

Hemiplegia, a paralysis of one side of the body, is the classic sign of neurovascular disease of the brain. It is one of many manifestation of neurovascular disease, and it occurs with strokes involving the cerebral hemisphere or brainstem.[6]

Stroke is the commonest cause of hemiplegia. A stroke is the rapidly developing loss of brain function(s) due to disturbance in the blood supply to the brain. This can be due to a haemorrhage. As a result, the affected area of the brain is unable to function, leading to inability to move one or more limbs on one side of the body, inability to understand or formulate speech, or Visualfield defects.[7]

In the past, stroke was referred to as cerebrovascular accident or CVA but the term "stroke" is now preferred.

The traditional definition of stroke, devised by the world Health organization in the 1970, is a "neurological deficit of Cerebrovascular cause that persists beyond 24 hours or is interrupted by death within 24 hours".[8]

This definition was supposed to reflect the reversibility of tissue damage and was devised for the purpose, with the time frame of 24 hours being chosen arbitrarily.

The 24- hour limit divides stroke from transient ischemic attach, which is a related syndrome of stroke symptoms that resolve completely with 24 hours.

With the availability of treatments that, when given early, can reduce stoke severity, many now prefer alternative concepts, such as **brain attack** and **acute ischemic cerebrovascular syndrome** (modelled after heart Attack and acute coronary syndrome respectively), that reflect the urgency of stroke symptoms and the need to act swiftly.[9]

A stroke of any type can cause spastic hemiplegia. There are other certain causes for spastic Hemiplegia.

Head injuries can cause hemiplegia as well, as can brain tumors. Sturge Weber syndrome, a congenital condition characterized by vascular problems.

Meningitis, encephalitis, and other brain infections can also cause spastic hemiplegia. Some cases of spastic hemiplegia are caused by hereditary disease known as leukodystrophies. In addition, if malformations of the arteries and veins called arteriovenous malformations (AVM) are present in the brain they can cause spastic hemiplegia. Seizures sometimes are present in spastic hemiplegia. Migraine syndrome can also lead to spastic hemiplegia.

Spastic hemiplegia can cause trouble with walking; a patient may have difficulty with balance. The muscles on one side of the body may be stiff and weak (This is the most common symptom).

Spastic hemiplegia can lead to limb deformities. A common problem is equinus ankle. Because of the spastic muscles the foot is not able to flex normally and is drawn in to a position which leads to toe walking.

Adults with toe walking caused by spastic hemiplegia do not out grow the problem and it can lead to an increasingly awkward gait, as well displacement of the hip joints and a tendency to fall.

There are a number of spasticity measuring tools, which range from simple questionnaires and goniometry evaluations to more technologically complicated electromyographic and biomechanical analysis of limb resistance to mechanical displacement and video monitoring assessment of joint mobility.

The Oswestry scale of grading, Degree of adductor Muscle tone are some of the tone intensity scales used to assess spasticity. Other two rarely used methods of observing the spasticity phenomenon is to assess the number of episodic spasms. The Penn spasm frequency score is an ordinal ranking of the frequency of leg spasms per day and per hour. One problem with this scale is that patients usually report that the number of spasms occurring per hour is often affected by their activity at the time. For example, they tend to report few spasms if resting comfortably, more if physically active. Also, the duration of each spasm is not taken in to consideration.

The causal observation of the free swing of the knee in the "pendulum test" was formalized and provided objective data by the use of video motion analysis. The advantages of video motion analysis of the pendulum test include the ability to do the analysis anywhere the videorecorder is available, freedom from the attachment of cumbersome recording devices to the patients, and processing by a non-biased 'blinded" observer (who has had no contact with the patient).[3]

However, of the many clinical monitoring tools, most commonly used assessment method is the Ashworth scale. This has the advantage of ease of use in the clinical setting. This asset has been utilised in a number of pharmaceutical trial of antispasticity medications in which a simple measurement tool can be used easily by participating clinicians to assess the efficacy of the intervention.

Ashworth developed the most commonly used scale in 1964 and in 1987 Bohannon and smith [10] modified the original Ashworth scale by adjusting the lowest number from 0 to 1, and the highest scale from 4 to 5. Another modification from the original scoring scheme was the addition of a point between 1 and 2, where 1 was a catch earlier in the joint motion range nearer to midpoint.

As proposed originally, the Ashworth Scale is a simple five point Likert scale in which the observer's subjective opinion of the subject's resting muscle tone ranges from 'normal' at the lowest grade to rigid at the highest.

A recent comprehensive review of engineering and medical literature concluded that the Ashworth scale is in common use and has significant interrater agreement and food reliability, but it is not a functional outcome measure and can be biased by evaluator subjectivity. A monitoring test should be able to assess the change in spasticity during therapy, but also to assess the functional effects of interventions.

The modified Ashworth scale (MAS) is the most widely used method for assessing muscle spasticity in clinical practice and research as described earlier.

Grade	Description
0	No increase in muscle tone.
1	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the ROM when the affected part (S) is moved in flexion or extension.
1+	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM
2	More marked increase in muscle tone through most of the ROM, But affected part(s) easily moved.
3	Considerable increase in muscle tone, passive movement difficult
4	Affected part(s) rigid in flexion or extension.

Modified Ashworth Scale for Grading Spasticity [10]

The MAS was recorded in patients with post-stroke lower limb muscle spasticity and correlated with the excitability of the α motor neurons. The latter was evaluated by measuring the latency of the Hoffmann reflex (H Reflex) and the ratio of the amplitude of the maximum H reflex (H_{max}).to that of the compound motor action potential of the soleus muscle (M_{max}).[11]

The H reflex was described by Paul Hoffmann in 1918, hence call H- Reflex. Its general characteristics has been reviewed in detail by Mayer and Maswdsley (1965).

The **H-reflex** (or Hoffmann reflex) is a reflectory reaction of muscles after electrical stimulation of sensory fibers (Ia afferents stemming from muscle spindles) in their innervating nerves (for example those located behind the knee)

The reflex arc of H-reflex includes:

1. Large fast conducting group Ia fibers
2. Spinal cord where afferent fibers synapse with α Motor neurons
3. Efferent motor fibers supplying the muscle

H-reflex does not include muscle spindle activation but rest of the arc is similar to tendon reflex produced by muscle stretch; therefore, there is a high correlation between Achilles reflex and soleus H-reflex

The exclusively monosynaptic nature of either H or tendon reflex has however been questioned.

H-reflex is facilitated by submaximal stimulation and inhibited by stronger stimulation. The inhibition of H-reflex on stronger stimulation is attributed to collision of orthodromic impulses by antidromic conduction in motor axons. This occurs in efferent pathway because of faster conduction in the afferent(Ia) fibers. Besides the collision of impulses, there are number of other inhibitory mechanisms such as:

1. Renshaw cell inhibition
2. Supraspinal inhibition
3. Inhibition by adjacent motor neurons

H-reflex can be enhanced by the maneuvers, which increase motor neuron pool excitability such as muscle contraction. Post-tetanic potentiation can elicit H-reflex when this is otherwise not recordable.[12]

The soleus is the most commonly used muscle for H-reflex studies in humans, while limited comparable data have been produced from the gastrocnemii muscles.[13]

The H-reflex test is performed using an electric stimulator, elicited by submaximal stimulation which gives usually a square-wave current of short duration and small amplitude (highest stimulations might involve alpha fibers, causing a M-wave, compromising the results), and an EMG set, to record the muscle response. That response is usually a clear wave, called H-wave, 50ms after the stimulus, not to be confused with M-wave at 25-30ms, which might appear in too intense stimulations.

The M response (Magladery and Mc-Dougal, 1950) is simply the response of a muscle as recorded electromyographically (EMG) to stimulation of the motor nerve. However, W.B.Matthews used the maximum M response as an indication that the electrodes had not moved during the experiment and also to exclude neuromuscular block as a cause of relief of spasticity.[15]

If the purpose of the test is to compare performance from different subjects, H-reflex should be used. In that case, in fact, latencies(ms) and amplitudes(mV) of H-wave can be compared.[14]

H-reflex is used to assess fitness of astronauts. H-reflex was the first medical experiment completed on the international space station.[13]

The H-reflex is monosynaptic and is generally considered to be the electric analogue of the stretch reflex. It is evoked by electric stimulation of lowest threshold muscle spindle afferents in the tibial nerve at the knee at a latency of about 30ms. At higher intensity stimulation, an M response is recorded at a shorter latency of about 5ms. The ratio of maximal H-reflex amplitude to maximal M-response amplitude is thought to represent the number of motoneurons recruited through the monosynaptic reflex as a proportion of the motoneuron pool.

Angel and Hoffmann found an increase in the H_{max}:M_{max} ratio in stroke patients in static conditions. Increased H_{max}:M_{max} ratio was also found in static spinal cord-injured patients. Therefore, they explored the possibility of using the ratio between the maximum H reflex and the maximum M response as a simple and precise method of measurement.[16]

There is general agreement that the major component is a monosynaptic reflex elicited by submaximal stimulation of a mixed motor and sensory nerve and in a normal adult, it can usually only be elicited in the triceps surae from stimulation of the medial popliteal nerve. In many respects the reflex resembles the ankle jerk with the important difference that the peripheral receptor organs are not involved, fast-conducting afferent fibers being stimulated directly.

The H-reflex is usually increased in a spastic lower limb while the M response is unaffected. Both responses can be elicited using the same stimulating and recording electrodes and for purposes of comparison, it is essential that they should be so.[14]

The primary difference between the H-reflex and the spinal stretch reflex is that the H-reflex bypasses the muscle spindle and therefore, is a valuable tool in assessing modulation of monosynaptic reflex activity in the spinal cord. The H-reflex is an estimate of alpha motoneuron excitability when presynaptic inhibition and intrinsic excitability of the alpha motoneurons remain constant.[17]

M wave reflects the excitability of muscle membrane related to the change in force during muscle fatigue and the amplitude ratio of H/M has been considered as the index of a relative excitability of alpha motoneuron pool.

The degree of the motor neuron excitability, as measured by the Hoffmann reflex (H reflex), has been shown to correlate with the clinically observed increase in the myotatic stretch reflex activity and to discriminate between spasticity and normal muscle tone. Both the latency and ratio of the maximum amplitude of the H-reflex to that of the motor response (that is, the compound motor action potential) of the soleus muscle (H_{max}:M_{max} ratio) have been shown to be reliable measures of the motor neuron pool excitability.[11]

Group passive movements of joints, e.g. flexion and extension of hip, knee and ankle, are preferable, but a very high standard performance on the part of the physiotherapist is required to obtain results.[18]

CPM device was created by Robert B. Salter, M.D. and along with help from engineer John Saringer, a device was created in 1978. Basic research and clinical studies reported by Salter have demonstrated the effectiveness of CPM in a number of areas.[19]

CPM device covers a range of mechanical devices designed to move the patient's joint or extremity without the use of the patient's muscles through a prescribed range of motion over extended periods of time.

Typical CPM devices consist of a limb support resembling a splint or brace, a motor drive and a control unit.

The limb requiring the CPM is strapped into the device's support. The range of motion parameters, speed, cycling time and duration are then set. The rate of motion is determined; usually 1 cycle/45sec or 2min is well tolerated. Most CPM devices have mechanical safety releases, resistance sensors and/or automatic shut-offs for safety purposes. The most advanced CPM devices use microprocessors and load cells to measure resistance and automatically adjust parameters.

Patients on CPM devices should be monitored for problems with limb positioning and interference with device operation. For instance, bedsheets may become tangled in the device, or the patient's limb may become trapped, resulting in injury.

Patients with urinary incontinence should be monitored carefully during CPM, because body fluids are an electrical hazard. It is important that patients learn to use and develop motor control of the ROM as motion improves. All patients on CPM devices should be monitored for unnecessary discomfort, pain and chafting.

CPM devices are low maintenance equipment since they are designed for continuous use and to be adjustable, easily controlled, versatile and portable. Some are battery operated. They do, however, require frequent checks for wear of moving parts and malfunctioning.[20]

Rehabilitation after stroke remains a challenge. Passive movement is commonly used to help the stroke survivor recover from physiologic impairments caused by the lesion of the central nervous system. When spastic hypertonia is identified, joint mobilizations and muscle stretching are considered an integral part of the patient's daily management.[21] CPM is one of them, it is in contrast to intermittent passive motion, is motion, that is uninterrupted for extended periods of time. It is usually applied by a mechanical device that moves a desired joint continuously through a controlled ROM without patient effort. The motion is passive, so that muscle fatigue does not interfere with the motion.[19]

Passive movements are produced by an external force during muscular inactivity or when muscular activity is voluntarily reduced as much as possible to permit movement. The rhythm of continued passive movements can have a soothing effect and induce further relaxation and sleep. The movement is made imperceptibly and progressively slower as the patient relaxes.[18]

Study shows that continuous passive movement decreases the reflex excitability, spasticity or hypertonia.

One study shows that passive movements induced a decrease of spastic hypertonia in stroke patients through a combination of reflexive and mechanical factors. Habituation of reflex activity to repeated stretch may result from a decrease in synaptic transmission caused by inactivation of presynaptic calcium channels. The role of these mechanisms is velocity dependent and differs for flexion and extension movements. [21]

The present study therefore sought to study the effect of CPM on spasticity.

AIMS AND OBJECTIVES:

1. To study the H-reflex and H/M ratio in spastic hemiplegics both on the affected and normal side.
2. To study the association of H/M ratio to clinical grades by MAS.
3. To study the effect of continuous passive motion on H-reflex, H/M ratio and spasticity.

2. REVIEW OF LITERATURE

A M O Bakheit et al, recorded the MAS in patients with post-stroke lower limb muscle spasticity and correlated with the excitability of the alpha motor neurons. The latter was evaluated by measuring the latency of the Hoffmann reflex(H-reflex) and the ratio of the amplitude of the maximum H reflex (H_{max}) to that of the compound motor action potential of the soleus muscle (M_{max}). Data on 24 randomly recruited patients were analysed. Patients were divided into two groups according to their MAS score: 14 had a MAS score of 1 (group A) and 10 scored 2 (group B). The two groups were comparable with respect to age and sex, but in group A there was a longer period since the stroke. The H-reflex latency was reduced and the H_{max}:M_{max} ratio was increased in both the groups. The H_{max}:M_{max} ratio values were higher for group B but the differences were not statistically significant. There is a relation between the MAS scores and alpha motor neurone excitability, although it is not linear. This suggests that the MAS measures muscle hypertonia rather than spasticity.[11]

J T C Hsieh et al, studied to assess published psychometric evidence for SCI spasticity outcome measures and also identified the influence of spasticity on function to understand treatment effects and guide service delivery. They concluded that since spasticity is multidimensional, focusing on one or two spasticity outcome measures can misrepresent the extent and influence of spasticity on SCI patients. Different scales measure different aspects of spasticity and individual tools correlate weekly with each other. Spasticity may be better measured with an appropriate battery of tests, including the Ashworth scale(AS) or modified Ashworth Scale(MAS), along with Penn Spasm Frequency Scale(PSFS). These tools would benefit from further reliability and responsiveness testing. Tools that assess the influence of spasticity on patient activities, participation and quality of life are important but lacking. [23]

In their study, showed significant benefits for the improvement of motor function in paralyzed arms of patients with acute or chronic stroke who are trained with interactive robotic techniques. Bruce T. Volpe reported that these data are consistent with a growing focus on impairment training that may occur a long time after the acute event. A randomized trial that matches robot training sessions with physical therapy sessions to test whether one or the other or both are effective at improving motor outcome. Simpler machines that only move the arm passively demonstrate benefits for shoulder joint stability. Currently, the passive motion device is used to reduce tone and at times, for pain management. It may be that early training with passive motion devices maintains the shoulder joint so that additional. More aggressive interactive therapy can proceed.[22]

In their article, Ralph spira studied a group of 21 adolescents suffering from cerebral palsy, who presented spasticity of the lower limbs as the main motor disorder, were investigated by means of establishing the H_{max}:M_{max} ratio in the triceps surae muscles of both legs. The results showed considerable increases of the ratio in all cases studied. This is taken to indicate an increase clinical spasticity. In cases where the degree of clinical spasticity differed grossly from one leg to another, the H/M ratio invariably showed a dinstinctly higher value in the more spastic limb. When investigations were repeated after a period of several months of controlled activities directed to the lower limbs, they showed a trend to reduction of the H/M ratio. These results suggest that H-reflex investigations might be used for estimation of the degree of spasticity in affected children. This method might also help in objectively evaluating the response to various therapeutic and rehabilitative measures undertaken in these patients.[24]

Higashi T et al, studied the motoneuron pool excitability of hemiplegic patients assessing recovery stages by using H-reflex and M response. They used H_{slp}/M_{slp} (developmental slope of H-reflex and M wave) and Brunnstrom stages. Results showed that H_{slp}/M_{slp} had better predictive value than conventional indicators of motoneuron pool excitability. H_{slp}/M_{slp} appeared to be a better match for the bell-shaped pattern of the Brunnstrom stages. H_{slp}/M_{slp} is the preferred index for evaluating the motoneuron pool excitability of the spastic side of hemiplegic patients.[25]

Leis AA, Kronenberg MF, Stetkarore I et al, in patients with CNS (central nervous systems) lesions with UMN signs-reflex may be abnormally widespread and can be elicited in muscles in which, it is not normally elicited such as tibialis anterior and small muscles of the hand. H/M ratio tends to increase in these patients. H-reflex is depressed during spinal shock and cataplexy.[26]

UK Misra and J. Kalita stated that H-reflex studies have been employed for monitoring the excitability of AHC pool in different CNS disorders such as stroke.[12]

In their study they aim to investigate the changes of the H-reflex, in soleus during hip and knee joint movements in stroke patients. Tanabe S. et al, carried out an experiment on five stroke patients with spastic hemiplegia (2 males and 3 females, 48-71 years old) soleus H-reflexes were measured 200 times, for each joint movement speed stimulus was given at random intervals (4-5 seconds) during the joint movement. Two movement speeds were used to investigate the effects of movement speed. For both fast and slow movements, the amplitude of the soleus H-reflex decreased in the middle flexion phase. In contrast, the amplitude of the soleus H-reflex increased in the middle extension phase. For the fast movements, the soleus H-reflex was smaller in only a smaller in only a small angle range during the flexion and extension phase in comparison to the slow movement. The soleus H-reflex during during the flexion phase was significantly smaller than during the extension phase at almost all angles for both speeds. The soleus H-reflex for both speeds was smaller than the soleus H-reflex at rest for the whole angle range.[27]

One study aimed to elucidate whether hip and knee joint movement modulates soleus H-reflex, the authors, Tanabe S et al., measured the soleus H-reflex for a very fine angle during movement. Eight healthy subjects participated. The hip and knee joints were passively flexed and extended ranging from 0 degress to 120 degrees simultaneously. In a flexion phase,

H-reflex decreased. It decreased more during fast movement. In the extension phase H-reflex increased markedly. Author found that the stretch of the muscles around the knee and hip joints caused the changes in soleus H-reflex.[28]

The authors studied the effects of therapeutic passive exercise of hip and knee joints on the soleus H-reflex, Sugawara suggested the results that in both exercises, H/M decreased as flexion progressed with fast or slow movement. The quadriceps femoris inhibited soleus muscle muscle alpha-motoneurons in both experiments. H/M ratio increased on initiation of extension in the two-joint exercise. In the single-joint exercise, inhibition was retained through the extension phase during both fast and slow movements. Hamstring effects were strong during extension of the leg. In both exercises, the H/M for fast movement was lower than for slow movement at all angles, indicative that H/M ratio decreases with fast movement irrespective of the effects produced by the hip joint.[29]

Delwaide PJ et al, Studied in normal individuals that amplitude of H-reflex decreases on vibratory stimulation. In a patient with UMNL the vibratory inhibition is less than normal possibly due to presynaptic inhibition.[30]

Ya ju chang fang CY et al studied to examine the effect of ankle continuous passive motion on the reflex excitability and overall hypertonia of calf group comprised eight individuals with chronic SCI and without physical disabilities. The SCI group comprised eight individuals with chronic complete spinal cord injury. The controlled group comprised eight individuals with chronic complete spinal cord injury. The subject received 60 min of continuous passive motion on the ankle joint. The soleus H-reflex was elicited by tibial nerve stimulation just before, immediately after, and 10 min after CPM(Continuous passive motion). The MAS (Modified ashworth scale) score at the ankle joint was recorded for the SCI group just before and 10 min after CPM therapy. After 60 min of CPM of the ankle joint, the H-reflex amplitude at the soleus muscle was depressed in individuals with and without SCI. This depression persisted up to 10 min after CPM only in individuals without SCI.

In individuals with SCI, the median of MAS scores decreased from 2 to 1.25 after CPM. 60 min of CPM of the ankle joint decreased reflex excitability and overall hypertonia in people with or without SCI. the depression of overall hypertonia persisted longer than the reflex excitability in people with SCI [31]

Okada et al., states that the therapeutic exercise Machine (TEM) is a newly developed exercise machine which aims to help stroke patients to recover their walk-in function using kinetic therapy on the lower extremities. TEM has a function to perform compliant motion as is normally performed by a therapist's hand. Therefore, TEM can accomplish the evaluation of a therapist's technique that is not available for conventional machines. The effects of complaint motion against an impulsive load are shown, and the short term effects of a continuous passive range of motion exercise (CPROM-E) in spastic patients and normal subjects are observed. In the normal subjects, the peak torque of the hip decreased steadily and significantly by about 5 % after CPROME-E, although the peak amplitude of the surface integrated- electromyogram (I-EMG) was not always reduced. In the spastic patients, the peak torque of the hip decreased significantly, by about 35%, and the peak amplitudes of I-EMG also decreased significantly. These results suggest the TEM could make relax spasticity and reduce the level of I-EMG. [32]

He studied to quantify changes in spastic hypertonia during repeated passive isokinetic knee movements in stroke patients and assess the role of muscle activity. Godelieve Nuyens, found that passive movements of the knee induced a decrease of spastic hypertonia in stroke patients through a combination of reflexive and mechanical factors. The role of these mechanisms is velocity dependent and differs for flexion and extension movements. [21]

Daniel Lynch, Mark Ferraro et al, did a pilot study of patients with a first stroke and hemiparesis, sought to determine whether treatment of the upper limb with CPM that was device delivered would alter impairment, disability or the associated adverse symptoms of shoulder joint instability, pain and tone. Patients were randomly assigned to receive daily CPM treatments or participate in self ROM groups under the supervision of an occupational therapist. All Patients received standard daily post stroke therapy for 3.5hrs per day. 32 were completed the study and were evaluated using standardized measures for motor impairment (Fugl Mayer, Motor status scale and medical research council motor power), adverse symptoms (gleno-humeral stability, pain and tone) and disability (FIM). Concluded that Device-delivered CPROM may offer and enhanced benefit for some adverse symptom reduction in the hemiplegic arm after stroke over traditional self ROM exercise. [33]

While studying , pinelli and valle (1960) had found the relationship between the height of the two responses at different stimulus strengths and between different individuals to be so variable that they rejected the H/M ratio as a measures of spasticity. [34]

Angel and Hofmann (1963) hoped that by using the ratio between the height of the maximum H reflex and that of the maximum M response, the effect of such factors as electrode placement, thickness of skin and, in particular, stimulus strength, might be excluded. They did not, in fact, demonstrate that with different electrode placements a constant ratio could be obtained during the period of the experiment, nor did they clearly establish that an investigation of purely monosynaptic reflex stimulation of the motor pool could provide a valid index of the complex phenomenon of spasticity. Nevertheless, the method was simple and ingenious and might well have proved a notable advance.[16]

Little and Halar recorded how increased H-response amplitude and H_{max}:M_{max} ratio developed along with spasticity in 6 SCI patients during months 1 to 3 postinjury.[35]

In his study, Hugon M postulated one neurophysiologic mechanism to explain hyperactive muscle responses to stretch is increased central synaptic excitability (CSE) from primary (IA) Spindle afferents to motoneurons; another is muscle spindle sensitivity. The soleus H-reflex has previously been used to investigate the CSE of spinal motoneurons in normal subjects and in patients with diseases such as stroke and spinal cord injury (SCI) under static (Iying, sitting) conditions.[36]

Keenan et al suggested that poor motor control rather than spasticity was the major factor affecting ambulation and balance of persons with stroke. In the present investigation the H-reflex is used to describe the changes in excitability at the central synapse between IA spindle afferents and motoneurons of persons with spastic hemiplegia before and after passive movement on CPM devic.[37]

Authors studied that increased H_{max} :M_{max} ratio in community walkers post-stroke without increase in ankle plantarflexion during walking and concluded that individuals with community level walking ability after stroke have significantly less repeatability of ankle joint movement than controls at both midswing and midstance. Simultaneously soleus H_{max} and M_{max} ratio in community walkers post-stroke without increase in ankle plantarflexion during walking and concluded that individuals with community level walking ability after stroke have significantly less repeatability of ankle joint movement than controls at both midswing and midstance. Simultaneous soleus H_{Max} and M_{Max} testing showed a significant reduction in the H_{max} and H_{max} and H_{max} : M_{max} ratio at midswing in controls only. This inhibition at midswing was lost by the IWS group without significant increase in H_{max} suggesting that central synaptic excitability was within normal range, and possibly accounting for the absence of excessive ankle plantarflexion during walking in the IWSS group with community level walking ability. [14]

M. Garrett, T. Kerr et al studied phase-dependent inhibition of H-reflexes during walking in humans is independent of reduction in knee angular velocity. This investigation showed that restricting knee movement during the swing phase of walking was not accompanied by loss of H-reflex attenuation. A highly significant reduction in the soleus Hmax/Mmax ratio at midswing compared with midstance during treadmill walking under both unrestricted and restricted knee conditions demonstrated the phasic modulation of reflexes.[38]

He studied the effect on spasticity after performance of dynamic-repeated-passive ankle joint motion exercise in chronic stroke patients and Chin-Li Wu concluded that 15 minutes of dynamic-repeated-passive ankle joint motion exercise with weight loading in the standing position by this simple constant-speed machine is effective in reducing ankle spasticity and improving ambulatory ability. In addition, subjective satisfaction with the short-term therapeutic effect was mainly good (ranging from acceptable to very good). [39]

Douglas Watt investigated the effects of altered gravity on spinal cord excitability by using H-reflex, summarized that prolonged weightlessness results in a loss of muscle strength, muscle volume and bone density, particularly in the legs. Confirm that spinal cord excitability is gradually diminished during prolonged exposure to microgravity. Spinal cord excitability appears to decrease after approximately 5 days in space. This would make inflight exercise less efficient and/or less effective at maintaining muscle mass and bone calcification.[40]

Knikou M et al studied the effects of changes in hip joint angle on H-reflex excitability in humans, examined the amplitude modulation of the soleus H-reflex during controlled variations of the hip joint angle in 21 healthy adult human subjects and provided the evidence for the existence of a spinal mechanism, determined principally by the hip joint angle, which promotes switching between inhibitory and facilitatory pathways during hip flexion and extension.[41]

The H-reflex for methodologic considerations and applications for use in sports medicine and athletic training research was studied by Riann M. Palmieri et al and concluded that when eliciting H-reflex measures, great care must be taken to obtain valid and reliable results. If measured properly, the H-reflex can provide information regarding neural function after injury.[17]

Richard W. Bohannon and Melissa B. Smith studied the Interrater reliability of a modified Ashworth scale of muscle spasticity and concluded that from the two raters, who performed manual tests of elbow flexor muscle spasticity, agreed on 86.7% of their ratings of the level of spasticity. Their ratings, which were based on a modified Ashworth scale, were significantly correlated($p<.001$). the reliability and usefulness of a modified Ashworth scale merit further clinical investigations.[10]

In his study, Matthews C. Hoch stated that intersession reliability of H/M ratio is greater than the H-reflex at a percentage of Mmax and found that an interclass correlation coefficient (ICC 2,1) has strong intersession reliability for both measurement techniques. Assessing motoneuron activation using maximal amplitude ratios demonstrated a robust ICC of 0.979 compared to an ICC of 0.882 when Hoffmann reflex was assessed at 20% of the maximal muscle response.[42]

Yeh, C.Y., Tsai, K.H.; studied the effects of prolonged muscle stretch on spasticity by an assessment system and concluded that the prolonged muscle stretch(PMS) has been proven to be an effective approach to reduce excessive muscle tone.[43]

The soleus H-reflex and motor unit number estimation after tibial nerve block and neurotomy in patients with spastic equinus foot was studied by T. Deltombe et al and concluded that the diagnostic nerve block predicts the spasticity and gait improvement, which is expected after neurotomy. The clinical improvement was similar after block and neurotomy. Nerve block is associated with a 50% decrease in the soleus Hmax/Mmax ratio and soleus motor unit number estimation. The median 80% neurotomy is associated with an 80% decrease in the soleus Hmax/Mmax ratio and soleus motor unit number estimation.[44]

Harburn KL, Hill KM et al did a pilot study on spasticity measurement in stroke. The ability to objectively measure spasticity, related to cerebral stroke, is important in the rehabilitation therapies since many therapeutic modalities have been developed over the years to reduce spasticity. The technique combined biomechanical and electrophysiological measures to investigate a homogenous stroke sample. It incorporated the H-reflex in soleus, during passive ankle movements, as a measure of faulty neural inhibition. The final objective in this line of research is to develop a valid, reliable and sensitive spasticity measurement system that could be used to judge the efficacy of physical neurorehabilitation treatments currently employed to reduce spasticity following stroke.[45]

Vanessa Pelegrino Minutoli et al, studied the effect of isokinetic continuous passive mobilization in spastic hemiplegia. Five patients of both sexes between 40-55 years old with a history of CVA and accompanying spasticity were enrolled in the study. All patients presented degree 2 spastic extensor muscles of the knee joint at the modified Ashworth scale and degrees 0,1 and 1+ flexor muscles at the same scale. All the individuals were submitted to continuous passive mobilization by an isokinetic dynamometer at speeds of 1200/second and 1800/sec with 30 repetitions of each. The results showed a significant reduction of passive resistance.[49]

Vanessa A B Scholts, Anita Belen et al, reviewed the instruments used for the clinical assessment of spasticity in children with cerebral palsy, and evaluate their compliance with the concept of spasticity, defined as a velocity dependent increase in muscle tone to passive stretch and concluded that the instruments that are most frequently used for the clinical assessment of spasticity in children with CP do not comply with the concept of spasticity. Only the original “Tardieu scale” is a suitable instrument to measure spasticity.[50]

3. METHODOLOGY

The study proposal was approved by Human Research Ethical Committee of H M Patel Center for Medical Care and Education, Karamsad.

The study design was Interventional Cross-sectional.

The study setting- The adult NPT (neurophysiotherapy) department of K. M. Patel institute of Physiotherapy, Shree Krishna hospital, Karamsad, some patients were recruited from community provided they have documents confirming spastic hemiplegia.

The study period was between July’09 to Jan’10.

Inclusion criteria:

1. Known cases of spastic hemiplegics, with or without morbidity

2. Age group: 35years to 60 years (including both males and females)

Exclusion criteria:

1. Age group: <35years and >60 years
2. Cognitive impairment
3. Any musculoskeletal problems
4. Other neuromuscular disorders and problems
5. Peripheral vascular diseases
6. Cardiovascular respiratory system disorders-pacemaker heart

Materials:

1. EMG-NCS unit with accessories, RMS-II of recorders and medicare systems inc. India
2. CPM device, Nisha electrocare
3. Plinths, wooden stools, pillows, steppers (for the participants to ascend and descend from the plinth), measuring tape, marker, pencil, sharpner, scale and eraser

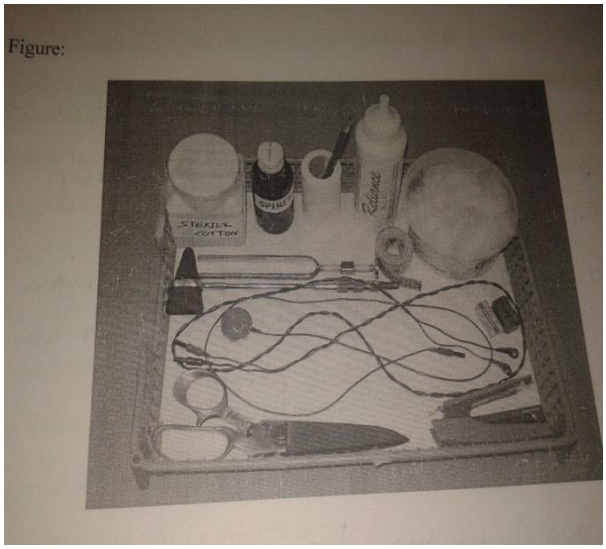


Figure: tray with materials

Methodology:

Participants were recruited as per subject recruitment procedure bearing in mind the inclusive and exclusive criteria.

The total study sample was 21 participants- 13 males and 8 females.

All the participants were the spastic hemiplegic patients in the study group and the normal lower limb served as the control.

Written informed consent was obtained from every participant after explaining the details of various non-invasive tests, which were to be performed on their lower limbs.

The participants were evaluated in detail for neurological examination as in format (Annexure 3).

The following outcome measures were utilized in the study before and after conducting the experiment;

1. MAS (Modified Ashworth Scale)
2. Soleus H-reflex
3. Soleus H_{max}:M_{max} ratio

The modified Ashworth scale:

Spasticity of the following group of muscles was assessed clinically by the MAS. As shown in annexure 4, the scale measures the degree of muscle hypertonia on a six point scale, ranging from 0 to 4. Resistance to passive muscle stretch was measured at the hip, knee and ankle joint of the hemiplegic lower limb with the participant lying flat in the supine position, following flexion and extension of the limb. The MAS was graded before and after intervention(passive movements on CPM device).

Soleus H-reflex studies were performed using RMS-II of recorders and medicare systems inc., India.

All the subjects were positioned prone (except for two who were seen in side position as they were not comfortable lying prone) with leg firmly supported on pillow and the feet hanged freely at the edge of the plinth, for H-reflex studies.

Skin was prepared and scrubbed before placing the recording electrodes. The popliteal crease was marked for stimulating electrode, cathode was kept proximal to anode to avoid anodal block, applying the recording cathodal disk electrode (1cm by 1cm) at the distal edge (motor point) of calf muscle and the anodal disk electrode (1cm by 1cm) on achilles tendon, ground electrode (3cm by 3cm) was positioned in between the stimulating and recording cathodal disk electrode.

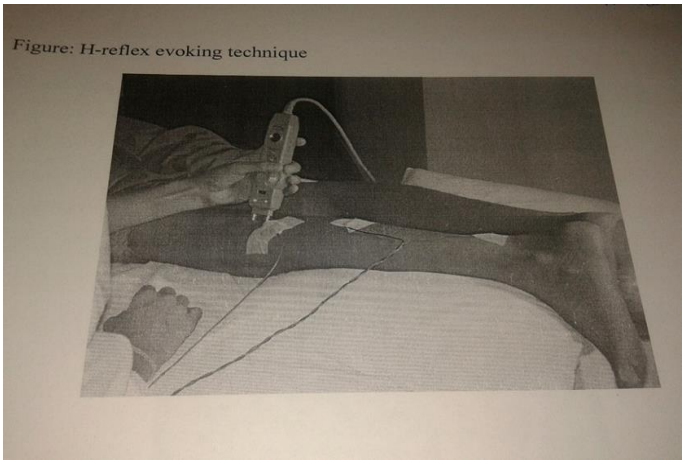


Figure: H-reflex evoking technique

Low frequency filter was set at 20Hz and high frequency filter at 10KHz. A square wave pulse of 1ms duration is used for preferential stimulation of large sensory fibers. Stimuli below 0.1ms duration may activate motor fibers rather than sensory. Stimuli frequency should not exceed 1 in 5 sec to exclude any effect of prior stimulus.

Submaximal intensity was used to elicit H reflex with sweep at 15ms/D. latency was measured at initial negative deflection and amplitude was measured from peak to peak amplitude.

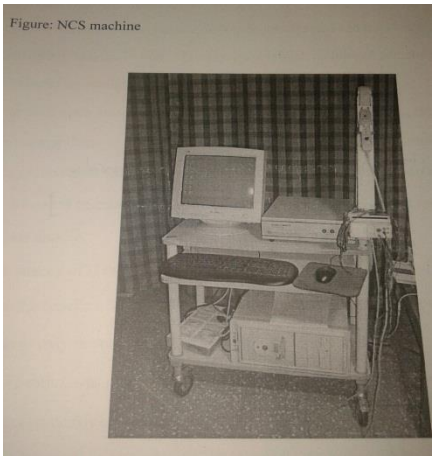


Figure: NCS machine

Elicitation of an H response, at a latency of about 30ms, without an M response identified the optimal stimulating position. At this strength a small M response may also be present. Attention to M response may help in monitoring the

strength of stimulation. At least five H responses should be studied for analysis. By increasing the stimulus strength to supramaximal, maximal M response can be recorded and three M responses are measured for analysis. The maximal M response was determined by increasing stimulus intensity to the point at which no further increase in direct motor response was obtained and the H response was abolished. Maximal M and H responses obtained, monitored and checked for similarity in configuration.

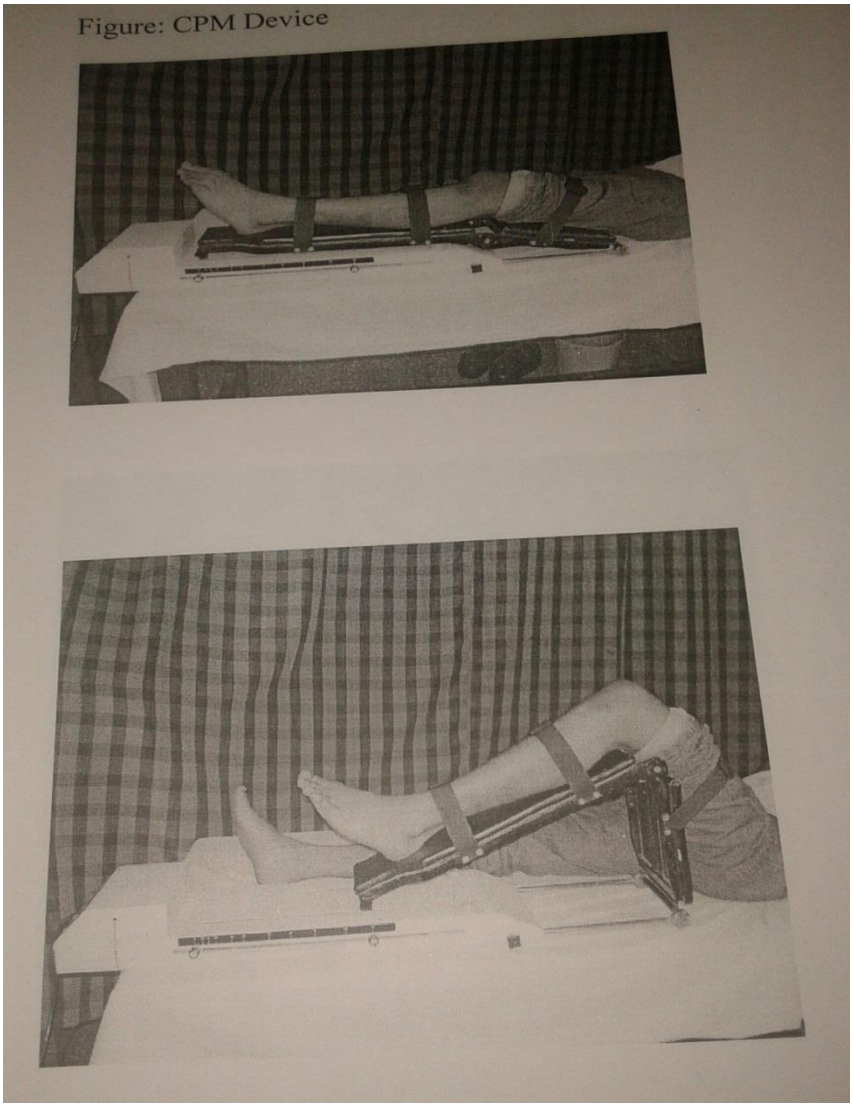
For each participant calculation of an amplitude ratio (H_{max}:M_{max} ratio) defined as the amplitude of the maximal H response as a ratio of the maximal M response. This ratio abolished differences arising from intrasubject variation in electric resistance. The maximum M response represents the total motor response of the soleus muscle.

The latency of H reflex is measured from the stimulus artefact to the first deflection from the baseline. The amplitude is measured from base to peak of the negative phase or may be measured from peak to peak.[14]

The H-reflex was elicited on intact and on the affected lower limb before continuous passive motion on CPM device.

After the H-reflex elicitation, participant was instructed to lie down in supine, CPM device was placed under the affected lower limb, thigh is placed over the thigh piece, leg over the leg piece, ankle and foot was supported without ankle supporter as our CPM device has no ankle supporter. CPM was applied for 20min without rest in between. Any discomfort from the patient was kept in mind. After the passive movement on CPM device, the H-reflex study was elicited again to study the effect of passive movement given on CPM device. Only the affected lower limb was studied for H-reflex after passive movement on CPM device.

Figure: CPM Device



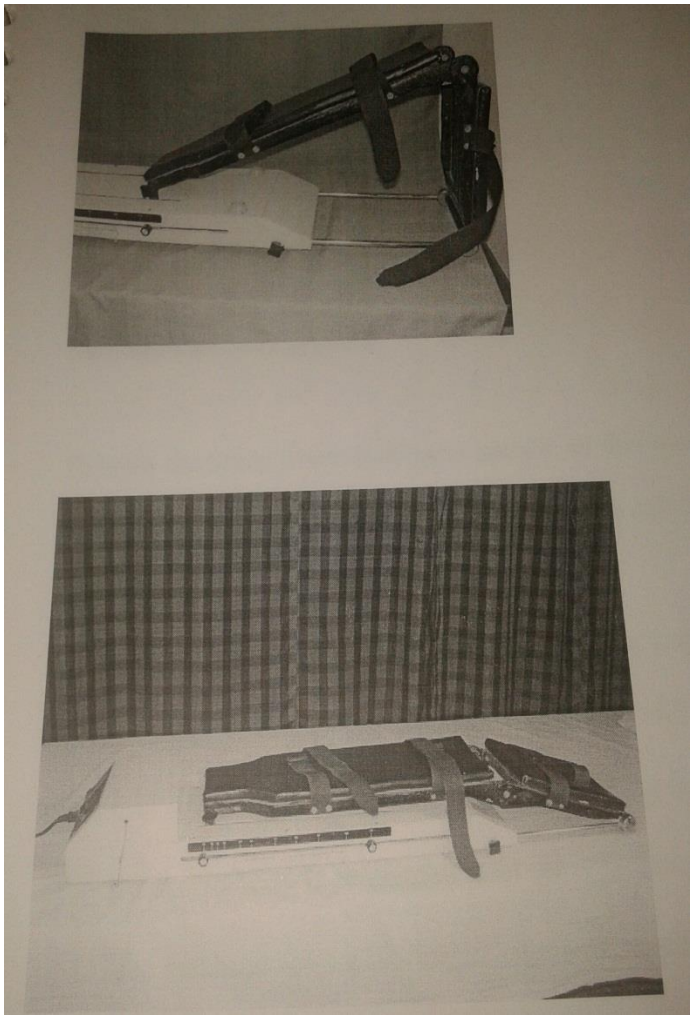


Figure: CPM device

To know the rate at which continuous passive motion was given, we calculated the velocity of CPM delivered by the CPM device. We calculated both the linear and angular velocity of CPM device.

Calculation of linear velocity: the machine covers total 58.0cm of distance in linear motion (sliding uo-29.0cm and sliding down-29.0cm). it completes one repetition in 111 sec (sliding up-59.0sec and sliding down-52.0sec) between 00 to 1100. Linear velocity is calculated in m/sec. (58.0cm=0.58m)

In 111 sec machine covers 0.58m, in 1 sec how much? 0.58m/111sec=0.005m/sec.

The machine delivers passive movement at 0.005m/sec.

We calculated individual angular velocity for sliding up and down of CPM device.

$\delta\theta = 1000$ δt=59.0sec and 52.0sec

in sliding up CPM device takes 59.0sec and in sliding down CPM device takes 52.0sec.

Angular velocity= $\delta\theta/\delta t$=1000/52.0sec=1.910/sec for sliding down, in one sec the device delivers 1.910

Angular velocity= $\delta\theta/\ \delta t$=1000/59.0sec=1.690/sec for sliding up, in one sec the device delivers 1.690

In the present study, the CPM device were calculated to delivered repetitions for passive movements in 20 minutes were 12.

For statistical purposes the values of latency of H-reflex, amplitude of H and M wave i.e. H/M ratio and scored MAS before and after passive movements on CPM device of the affected lower limb were studied and compared with the intact lower limb.

4. DATA ANALYSIS

The data were fed into the computer in Microsoft Excel sheet. For statistical analysis, SPSS (Statistical Package of Social Science) software was used.

Frequency/cross tables were made to give the basic idea of the data. Chi-square test was applied to test the significant association between two attributes.

Descriptive statistics (Mean, Standard Deviation-SD, and Standard Error-SE) were calculated to get baseline idea of the collected data. Student's test was used to compare mean value of variable between two lower limbs. We have put 5% as level of significance.

5. RESULTS

The total number of participants in the present study were 23, of which 2 were excluded from the study as the H-reflex was unrecordable.

Out of 21 participants, 13 (62%) were males and 8(38%) were females, 14(66%) were left hemiplegics and 7(34%) were right hemiplegics. 4(19%) were Diabetics (table 1).

MAS grades were used to grade the spasticity clinically of the following muscle groups, hip extensors, knee extensors and ankle planter flexors, before and after the experiment i.e. passive movements on CPM device as seen in table no 2,3,4.

The hip extensors group(table 2) overall(76%) showed minimum spasticity ranging 0 to 1 which did not show any change, however, 23% showed grade 1+ spasticity which post CPM declined to 1.

Statistical analysis was not applicable to this group.

In the other groups i.e. knee extensors and ankle planter flexors (ref table 3,4), there was, statistically significant decrease in spasticity, MAS specifically of the participants with the grades 1+ and 2, which post CPM reduced to 1 and 1+ respectively.

The statistical significance for the two groups knee extensors and ankle planter flexors as shown is p-value 0.001 and 0.004 respectively.

H-reflex study:

The H-reflex was studied before and after passive movements on CPM device. The following parameters of H-reflex were studied and analysed.

H-amplitude, H-latency, M-amplitude, M-latency and HM ratio.

Of these significant changes were noted only in H-amplitude and HM ratio. The H-amplitude of the hemiplegic side before the experiment was mean 6.00+_4.81, SE 1.05 and after the experiment was 4.18+_3.37, SE 0.73. p-value 0.001

The HM ratio before the experiment was 0.51+_0.5, SE 0.10 and after the experiment was mean 0.28+_0.21, SE 0.04. p-value 0.019(table 5).

Comparison of MAS and HM ratio:

Table 6 shows the values of spasticity grade by MAS and HM ratio pre CPM, ANOVA as seen did not show statistical significant association of the two. However, there was stronger relationship between the spasticity by MAS and H/M ratio post CPM (p-value 0.001) rather than pre CPM (p-value 0.17)

There was no change seen in the H-latency and M-wave parameters (amplitude & latency) between affected and intact limb.

6. DISCUSSION

Clinical evidence has shown that regular mobilizations help prevent contractures and can reduce the severity of spastic tone for several hours. Most concepts specifically developed for the rehabilitation of stroke patients, such as neurodevelopmental therapy, include passive movement of limbs as a substantial technique to reduce muscle hypertonia.

Clinically, we assess spasticity by resistance. Modified Ashworth Scale(MAS), most widely used in clinical and research practice was used in our study. The grade of spasticity by MAS were assigned in this study following flexion and extension of the limb.

MAS grades clinically of the following muscle groups: Hip extensors, knee extensors and ankle planterflexors, before and after the experiment are as shown in table 2,3,4. The statistically significant drop in MAS grade (P-Value 0.001) in knee extensors and in ankle plantarflexors (p-value 0.004) was seen post experiment

Ya-Ju chang et al, examined the effect of ankle CPM on the reflex excitability and overall hypertonia of calf muscle in individuals with chronic spinal cord injury and without physical disabilities. They used MAS as measure for spasticity and concluded that MAS was decreased after CPM and the depression of overall hypertonia persisted longer than the reflex excitability in people with spinal cord injury. [31]

Several studies support the mechanism for reduction of spasticity as a response to repeated passive movements. Habituation of reflex activity to repeated stretch may result from a decrease in synaptic transmission caused by inactivation of presynaptic calcium channels. [21.,31,33,39] However, the exclusive role of neurophysiological mechanisms as a cause of enhanced response to stretch is debated.

Several researchers have found that increased stretch responses were not necessarily accompanied by enhanced EMG activity. Lamontage et al,[46] found that a decrease in resistance during repeated passive movements without concurrent changes in EMG activity was attributable to thixotrophic characteristics of stretched tissues. The term thixotrophy refers to the property of certain systems becoming less viscous when shaken and then return to the original viscosity, after a period of not being distrurbed. In muscles, thixotrophic changes may occur as a consequence of motion by tearing the cross bridges between the actin and myosin filaments. When agitation ceases, the bridges are reformed and muscle become stiffer again. Other researchers have concluded that mechanical change in the musculotendious unit may also be involved. The finding that hypertonia is not necessarily accompanied by enhanced EMC activity has caused some investigations to question the use of treatment modalities focused on the inhibition of reflexive responses in the treatment of spastic hypertonia.

The debate on the relative role of neurophysiologic and mechanical mechanisms in enhanced response to stretch may be partially attributed to a discrepancy in terminology used. Spasticity has been defined as a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes with exaggerated tendon jerks resulting from hyper excitability of the stretch reflex as one component of UMN syndrome. For clinical assessment however, spasticity has been operationally defined as increased resistance to passive movement. The lack of consensus concerning the pathogenesis and definition of spastic hypertonia shows the complexity of this phenomenon and makes it difficult to quantify.

Chin-Li Wu studied the effect on spasticity after performance of dynamic repeated-passive ankle joint motion exercise in chronic stroke patients and showed that 15 minutes of dynamic repeated passive ankle joint motion exercises with weight loading in the standing position, by this simple constant speed, machine is effective in reducing ankle spasticity and improving ambulatory ability.[39]

Daniel Lynch et al, studied whether treatment of UL with CPM that was device delivered would alter impairment, disability or that associated adverse symptoms of shoulder joint instability, pain and tone. They concluded that device delivered CPROM may offer an enhanced benefit for some adverse symptom reduction in the hemiplegic arm after stroke over traditional self ROM exercise.[33]

Spasticity is common to the injuries of the superior motor neuron of the spinal cortical reticular bulb pathway and results in a neuronal hyperexcitability α & γ moto neurons, medullary interneurons, afferent and efferent pathways the tonus theory is stressed secondary to the loss of the descending inhibitory influences (reticular spinal pathways), as a result of injuries that compromise the cortico-spinal tract. The loss of the descending inhibitory influences will result in an increased excitability of the γ fusimotor neurons and α motoneurons. The main neurotransmitters are GABA and Glycine (inhibitory) and Glutamate (Excitatory). In addition to Noradrenalin, serotonin and neuromodulators such as adenosine and several neuropeptides. [49]

In the present study, CPM device was used to deliver the passive movements in spastic hemiplegic individuals. The passive movements were applied by a mechanical device that moves a desired joint continuously through a controlled ROM without patient effort. The motion is passive, so that muscle fatigue dose not interfere with motion. We analysed the effect of CPM at two velocities, 1.69^0 /sec (for sliding up) respectively. Also, the machine delivered the passive movement at linear velocity of 0.005m/sec. (shown in methodology)

The number of movements performed is a factor that tends to interfere with the results. In the present study, the CPM device were calculated to delivered repetitions for passive movements in 20 minutes were 12.

According to vodovnik et al[52] a test of passive movement with more than 7 repetitions in individuals with spinal cord injury who present spastic hypertonia can induce the onset of a hypotonic condition called accommodation which suggest that the less severe spastic individuals present differences in spasticity behaviour.

To assess the degree of the α motor neuron excitability, we, therefore also measured the Hoffmann reflex(H-reflex) which is correlated with the clinically observed increase in the myotatic stretch reflex activity and is used to discriminate between spasticity and normal muscle tone.

In the study, following CPM, statistically significant drop in H-amplitude (4.18+3/37. SE 0.73, p-value 0.001) and H/M ratio (0.28+0.21, SE 0.04, P-value 0.019) was seen, indicating the effect of continuous passive motion on spasticity.

Ralph spira et al studied spasticity of the lower limb of adolsescents suffering from cerebral palsy. They were investigated by means of establishing the H/M ratio in the triceps surae muscle of both legs. The results suggest that H-reflex investigations might be used for estimation of the degree of spasticity and the method might also help in objectively evaluating the response to various c and rehabilitative measures undertaken in these patients. [24]

Leiss AA et al, suggested that in patients with CNS lesions with UMN signs, H-reflex may be abnormally widespread and can be elicited in muscles in which it is not normally elicited such as tibialis and small muscles of the hand. H/M ratio tends to increase in these patients. H-reflex is depressed during spinal shock and cataplexy.[26]

Uk Misra and J Kalita stated that H-reflex studies have been employed for monitoring the excitability of AHC poolin different CNS disorders such as stroke, dystonia, parkinsonism and cerebellar diseases.[12]

Several studies have shown the effects of therapeutic passive exercise of hip and knee joints on the soleus H-reflex and found the results that in both exercises, H_{max} :M_{max} ratio decreased as flexion progressed with fast or slow movements. The qudriceps femoris inhibited soleus muscle alpha motor neurons in both experiments. [27,29,31]

Little and Halar recorded how increased H-response amplitude and H/M ratio developed along with spasticity in six spinal cord injured patients during months 1 to 3 post-injury.[35]

Higashi T. Et al, studied the motoneuron pool excitability of hemiplegic patients assessing recovery stages by using H-reflex and M response. They used Hslp/Mslp (developmental slope of H-reflex and M response) and Brunnstrom stages. Results showed that Hslp/Mslp had better predictive value than conventional indicators of motoneuron pool excitability. Hslp/Mslp is the preferred index for evaluating the motoneuron pool excitability of the spastic side of hemiplegic patients.25

Hiersemenzel et al have demonstrated three stages of adaptational change in the excitability of the spinal neuron circuits in patients with spinal cord injury. They found that, although the H-reflex can be elicited shortly after the event, the H_{max}:M_{max} ratio reached its maximum in 8-24 weeks and remained stable thereafter.48 It was therefore important to examine patients at least six months after disease onset, as we did in the present study.

The present study also tried to compare MAS and H_{max}:M_{max} ratio. This showed that there was stronger relationship between the spasticity by MAS and H_{max}:M_{max} ratio post CPM (p-value 0.001), however, pre-CPM there was no statistical correlation (p-value 0.17).

A M O Bakheit et al recorded the MAS in patients with post stroke lower limb muscle spasticity and correlated with the excitability of the alpha motor neurons which was evaluated by measuring the latency of the H-reflex and the ratio of the amplitude of the Hmax and Mmax and concluded that there is a relation between the MAS scores and alpha motor neurons excitability but it is suggested that the MAS measures muscle hypertonia rather than spasticity.[11]

Some studies have shown the poor correlation between the results of the neurophysiological tests and the degree of spasticity, probably caused by problems inherent to the MAS itself. This scale, MAS, which relies in the subjective judgement of the examiner, measures resistance to passive muscle stretch. This resistance often reflects a combination of spasticity, thixotrophy, and fixed muscle contractures.[53, 54]

In the present study, however, post CPM the spastic grades significantly reduced and subjectively the patients reported increased benefit during gait in the form of reduced heaviness, improved movement strength and overall confidence, developed easy ambulation after the CPM for 20 minutes.

7. CONCLUSION

1. The study supports the use of passive movements in the reduction of spastic hypertonia. Subjectively there was significant benefit reported by the patients in acitvities like reduced heaviness in walking, immediately following CPM.

2. MAS and H reflex parameters i.e. H-amplitude and H/M ratio were both significantly high pre CPM. In both there was significant reduction seen post CPM.

3. The spasticity grades on MAS of knee extensors and ankle planter flexors showed statistically significant reduction post CPM.

4. MAS was seen to be statistically significantly correlated to H/M ratio post CPM, this relation was not statistically significant pre CPM.

5. The study indicates both MAS and H/M ratio to be useful tool to assess the hypertonia in clinical practice.

6. There was no change seen in the H-latency and M-wave parameters, amplitude and latency between affected and sound limb.

LIMITATIONS:

The sample size is small thus it would be better if this study was conducted on large scale.

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APPENDIX - A

TABLES:

Table 1: Frequency Table

Age	Frequency	Percent
35	4	19.0
36	2	9.5
39	1	4.8
43	1	4.8
45	1	4.8

47	1	4.8
51	2	9.5
52	2	9.5
55	1	4.8
58	3	14.3
59	1	4.8
60	2	9.5
Total	21	100.0

Table 2: MAS of Hip extensors before and after PM on CPM device

Muscle	No. of subjects	Pre CPM MAS	Post CPM MAS
Hip extensors	16(76.2)	1	1
Hip extensors	5(23.8)	1+	1
Total	21		

Chi-square test is not applied here.

Table 3: MAS of knee extensors before and after PM on CPM device

Muscle	No. of subjects	Pre CPM MAS	Post CPM MAS
Knee extensors	12(57.1)	1	1
Knee extensors	6(28.6)	1+	1
Knee extensors	3(14.3)	2	1+
Total	21		

Chi-Square Tests			
	Value	Df	p-value
Pearson Chi-Square	13.263a	2	.001

Significant relation: p value <0.05.

Table 4: MAS of Ankle planter flexors before and after PM on CPM device

Muscle	No. of subjects	Pre CPM MAS	Post CPM MAS
Ankle planter flexors	7(33.3)	1	1
Ankle planter flexors	8(38.1)	1+	1
Ankle planter flexors	6(28.6)	2	1+
Total	21		

Chi-Square Tests			
	Value	Df	p-value
Pearson Chi-Square	15.660a	4	.oo4

Significant relation: p value <0.05

Table 5: Comparison of H-reflex before and after PM on CPM device

Paired samples statistics					
		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	H-amplitude affected before	6.0029	21	4.81965	1.05173
	H-amplitude affected after	4.1857	21	3.37788	.73711
Pair 2	H-latency affected before	31.395	21	3.4334	.7492
	H-latency affected after	31.471	21	3.4308	.7487
Pair 3	M-amplitude affected before	13.3181	21	6.34035	1.38358
	M-amplitude affected after	15.127	21	7.1700	1.5646
Pair 4	M-latency affected before	4.324	21	.6024	.1315
	M-latency affected after	4.295	21	.5792	.1264
Pair 5	HM ratio affected before	.5057	21	.49323	.10763
	HM ratio affected after	.2852	21	.21444	.04680

Pair No.	T	Df	p-value
Pair 1	3.978	20	.001
Pair 2	-1.171	20	.255
Pair 3	-1.705	20	.104
Pair 4	.826	20	.419
Pair 5	2.550	20	.019

Significant difference: p value <0.05

Table 6: comparison of mean H/M ratio for different levels of MAS for ankle planter flexors, pre and post CPM

Descriptive statistics

Comparison of mean H/M ratio for different levels of MAS for Ankle planter flexors, pre CPM			
N	**MAS of ankle pl flexors**	**Mean of H/M ratio**	**Std. Deviation**
7	**1.00**	.2186	.1799
8	**1.50**	.6388	.6906
6	**2.00**	.6633	.3051
21	**Total**	.5057	.4932

ANOVA Table

			Sum of Squares	Df	Mean Square	F	Sig.
H/M ratio affected pre CPM. (Ankle pl. flx)	**Between groups**	**Combined**	.868	2	.434	1.954	.171
	Within groups		3.998	18	.222		
	Total		4.866	20			

Comparison of mean H/M ratio for different levels of MAS for Ankle planter flexors, post CPM			
N	**MAS of ankle pl flexors**	**Mean of H/M ratio**	**Std. Deviation**
13	**1.00**	.1877	.1432
6	**1.50**	.3583	.1837
2	**2.00**	.7000	.0707
21	**Total**	.2852	.2144

ANOVA Table

			Sum of Squares	Df	Mean Square	F	Sig.
H/M ratio affected post CPM. (Ankle pl. flx)	**Between groups**	**Combined**	.500	2	.250	10.712	.001
	Within groups		.420	18	.023		
	Total		.920	20			

Significant difference: p value <0.05