MOLLII SUIT MECHANISMS

Combined explanatory models

of the Mollii Suit:

Activation

Relaxation

Pain Reduction

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Introduction

The Upper Motor Neurone Syndrome (UMNS) is defined as alterations in the physiological motor control in the skeletal muscle after an insult and/or lesion in the Central Nervous System (CNS). One of the main components and outcomes is spasticity/rigidity, known collectively as a "positive" phenomenon and is characterised by muscle over-activity. Hyperactive spinal reflexes mediate most of these positive phenomena as lesions or insults in the CNS lead to disturbances of the control of spinal reflexes, which in turn leads to motor deficiencies such as spasticity, muscle weakness syndromes and/or different types of dystonia.

Dystonia

Dystonia is a neurological syndrome characterised by involuntary twisting movements and unnatural postures. There are many types of dystonia and many diseases and conditions may include dystonia as a symptom. In terms of tactics for programming the Mollii Suit we will include athetoid dyskinetics and ataxia under dystonia.

Dystonia is classified by:

- Clinical characteristics such as age of onset, body distribution, nature of symptoms and associated features such as additional movement disorders or neurological symptoms
- Cause which includes changes or damage to the nervous system and inheritance

Mollii can have a positive effect on the following types of dystonia through activating mechanisms to help with motor control:

- Focal
- Segmental
- General

Neuro-motor activity to reduce dystonic symptoms should include:

- Increase and preserve range of motion and mobility needed for function
- Strengthen weakened muscles that may be under-utilised in the presence of dystonia movements (there is always an imbalance between antagonists in dystonic symptoms)
- Promote awareness and maintenance of optimal body posture – the ability to flex the trunk, equals to an extent, the ability to control general over-activity when dystonic symptoms are present

Sensory input, in the Mollii Suit's case via electrical stimulation, is essential to activate this proprioceptive awareness. Motor control is not only present in dynamic activity, it is equally important for maintaining position and balance control.

Coordination is also achieved in a predictive, feed-forward manner. For example, to make a successful reaching movement, the body needs to be in balance (trunk control), then the muscular activity around the shoulder joint needs to be tightly coordinated with the muscular activity around the elbow joint to compensate for the interaction torques and to ensure a straight reaching trajectory movement.

Activation

Increasing muscle strength and motor activity

In general, muscle fibres respond to frequencies at 20 Hz (slow-twitch fibres), which is low enough to not cause fatigue but effective enough to have a lasting effect during the length of its active involvement. Voluntary contraction is more fatiguing than low level electrical stimulation at both the cardiovascular and nervous levels (neurotransmitter fatigue). Electrostimulation is only more fatiguing than voluntary contraction at the energy consumption level (ATP use) – up towards 80 Hz and beyond (depending on individual factors).

One of the reasons low level electrical stimulation does not lead to fatigue is the concept of subthreshold activation. Meaning that there will be no visible contraction due to this stimuli, but rather a pre-activation leading to easier voluntary movement. This is dependent on the right intensity/microampere, which needs to be individually assessed to not lead to overstimulation.

Subthreshold electrical stimulation reduces motor unit discharge variability and decreases the force fluctuations when performing voluntary movement.

Electrical stimulation at 20 Hz has been shown to produce an enhancement of beta synchronisation in the basal ganglia, which also should exacerbate antikinetic symptoms. Work from a research team called Brown's group indicates that sub-thalamic nucleus (STN) stimulation at 20 Hz increases Globus pallidus (GPi) synchrony (Brown et al., 2004).

Keywords in assessing and counteract dystonic symptoms:

- Flexion: Primarily trunk and hips, but flexion in all bigger joints inhibits overactive reflexes
- Midline: Help muscle groups that strive movements towards the midline and centre of gravity
- Trunk control: The trunk “anchors” movements in the extremities (feed-forward mechanism), meaning that any voluntary movement of limbs and head are preceded by an activity of certain trunk muscles depending on the type of movement

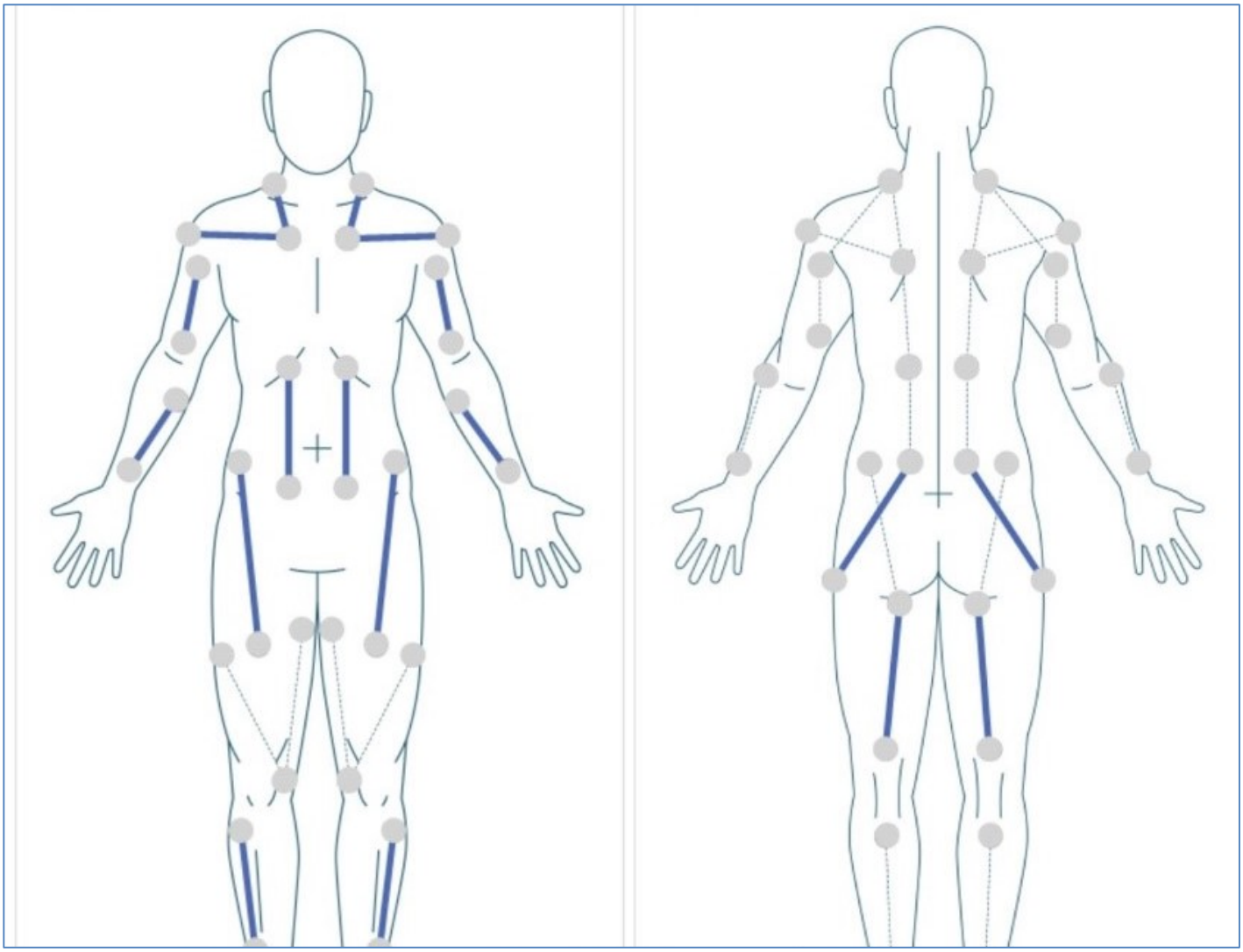

Figure 1 Dystonia/athetoid/ataxia programming Molliisoft

The picture above shows an example for settings of dystonic/athetoid/ataxic symptoms. There are of course often individual variations, but regarding the reasoning in the beginning of this chapter, this is how to programme for dystonic symptoms.

One thing to remember is the mixed types of cerebral palsy, where there can be a dystonic presentation from the waist and up and static spasticity in the lower limbs. Actually, ten percent of the CP population falls under the mixed type category. And seventy percent in that category displays these kinds of symptoms. The rest can be a mix of any other type of motor disorder related to cerebral palsy – an important note when it comes to assessment of children with CP. Adults with dystonia of other origin than oxygen deficiency from birth or as a fetus, seldom fall under the mixed type category.

Relaxation

Spasticity

Physiologically, spasticity is defined as a motor disorder characterised by a velocity dependent increase in the tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflexes as one component of the upper motor neuron (UMN) syndrome (Lance, 1980). The velocity dependent increase in resistance to passive stretch often melts suddenly resulting in clasp-knife phenomenon. The definition of spasticity was further elaborated by addition of several features, mainly by an MD named Robert R Young nine years later, to form a more comprehensive picture of UMN syndrome.

- In patients with spasticity, brisk tendon jerks sometimes accompanied by clonus and velocity dependent muscle hypertonia to stretch preferentially affecting certain muscle groups, are the effects of a combination of hyper-excitability of an afferent pathway to motor neurons and disturbed processing of other peripheral afferent pathways at the spinal cord level.
- In spasticity, other positive symptoms or signs such as flexor (or extensor) spasm, clasp knife phenomenon, Babinski sign, exaggerated cutaneous withdrawal (flexor, pain) reflexes, autonomic hyperflexia, dystonia, and contractures may limit voluntary movement and cause discomfort.
- In addition to the above features, several negative features are also included in spastic states such as paresis, lack of dexterity and fatigability.

Pure spasticity may be seen when an arm or leg appears to “catch” momentarily when a relaxed limb is quickly moved. There will be a stiffening and then relaxation so the motion can be completed.

When muscles at rest are overactive without any triggering factor, parts of the body assume abnormal positions, which are a major cause of disfigurement and social handicap.

In time, during this stage called “spastic dystonia” *(not to be confused with the dystonic explanation given earlier)*, there are changes in muscle composition, so the muscle becomes shortened and the range of motion limited. Muscles may reach a state of permanent contraction, or joints may become completely immobile.

Secondary Spasticity

The shortening of soft tissues initiates chain reactions, such as triggering a rhythmic contraction during walking, so the lower limbs bend or extend further than necessary, disrupting normal gait. This occurrence is called “secondary spasticity”, or hypertonia. It includes a nervous system component triggered by spasticity, and a biomechanical component arising from changes in the soft tissue.

The constant contraction of “spastic” muscles renders opposing muscles inactive, and the latter grow weak due to disuse. A vicious circle is created when the muscle that flexes the elbow is permanently contracted, and the inability to extend the elbow makes the opposing muscle become weak due to inactivity.

No matter what the spasticity is called, it needs to be assessed and addressed using reciprocal inhibition to counteract the symptoms.

Reciprocal inhibition

Reciprocal inhibition is the automatic antagonist alpha motor neurone inhibition which is evoked by contraction of the agonist muscle. Joints are controlled by two opposing sets of muscles, extensors and flexors, which must work in synchrony for smooth movement. When a muscle spindle is stretched and the stretch reflex is activated, the opposing muscle group must be inhibited to prevent it from working against the resulting contraction of the homonymous muscle. This inhibition is accomplished by the actions of an inhibitory interneuron in the spinal cord. This so-called natural reciprocal inhibition is a ubiquitous and pronounced phenomenon and plays a major role in the control of voluntary movements and reduction of static spasticity.

The ability to activate the antagonist equals the ability to deactivate the protagonist. Muscle spindle sensitivity to stretch reflects the balance of activity between antagonistic muscles (i.e. "antagonistic muscle balance").

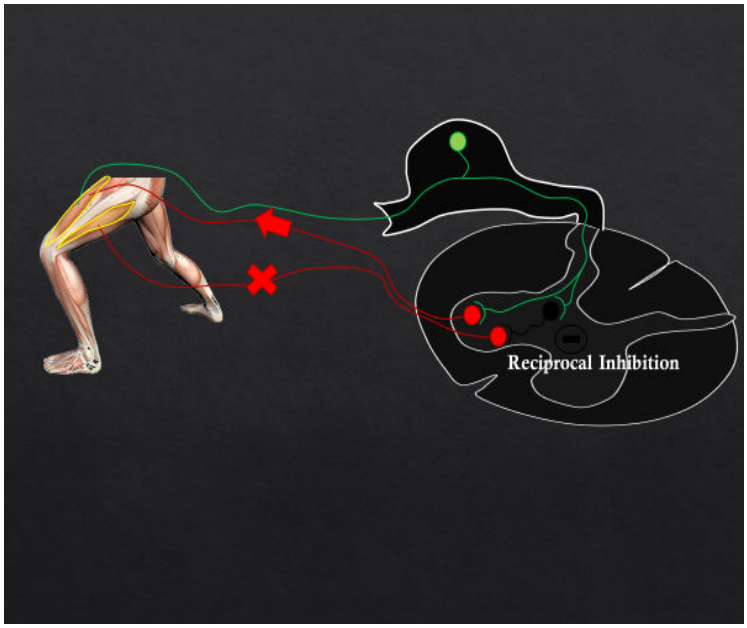

Figure 2 Reciprocal Inhibition

By helping to activate the weaker antagonist through low level electric stimulation, there will be reciprocal inhibition of the overactive/spastic protagonist. Since this is taking place at spinal cord level, thus not consuming enough energy to cause fatigue, it will open up possibilities for voluntary movement.

Programming to Reduce Spasticity

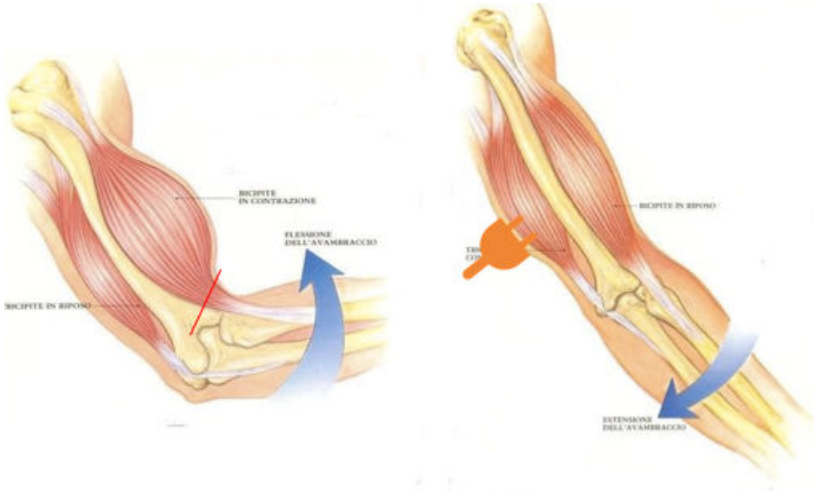

Figure 3 Spasticity in bicep muscle

This example shows an increase of activity in the bicep muscle, leading to a more or less rigid position of the parts of the arm depending on elbow joint function. The low level electrical stimulation is applied on the antagonist of the biceps muscle. This way of assessing where to induce the electrical stimulation can be applied on all limbs and body parts where spasticity is present.

Alpha Gamma Loop

When the body is injured, the gamma loop can become imbalanced. Sensory cells within the muscle spindles do not know how to interpret the input. The signal sends an improper impulse toward the brain and spinal cord. Since the impulse is abnormal, the alpha motor neuron also misinterprets the information. The efferent response, which would be a muscle contraction, is now a muscle spasm.

The alpha-gamma loop is the communication or flow of information between your alpha motor neurons and gamma motor neurons. It helps to synthesise sensory input to compute an appropriate motor response, or output. Similar to what is described about the importance of awareness of the body in space to keep a certain position.

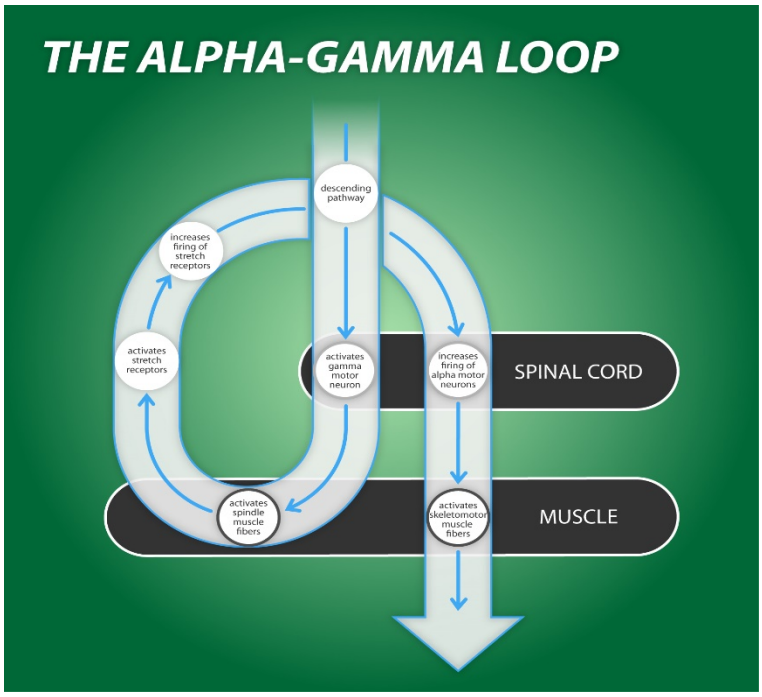

Figure 4 Alpha-gamma loop

The alpha-gamma loop is the next level of motor control compared to reciprocal inhibition. It is very difficult to be able to achieve improved motor control through alpha-gamma integration unless reciprocal inhibition is functioning beforehand.

Pain Reduction

Serotonin, endorphin and opioid release

Transcutaneous electrical nerve stimulation (TENS) is a commonly used non-pharmacologic and non-invasive treatment for pain. TENS reduces pain through both peripheral and central mechanisms. Centrally, sites in the spinal cord and brainstem that utilise opioid, serotonin, and muscarinic receptors are activated by TENS. Peripherally, at the site of TENS application, opioid and α-2 noradrenergic receptors are involved in TENS-induced analgesia.

TENS is the application of electrical current through electrodes placed on the skin for pain control. It can be applied with varying frequencies, from low (<10 Hz) to high (>50 Hz). This segment will concentrate on low intensity because of the Mollii Suit's parameters. Intensity may also be varied from sensory to motor intensities. Sensory intensity is when the patient feels a clear but comfortable sensation without motor contraction. Regardless of intensity, different frequencies activate central mechanisms to produce analgesia. Specifically, low-frequency TENS activates μ-opioid receptors in the spinal cord and the brainstem leading to a decreased sensation of pain.

Spinal serotonin concentrations are also increased during and immediately after treatment with low-frequency TENS, showing that the sites of applying electrodes are not crucial to get this effect. The numbers of electrodes applied are also important to get spinal serotonin increase. There is even a marked increase in beta endorphin and met-enkephalin with low-frequency electrical stimulation (ES).

Gate control mechanism

Low level ES also reduces pain through nociceptive inhibition at the presynaptic level in the dorsal horn, thus limiting its central transmission. The electrical stimuli on the skin preferentially activate low-threshold, myelinated nerve fibers. The afferent input from these fibers inhibits propagation of nociception of the dorsal horn.

According to the axonal diameter and the conduction velocity, nerve fibres can be classified into three types; A, B and C. The C fibres are the smallest among all the three types. The C fibres are the smallest of all three types. Among the A fibres are four subtypes: A-alpha, A-beta, A-gamma and A-delta. Among the A subtypes, the A-alpha fibres are the largest and the A-delta fibres are the smallest. A-alpha, A-beta and A-gamma carry sensations like touch and pressure to the spinal cord. The A-delta fibres and the C fibres carry pain signals to the spinal cord. A-delta fibres are faster and carry sharp pain signals while the C fibres are slower and carry diffuse pain signals.

When considering the conduction velocity, the A-alpha fibres (the large nerve fibres) have higher conduction velocity when compared to the A-delta fibres and the C fibres (small nerve fibres). When a tissue is injured, the A-delta fibres are activated first, followed by the activation of the C fibres. These fibres tend to carry the pain signals to the spinal cord and then to the brain.

When the pain signals are carried by the small fibres, pain signals are less intense compared to the other non-pain sensory signals like touch, pressure and temperature, the inhibitory neurons prevent the transmission of the pain signals. The non-pain signals override the pain signals and thus the pain is not perceived by the brain.

Pain management and the Mollii Suit

Movement itself, and increased blood flow due to that movement, reduces (to an individual extent) pain in itself. Programming of the Mollii Suit should always be according to whatever movement disorders are presented during assessment. But adding other sites of active electrodes will have an additional effect in pain management according to the mechanisms described. Lower intensity, that will not interfere with the activation/deactivation of motor symptoms, should be added through multiple sites if pain is a major factor.

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