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Cerebral Palsy: A clinical overview.

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Abstract
Cerebral palsy is the commonest cause of spasticity and physical disability in children and spasticity is one of the most common problems in those with neurological disease. The management of spasticity in children with cerebral palsy requires a multidisciplinary team effort at the earliest. There are various treatment options available for the management of spasticity. This article reviews the variety of options available for the management of spasticity in children with Cerebral Palsy.
Key Words: *Cerebral Palsy, Spasticity , Physical Therapy, Occupational Therapy, Constraint Induced Movement Therapy, Artificial Muscle Power.*

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I. Introduction

Cerebral palsy (CP), the most prevalent motor disorder of childhood, affects two to three per 1,000 live births. Wendy. M. Nehring - Shirley Steele[1] stated that, Cerebral palsy is a condition first described by Dr. George Little defined as a group of non progressive disorders that cause aberrant movement and posture resulting from central nervous system damage or insult in the early periods of brain development, usually in the first 5 years of life.

Cerebral palsy is a collection of diverse syndromes characterized by disorders of movement and posture caused by a non progressive injury to the immature brain.[2] The distinctive characteristic of these syndromes is a change in muscle tone and posture, both at rest and with voluntary activity. Spastic cerebral palsy,[3] the most prevalent form of cerebral palsy is characterized by motor in coordination, primarily in the lower extremities that impairs many functional abilities, most notably the ambulation.

Definition of Spasticity :

Spasticity as a pathologically exaggerated resistance of special character to passive moments if these performed with a certain minimal speed. According to Angela Forster[4], spasticity is the pathological state of increased muscle tone resulting from damage to the upper motor neuron. Often the small anterior horn cell is released from the higher control of the extra pyramidal system and fires spontaneously at an increased rate. The net result of this is ultimately to increased tone in the extrafusal muscle fibers, when the hypertonic spastic state appears.

Spasticity seems to be a major concern of the clinician and much emphasis is placed on it in therapy[5] However it has been two author's experience since embarking on a motor learning based approach to therapy that spasticity as described in the literature need not be major deterrent to the regaining of controlled motor behaviour in most spastic children.

Margaret Johnstone, [6] concluded that, spasticity once it becomes established will lock the affected limbs into non functional, useless & painful patterns which will put an end to all worthwhile rehabilitation.

Spasticity is defined as a velocity dependent increased resistance to passive muscle stretch associated with upper motor neuron paralysis[7, 8] . Spasticity can lead to functional problems with daily living activities such as dressing, feeding, washing, and toileting etc.[9]

Although the majority of children with spastic cerebral palsy are eventually able to ambulate, the acquisition of this skill is delayed and differs qualitatively from normal. The ability to perform the Lower Limb function is a major concern of the parents of children with Cerebral Palsy improving and maintaining this ability to ambulation is often considered to be the primary focus of most of the problem seen in children with spastic Cerebral Palsy.

II. Literature of Review:

There are epidemiological, clinical and review studies about management of spasticity in children with cerebral palsy.

Galjaard [10] described that Cerebral Palsy has both motor & anatomic groupings ; etiologically it is a set of disorders that is multi factorial & is diverse in clinical presentation.

Jane Styer – Aeveo[11] also describes, Children with cerebral palsy, results from a muscular imbalance between the plantarflexors and dorsiflexors. It is manifested as toe walking in the ambulatory child, children with more severe involvement may have difficulty with foot placement on the pedals of the wheel chair, assisted stand pivot transfers, and donning of shoes. Conservative management includes Passive stretching , with care taken to lock the subtalar joint before stretching toward dorsiflexion.

Helen Cohen[12] describes that the Golgi Tendon Organ (GTO) is most sensitive to force when the muscle is contracting. The tendon organ reflex provides negative feedback. To regulate the excessive development of muscle force. The type 1b afferent carries signals from the tendon organs located at the musculotendinous junction. The type 1b -IN that inhibits afferent influences the homonymous motor neuron pool through an inhibitory type. lb -IN that inhibits the alpha motor neuron of the muscle that is generating the force. This process is termed as autogenic inhibition.

Functionally the tendon organ serves more than a protective role, because of its exquisite sensitivity to Muscle force and it's anatomic location, which allows if to sample a large number of motor units, the GTO probably acts with the muscle spindle to regulate muscle tone & compliance of muscle.[13] Similar to the muscle spindle the influence of the GTO on the alpha motor neuron pool can be regulated centrally by convergent input to the type 1b -IN [14] input converges on the type 1b-IN from multiple sources, including muscle spindle type 1b afferents, joint capsule afferents, cutaneous afferents & exitatory inhibitory descending input from higher canters.

Inhibition techniques increase muscle length by relaxing and elongating the contractile components of muscle. [15] Spasticity is the presence of increased muscle tone, which is noted through the passive range of motion of a joint.

Mark L. Latash[16] have defined Spasticity is a disorder of spinal proprioceptive reflexes manifested as profound changes in reflexes to muscle stretch, with a strong velocity dependent component. Emergence of pathological reflexes & uncontrolled spasms, an increase in muscle tone and impairment of voluntary motor function. Spasticity can be associated with exaggerated unchanged, and even absent monosynaptic reflexes including the H - reflex, although an increase in monosynaptic reflexes is more typical.

Cooling has been used clinically for many years to reduce muscle spasticity.[17] There is good objective evidence to support this, for example cold producing a marked decrease in ankle clonus, described by Peterjan and Watts. The mechanism by which the reduced spasticity may be brought about have been studied extensively without being fully elucidated.

The decrease in spasticity associated with cryotherapy may have a positive effect on mobility and may allow an increased level of participation in a therapy program.The reduction in spasticity can be sustained for several hours, the exercise or activity should be initiated within that time frame.

Paul.D.Hooper describes that the body responds to the application of ice in stages. The initial reaction to cold is local vasoconstriction with a consequent reduction in blood flow. This is useful during the early stage when its necessary to control swelling, decreased nerve conduction velocity, reduced muscle spindle activity, which is the primary rationale for the use of ice immediately.

Commercial cold packs contain a semi gelled substance, covered in durable plastic they are manufactured in sizes similar to those of hydro collator packs, they may be applied either directly to the skin or can be used with a wet or dry interface, depending on the desired intensity of the cold application.[18] These cold packs confirm to irregular surface areas but maintaining a constant temperature is problematic. The patients should be positioned comfortably and wrapped appropriately for the duration of the ice application. Average time for a cold or ice pack application is 10 to 15 minutes.

Lehman & De Lateur, describes the effectiveness of cooling as a means of reducing spasticity for any particular patient it would seem advisable to apply sufficient cold. The mechanisms by which cold reduces spasticity & spasm are probably the same. [19] Many authorities point to the reduced velocity of nerve conduction or depressed sensitivity of receptors such as the muscle spindle while these undoubtedly occur.

Lynne. M. Stempien states that, tone may be decreased very shortly after the application of ice, probably due to decreased sensitivity in cutaneous receptors slowing of nerve conduction these structures are fairly deep & it would take several minutes to produce a sufficiently low temperature to affect them, where as a reduction of spasm or spasticity can be demonstrated clinically within 30 seconds of application of ice. In this time no structure other than the skin can be affected. This suggests that the skin stimulus produced by cold must have an effect on the general level of excitation or inhibition in the region of the anterior horn cells in the cord. Once spasticity have been reduced it is important that more long term treatment is given in order to sustain this

condition. Some attempt to contract the antagonists of the dominant spastic muscles in order that these muscles inhibit the dominant groups in a physiological way.

Miglietta [20] investigated the effects of cold on sustained ankle clonus and reported that clonus was either decreased or eliminated after cold whirlpool at 18.3.°C for 15 minutes the changes were maintained for several hours. The decrease in spasticity with cryotherapy may have a positive effect on mobility and many allow an increased level of participation in a therapy program, because the reduction in spasticity can be sustained for several hours the exercise or activity should be initiated within that time frame.

Stretching is probably an essential component for relieving muscle stiffness in spastic patients, although there are few quantitative reports demonstrates spasticity relieve following muscle stretch.[21] In fact the long-term efficacy of stretch for reducing spasticity has been challenged. Ranging can reduce the severity of spastic tone for several hours. The reason for the 'carry-over' is not completely clear, but it could be related to mechanical changes in the muscle tendinous unit or to plastic changes within the central nervous system. These plastic events may correlate with short and long-term modulation of synaptic efficacy associated with neuron transmitter changes on a cellular level.

Roberta B. Shepherd[22] stated that Contractures probably develop because of an imbalance of muscle activity, lack of active functional movement provided active stretch, prolonged positioning with muscles held at one length and the effect of these on growth. Growth has been shown in animals to develop partly on stretch. In other words, muscles may develop length associated changes because of lack of active use or persistent use in one part of a joint range. The muscles antagonistic to short muscles will length, there by adding a mechanical disadvantage to the neural disability. The hyper active muscles continue to shorten & become increasingly stiff, lessening their ability to generate force at all lengths necessary for specific functions.

Gabriella E. Molnar & Michael A. Alexander[23,24] stated that Hypertonicity is an increase in resistance to stretch by a muscle group upon external manipulation. It can result in decreased joint range of motion, which can improve with passive stretching. Muscle stretching is particularly important in the therapeutic realm the gastrocnemius muscle is often the first to develop contractures in the spastic hypertonic patients. The use of night time foot orthosis (AFO) may bring great benefits to these children.

In addition Joel A. Delsia[25] describes, Contraction of antagonist muscles also may be useful to inhibit reciprocally the muscle that is being stretched this is also has the effect of muscles that are tending to overcome the tightness. Stretching of muscle tissue has been shown to result in an increase in the number of sarcomeres in the muscle fibers[26]. Simple passive stretch of muscle has also been shown to increase oxidative capacity and to reduce the net protein breakdown in vitro[27]. Superficial cold reduces blood flow, decreases metabolic activity, lessens muscle tone, and inhibits spasticity and clonus. After 10 to 20 minutes of vigorous cooling, spastic muscle loses considerable tone and voluntary function may be easier to isolate.

Enhanced flexibility achieved through stretching promotes greater compliance of the muscle tendon unit. However, the therapist must determine the best method of stretching for each patient to gain the desired compliance.

Static stretching offers three distinct advantages:

1. Less danger exists of exceeding the extensibility limits of the tissues involved.
2. Energy requirements are lower.
3. Static stretching will not cause muscle soreness, and infact may relieve it.

These advantages are quite reasonable because connective tissue has a very high tensile resistance to a suddenly applied tension of short duration, while demonstrating viscoelastic and plastic elongation when placed under prolonged mild tension. Static stretching may minimize any impact on the Ia and II spindle afferent fibers and maximize the impact on the golgi tendon organ. Prolonged (or) chronic over stretching can result in an increased number of sarcomeres in the muscle fibers and may produce an improper muscle length relative to the lever system.

When range of motion has been lost and a contracture has developed more vigorous efforts are needed to reduce the contracture and gain the range of motion. When stretching a contracture the force needed fort stretch is larger for the first stretch than for subsequent ones, possibly because of initial breaking of adhesions or inter molecular cross - linkage . A slow rate of application of the stretching force produced move elongation than a rapid rate[28.] Physiological studies suggest that stretch should be of long duration with low force and a gradual increase in the force. The longer the stretching force is applied the more quickly the tissue will elongate. The amount of force applied is a compromise between the force necessary to cause elongation with out disrupting the tissue, and the force that can be tolerated by patient.

Devries *et al* reported significantly better improvement in the flexibility of plantar flexors and hip adductors. and extensors for subjects who trained using the static stretch (11 to 25% increase) technique compared to ballistic stretching (3 to 7% increase). The effectiveness of static stretching for increasing range of motion is good & the resistance to stretch is low.[29]

There are a number of approaches designed to stretch muscle and often soft tissues, which may limit flexibility. In general, flexibility exercises aim to increase the range of motion either with the limits muscles passive (Static) or with one or more of the muscles attempting to assist the stretch (dynamic).[30]

Susan *et al* stated that caution must be used to prevent fast ballistic stretching movement because spasticity is velocity sensitive. Stretching movements need to proceed slowly to gradually achieve the desired range. Each stretching position should be maintained for at least 30 to 60 seconds to allow the muscle to adjust to the new position. Passive stretching involves applying a mechanical force at the physiological end of range elongating the resting length of tissues. The terms static stretching is used to refer to a low load maintained stretch applied for an extended period of time (i.e., at least 15 10 to 30 seconds or longer depending upon the patient's tolerance. Inhibitory casting or splinting is another form of static stretching used to increase range and decrease tone. The benefit of static stretching using low loads include less danger of tearing the tissues, less muscle soreness and decreased energy requirements.Both static & ballistic stretching achieve similar result, but, ballistic stretches are more likely to produces muscle soreness.[31]

The general agreement that flexibility exercises should be directed at including plastic rather than elastic changes in connective tissue, because plastic changes will produce move permanent changes in tissues length. It is thought that static stretching has its greatest effect on the non counteractile connective tissue, elements, where as neuromuscular facilitatory influences the length of the contractile elements by adding sarcomeres to its length.

Measurement of spasticity

According to Pederson the measurement of spasticity is complex & difficult but its compulsory to obtain a quantitative measurement of spasticity m order to prove the effectiveness of treatment.[32] The universal goniometer is a protractor like device with two steel or plastic arms that measure the joint angles at the extremities of the Range of motion.

Morrey *et al* describes, joints and their related structures are assessed by performing various active & passive joint motions.[33] Joint motion is a necessary component of most functional tasks. Careful examination of joint motion for range of motion (ROM), effect on symptoms, and patterns of limitation helps to identify impairments causing functional disabilities and determine the structures that need focused treatment.

The average standard deviation between measurements made on the same objects by different testers to be 4.2 degrees for upper extremity motions and 5.2 degrees for lower extremity motions. The use of standardized positions, Stabilization of the body part proximal to the joint being tested bony land marks to align the goniometer and the same examiner rather than multiple examiner for repeated testing all help to improve the validity & reliability of goniometric measurement.

Modified Ashworth scale for grading spasticity ;- Spastic hypertonia can be assessed using the Ashworth scale, a 5-point ordinal scale or the modified Ashworth scale. This latter scale was developed to provide an additional intermediated grade and has been shown to have high interrater reliability.

Recent advances:

Constraint Induced Movement Therapy :

Constraint Induced Movement Therapy (CIMT) is a new treatment technique that shows the improvement in the arm motor ability and the functional use of a paretic arm - hand. CIMT forces to use the affected side by restraining the unaffected side. A child with hemiplegic cerebral palsy can learn to improve the motor ability of the more affected parts of their bodies and thereby decreases to depend mainly on the less affected parts.[34]

In this concept, the less affected arm-hand was immobilized in a sling[35] but soon an emphasis on intensive, repetitive training of the more affected arm-hand evolved. In this method, the patients wear a mitt on the less-affected arm of their waking hours and need to perform repetitive exercises on the affected arm six to seven hours per day. [36]

Study shows that, CIMT improves the movement on the affected side. Patient shows a greatest improvement in the functional use of their affected arm in their daily lives. In addition, CIMT patients were able to speed their completion of tasks in lab testing while comparing to placebo patients in upper extremity function after constraint-induced therapy have been reported in all stages after the onset of stroke[37] .

Artificial Muscle Power:

Cerebral palsy mainly affects the central nervous system and physical movements, it can be difficult for individuals with Cerebral Palsy to walk or to develop fine motor skills. Researchers at the University of Delaware's College of Health Sciences from USA, are trying to develop a motorized ankle device that includes an actual artificial muscle with "smart materials" such as dielectric elastomer actuators, the life-like soft muscle

tissues will contract after receiving the electrical signals. They are nearly mimicking the natural movement of the human muscle. They are aiming to release this soon for the market; their research represents a major breakthrough for all kinds of neuromuscular disorders.

III. Conclusion

The management of spasticity following a cerebral palsy is a most challenging complex task to the treatment team. Initial management should focus on the elimination of externally exacerbating causes. If the spasticity interferes with function, causes pain, and produces deformity, then the most appropriate treatment goals should be established.

The treatment needs to be evidence-based and depends on the degree of functional failure caused by the spasticity and its location. This management often requires a variety of different approaches including oral medications, botulinum toxin, Physiotherapy, Occupational therapy and often surgical interventions such as selective dorsal root rhizotomy and orthopaedic surgery.

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