ORIGINAL ARTICLE

Surgeries and physical interventions in the treatment of cerebral palsy spasticity

Cirurgias e intervenções físicas no tratamento da espasticidade na paralisia cerebral

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ABSTRACT

Although the espasticidade treatment in patients with cerebral palsy is based essentially on cinesioterapia and drugs administrated as chemical blockades infiltration or orally, there are situations where such aid does not have the desired effect, either because of the seriousness of the espasticidade or contra-indications and adverse effects of the botulinum toxin. We can also see human musculoskeletal apparatus deformities resulting from constant deforming stimulus of espasticidade and amendment biomechanics that it imposes on the member positioning during the gait, ortostatismo or other rest positions. In this situation, are indicated surgical procedures which aim at improving the condition of positioning biomechanics, adequacy

and efficiency of movement in general. This review article has the aim to provide alternative forms of drug administration for the control of espasticidade, as baclofen bump that requires a chirurgical procedure and others indications and surgeries for the control of espasticidade and its consequences as deformities. We will discuss also the indications for physiotherapy procedures and ortheses indications with the indication to reduce spasticity.

Keywords: Muscle Spasticity, Cerebral Palsy, Botulinum Toxin Type A

RESUMO

Apesar de o tratamento da espasticidade em pacientes com paralisia cerebral fundamentar-se, essencialmente, na cinesioterapia e medicamentos administrados como infiltrações ou por via oral, ocorrem situações nas quais tais intervenções não surtem os efeitos desejados, seja em virtude da gravidade da espasticidade ou da contra-indicação e efeitos adversos ao uso da toxina botulínica. Também podem ocorrer distorções do aparelho locomotor resultantes do constante estímulo deformante da espasticidade e da alteração biomecânica que ela impõe ao posicionamento de membros durante a marcha, ortostatismo ou outras posições de repouso. Nestas últimas situações, estão indicados procedimentos cirúrgicos que visam a melhoria da condição biomecânica,

adequação do posicionamento e maior eficiência da movimentação em geral. Este artigo de revisão tem por objetivo apresentar as formas alternativas de administração de drogas para o controle da espasticidade, como a bomba de baclofeno, cujo implante exige procedimento cirúrgico, outras indicações e intervenções cirúrgicas para o controle da espasticidade e de suas conseqüências como deformidades e alteração de função além de procedimentos fisioterápicos e uso de órteses sempre com o objetivo de redução dos quadros espásticos.

Palavras-chaves: Espasticidade Muscular, Paralisia Cerebral, Toxina Botulínica Tipo A

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Conflict of Interest Statement

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Intrathecal Baclofen Pump

The Intrathecal Baclofen pump was described by FENN and associates in 1994 and approved by the FDA for spasticity treatment in 1996. $^{\rm 1,2}$ Baclofen is an agonist of GABA (gamma aminobutyric acid) which minimizes the effects of injured upper motor neurons, depressing the transmission of monosynaptic and polysynaptic reflexes in the spinal cord by stimulation of GABA $_{\rm b}$ receptors, without affecting neuromuscular transmission. $^{\rm 3,4}$

The administration of intrathecal baclofen significantly reduces the amount of medication when compared with usual oral doses, approximately 1% of those without side effects, especially lethargy.⁵⁻⁷ This reduction is possible because the drug is placed directly next to the hyperactive synaptic spinal connections and this also reduces the cognitive side effects that limit the use of drug,⁸ making the drug proportionally more potent when compared to oral administration and with an average life in the cerebrospinal fluid of five hours.²

Such administration is accomplished through the use of an electronic pump, connected to a subcutaneous reservoir, where medication is stored, and a catheter that is placed in the subarachnoid space at an appropriate spinal level,⁷ normally at T11-T12. The pump is remotely programmable to release the drug and the reservoir contains an indicator of the drug level. A percutaneous refill is usually administered every 9-12 weeks and may reach 24 weeks.⁹ Occasionally it is also necessary to change the pump battery, which has average life of 4-7 years.^{2,10}

Inclusion criteria for the baclofen pump's protocol of Westmead Children's Hospital (Sydney, Australia) for the selection of pediatric patients are: severe spasticity, families identified with realistic goals of treatment, patients with at least 15kg of body weight,² who previously used oral anti-spastic medication and had success in the dose test - bolus (50-100µg) - before implanting the pump.² Exclusion criteria are: intracranial pressure greater than 20mm of mercury.¹¹

The baclofen pump, in general, is indicated for patients with severe spasticity, with the aim of reducing or preventing muscle contractures, increasing comfort, improving positioning and facilitating care in non-functional patients when other treatments have failed. The criterion for defining severe spasticity is the patient having grade 3 or higher on the Ashworth scale, indicating significant muscle hyperactivity. The decrease in the Ashworth scale degree from the beginning of treatment with pump will determine the therapeutic outcome related to the bo-

lus infusion of the medication. The pump also allows dose titration during the day, a moment of higher symptoms or increased activity.

The drug dose to be administered depends on the effects and adverse reactions. The typical dose is 50 micrograms administered under monitoring, ranging from 25-100 micrograms / kg / day in a continuous infusion regime, and total daily dose may reach 400-600 micrograms. The dose is adjusted on the first year of use and usually increases during this period. The average life of the drug infused into the cerebrospinal fluid is 5 hours. 2

The use of baclofen pump can be combined to other treatments such as chemical blocks or surgery.¹³ Usually patients with lower limbs spasticity are candidates for this treatment, but it has also been proven useful for upper limbs spasticity.^{6,7}

For a successful implant a prior study of patient response potential is required. Infusion of a medication bolus through a spinal tap serves as a therapeutic test,⁶ although it is not predictive.

The baclofen pump is contraindicated when there is an infection near the implantation site, mental changes, respiratory complications and allergy to oral baclofen.¹³ The presence of convulsions should be carefully evaluated before indicating the pump,¹⁰ since its use can trigger convulsive responses.

Due to the size of the pump its use in young children may be limited,⁶ although there are special pumps for implantation in children younger than 3 years or weighing more than 15kg.⁷

Controlled clinical studies with baclofen pump show that this therapy can reduce muscle hyperactivity and improve function for prolonged periods, moreover improvement in motion range, musculoskeletal function and speed in gait, are described, although the effects on the gait are unpredictable. The pump can also reduce the need for orthopedic surgery in the lower limbs and improve the function of upper limbs.

Albright et al in 2001 showed 86% of improvement in the quality of life range and 33% of improvement in gait velocity, surgical complications occurred in 38% and 26% for drug complications in a series of 77 children treated with baclofen pump.¹⁴

The problems related to the pump include: surgery need for implantation, high cost, infection risk, risk overdose, pump dysfunction, obstruction or displacement of the catheter. Other complications include catheter infection, loss of cerebrospinal fluid, meningitis and the risk of skin injury at the implant site. ^{2,8,15}

Moreover, we can face two situations:

medication abstinence and *overdose*. Abstinence-related symptoms include: itching, dysphoria, irritability, increased spasticity, tachycardia, fever and changes in blood pressure. Lack of treatment for these symptoms may progress to: severe hyperthermia, convulsions, rhabdomyolysis, disseminated intravascular coagulation, altered mental status, psychomotor agitation followed by multiple organ failure and death. The treatment of this condition includes high doses of oral baclofen or infusion by lumbar puncture and intravenous diazepam.

Symptoms related to *overdose* include sedation, nausea, hypotension, urinary and fecal incontinence, convulsions, respiratory depression and coma.² Life risk by escaping the drug (overdose) is a possibility and families should be educated to recognize the symptoms, which include weakness, fatigue, confusion, hypotonia and lethargy. A baclofen overdose due to an error in pump programming is also a possibility, which may lead to excessive sedation, respiratory depression and coma.¹³

The cost of deploying and maintaining a baclofen pump is high,¹ but a recent study of costbenefit analysis showed that in a period of five years, despite an estimated cost increase of U.S. \$49,000 (forty nine thousand U.S. dollars), there is an addition of 1.2 years adjusted to the quality of life, which is considered a good costbenefit relation.⁷ Other drugs such as Fentanyl and Clonidine are being tested through this administration route administration.⁵

In summary, intrathecal baclofen therapy shows long term efficacy in patients with spasticity, inducing a reduced need for orthopedic interventions and promoting rehabilitation. This is a reversible procedure that improves the quality of life of patients and their families and can also reduce the health care costs by reducing the need for treating sequelae that untreated severe spasticity can cause.⁴

According to the 2009 European Consensus for spasticity treatment in children with cerebral palsy update¹⁶ for the baclofen pump we have:

Indication of treatment - from a GMFCS of (III) - IV or V

Goal - spasticity reduction to achieve quality of life. The extent of side effects depends on the experience of the centers where the procedure is performed. Functional Improve: improves the ability to stand, improves mobility and orthoses tolerance. Improves quality of life: simplifies care, decreases pain, improves sleep, reduces sedatives drugs doses, weight gain. Prophylaxis: contractures, sub-dislocation of hips and scoliosis.

Principle - Agonist of the inhibitory GA-BA-B neurotransmitter: Modulation of spinal circuits. Intrathecal administration with a programmed pump via spinal catheter promotes an effective treatment with doses 100-1000 times lower compared to oral administration.

Limitations and controversies - Technical complications and infection. Probable negative influence on scoliosis.

Selective Dorsal Rhizotomy (SDR)

The selective dorsal rhizotomy (SDR) is reserved for a select group of children with spastic diplegia who have significant lower limb spasticity, good strength and selective motor control. ¹⁷ Other authors also recommend that children selected be able to walk, have pure spasticity, age between 3-8 years, intact cognition and good social structure, ^{11,18} moreover it is recommended III or IV degree in the GMFCS (*Gross Motor Function Classification System*).²

The SDR is indicated to reduce spasticity and improve functional gait, besides facilitating quadriplegics care. 11 Ideally, the SDR should be performed before the contracture becomes fixed or that the child requires an extensive program for the deformities correction within the spasticity management. The SDR requires an intensive rehabilitation program and many children may have difficulties due to cognitive and behavioral problems. Studies, controlled and uncontrolled, have shown a permanent reduction of spasticity and improvement in gait after the SDR procedure. On the other hand there are some studies relating the SDR to spine, hips and feet deformities;17 despite this, complications such as sensory modification, bladder and bowel dysfunction, and low back pain are infrequent - roughly 10%.2,6,7

The reasoning which justifies physiologically the making of a SDR is the neurophysiological evidence that spasticity is the result of decreased inhibition from upper motor neuron in the corticospinal tract and interneurons circuit.

The surgical procedure involves a laminoplasty with deafferentation of roots from L2-S2 followed by electrical stimulation of individual sensory roots. The selection of the roots to be sectioned is based on lower extremity muscle response to electrical stimulation.^{2,7,19}

Between 25% and 50% of the dorsal roots are cut, however, despite the sophisticated detection and monitoring in experienced hands, some uncertainties remain about the optimum amount of roots to be operated;¹¹ in addition it is estimated that around 50% of the sensory roots at the SDR level be also sectioned.²

After surgery, the spasticity reduction usually

reveals a muscular weakness and intense physical therapy is needed to establish appropriate motor patterns and strengthening programs.²

A 2002 meta-analysis suggests that the decision to do or not to do the SDR depends on the comparison between the early score in GMFM (*Gross Motor Function Measurement*) with the estimated score after the procedure. This last score must be at least four percentage points above the estimated score for a noninvasive procedure in order to justify the risks, effort and time involved. ^{19,20} This meta-analysis suggests that SDR can result in strength, gait speed, self-care and GMFM scores gains when combined with physical therapy.

The SDR is followed by a spasticity reduction with a clear improvement in the motion arcs and dynamic function of the gait; however this procedure does not affect the selective movement control, balance nor the fixed deformities, which may even worsen after the surgery. We must remember that the spasticity, part of the upper motor neuron syndrome, is not the most important factor in determining the prognosis of children with cerebral palsy; weakness and loss of movement selective control are more important and more difficult to manage.²¹

Orthopedic Surgery

Selection, adjustments and procedures in orthopedic surgery have changed significantly in recent years. Surgeries in the lower limbs should now be deferred until maturity of the gait, which occurs at around 6-10 years,6,7 the results of surgeries performed before 8 years are poor due to the existing growth potential.²²

The introduction of the GMFCS and the FIM had a major impact on surgical indications. Thus, surgeries with functional gait purpose should be performed in children with GMFCS I and II while the surgeries with pain relief purpose should be made in levels IV and V.²³ We know that children with GMFCS V have 65% of chance to develop hip dislocation.

The surgeries should be performed as a single-event multilevel surgery - SEMLS 'i.e., surgery performed at a single time and at multiple levels 11,23 involving derotational osteotomy combined with stretching and tendon transfer on hips, knees and ankles in the same surgery. They are indicated for fixed deformities that result in: motor progress interruption, pain, orthoses use intolerance or difficulties in care, 24 aiming at function improvement, deformities prevention, lessen the pain arising from the sub-luxation or dislocation of the hips, occurrence of pressure

ulcers prevention, positioning improvement, cosmetic and hygiene conditions improvement and facilitate the use of orthoses.²³

The early and aggressive treatment of spasticity by chemical blockage, botulinum toxin type A and / or phenol, oral medications, baclofen pump in combination with physical rehabilitative programs and the use of orthotics and adaptations, has avoided deformities and delayed surgical indications to late childhood. 21,24 At this stage the three-dimensional gait analysis helps to indicate the best surgical procedure. Planning is essential and includes rehabilitational multidisciplinary postoperative programmes, 23 including the use of plaster, bracing, oral antispasmodic medications, botulinum toxin or phenol blocks besides exercise and functional stimulation. 23

The orthopedic surgical options that can influence lower limbs spasticity include stretching of the muscle-tendon unities and tendon transfers. The relief of tension in the muscle-tendon unit decreases the stimulation of muscle spindles, which reduces muscle spasms and spasticity.²⁴

The post-orthopedic surgery reduction of spasticity is usually temporary and cannot be understood as a definitive procedure, it is more used for the treatment of bone deformities and fixed contractures as for the spasticity treatment.²⁴

Orthopedic surgery intervention in the spasticity treatment is limited to selected cases. The surgical indications may include: improvement in function, deformities correction or for cosmetic reasons.²⁵

The orthopedic surgical procedures can be classified into four major categories: 1 - stretching the muscle and tendons or both 2 - tendon transfers, 3 - osteotomies, 4 - arthrodesis.

Tenotomies are indicated to release the tendons of severely spastic muscles, they can be performed in patients without voluntary movement and they have poor results and are more suitable for adductor thigh. Stretching of tendons is made to weaken spastic muscles and position joints in as natural as possible angles. Tendon transfers are used for those muscles that maintain some residual function and can produce useful movements. Stretching and tendon transfers are very useful if the volunteer movements are preserved.²⁵ These procedures are more frequent in the lower extremities and tend to improve the gait development.

Untreated spastic muscles lead to bone deformities and changes in the joint mechanisms. In these cases the osteotomy may occasionally be indicated; in other cases the joint fusion, arthrodesis, may be indicated to place the joints in optimal position.²³

I - Stretching the muscle and tendons or both

This procedure involves the stretching of the muscle tendon units. A tendon can be lengthened by zetaplasty (Fig.1) at the level of the muscle-tendon union or through the fascia recession (Fig. 2). At the tendon stretching there is some spasticity reduction by the change in Golgi receptors and in muscle spindles.²³ We must remember, however, that although the tendon is stretched the muscle fibers may remain shortened, which continues to be deleterious for the muscle. The complications of this procedure are usually iatrogenic including over-stretching with loss of muscle strength.²³

II - Tendon Transfers

Lower limbs: a gait analysis and overall assessment of a spastic child are particularly important when discussing the tendon transfer surgical technique. When spasticity occurs in a group of muscles, the joint responds by causing a flexion through the more active muscle. Through the technique of tendon transfer, the surgeon can relocate the isolated hyperactivity from a spastic muscle to where it can act as an antagonist of another spastic muscle of the same muscular group, adjusting the joint despite the spasticity.²³

The gait analysis guides the priorities for intervention aimed at improving specific parameters of gait. For example, a gait analysis can verify the dynamic response of the joint kinematics and muscle activation in order to plan the surgical options.²⁶

A quadriceps hyperactivity during the swing phase of gait, resulting in a rigid member, may indicate the need for transfering the distal portion of the rectus femoris tendon to the middle-posterior portion of the knee. The rectus femoris will continue to have its spastic nature but in this new position will act by flexing the knee. This will help the knee flexor muscles and will reduce the torque produced by the internal residual quadriceps.²⁶

The equinus foot deformity is commonly seen in individuals with spastic diplegia in cerebral palsy. Spasticity in the gastrocnemius / soleus complex and tibialis posterior causes plantar flexion and ankle inversion. To correct this deformity, the transfer of part of the posterior tibial tendon is indicated in association with Achilles tendon lengthening. By dividing the posterior tibial tendon, the activity of the remaining tissue is reduced. The other portion of the tendon is attached to the distal Achilles, passed through the interosseous membrane and attached to the dorsal part the foot. The increase in tendon length will reduce the tension produced by the

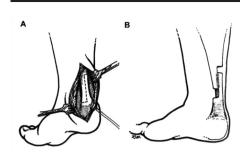


Figure 1 - Zetaplasty of the achilles tendon schematic
A - Intact tendon with surgery marking
B - Stretched and sutured tendon

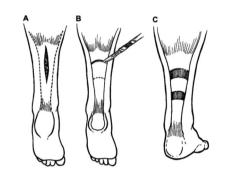


Figure 2 - Procedure over the gastrocnemius-soleus that only lengthens the highest portion of the tendon of the gastrocnemius

gastrocnemius / soleus spastic complex and will allow the joint dorsiflexion. The analysis of gait after surgery shows a decrease in hyperactive dynamic equinovarus deformity in both phases of gait: support and balance.²⁶

This technique, however, may result in failure of the procedure in 10-60% of cases where the primary problem is transformed into a secondary deformity of the calcaneus or into a valgus by overcorrection.²⁵ Another technique involves the division and transfer of half of the tendon to the posterior tibia, reinserting it into the peroneus brevis tendon level.

The procedure of the division and transfer of the tibialis anterior tendon is indicated for the correction of equines-varus posture, to help reduce excessive supination of the sub-talar joint. In this procedure, the anterior tibial tendon distal division is tunneled to the cuboid and cuneiform bones in order to create an eversion force. This surgery is often combined with lengthening of the Achilles tendon.²⁵

Superior Members: The orthopedic procedures for the upper extremities are more controversial. The most typical hand deformity in spastic patients is the palmed thumb. It can be classified in four types:²⁵

Type 1: weakness or paralysis of the thumb's long extensor. Surgical treatment involves the transfer of the palmaris longus tendon or the carpi radialis flexor to the long extensor of the thumb.

Type 2: thumb abductor, flexor pollicis and abductor pollicis brevis spasticity or contraction. Surgery to release the tenar muscle and first dorsal interosseous is recommended to allow forceps movement.

Type 3: abductor pollicis longus weakness or paralysis. The surgery consists in the repositioning of the thumb's long abductor muscle around the flexor carpi radialis and advancing distally its insertion.

Type 4: flexor thumb's spasticity or contracture. The surgery consists of a stretch on Z shape of the thumb's long flexor near the transverse carpal ligament to improve the position of the thumb.

The wrist imbalance is another major problem resulting in function loss of up to 60%. The wrist may take three unnatural positions:

Flexion contracture of the wrist with hyperextension of metacarpophalangeal and proximal interphalangeal flexion: this position can be treated with the division of the flexor carpi radialis tendon and transfer to the short radial extensor.

Ulnar carpus deviation: In this condition the radial carpal extensors are weak and the extensor carpi ulnaris tendon is sub-dislocated on the ulnar styloid process, causing the extensor carpi ulnaris to behave as a functional wrist flexor. This condition can be treated by tendon transfer of extensor carpi ulnaris to the extensor carpi radialis brevis.

Wrist in marked flexion with contraction of all three flexor carpi. In this condition a Z-lengthening is done on the radial, ulnar and long carpal palmar flexor tendons.

We should note that the orthopedic procedures involving the upper extremities are not universally accepted as a tool for functional improvement in patients. The complex actions required by the fingers and hands can be significantly altered by these procedures. An overstretching, however small, on the fingers flexors may result in a decrease in grip strength, so the upper extremities complexity limits the use of surgical transfers on the limb.²⁵ A good initial voluntary control can be a good predictor for surgical outcome in upper limb.²⁷

III - Osteotomies

In some children who walk, rotational abnormalities (increase in the femoral anteversion angle and tibial internal or external torsion) may occur due to change in the lever arms.

All children are born with an increase in the femur's anteversion angle, but normal ambulation leads to a natural derrotation. This mechanism can fail in a child with cerebral palsy and change the biomechanical alignment and gait. In these cases the osteotomy may be indicated. In children unable to walk, the hips dislocation can cause changes in sitting posture, arthritis and pain; in these cases the osteotomy may be indicated for the reduction of dislocation and femoral head coverage. Multiple osteotomies of the tibia, fibula and feet bones can be useful in aligning the ankle and foot.²³

IV - Arthrodesis

In some situations the arthrodesis are indicated to correct and maintain joint alignment. Hip arthrodesis is a rare procedure because it can lead to difficulty in sitting and standing after surgery. Good results are obtained with thumb to the wrist arthrodesis and the first metatarsal to the hallux. Said extra-articular arthrodesis Grice or Green type are still used to stabilize the foot, despite the fact that osteotomy with calcaneus lengthening has been more used for correction of foot valgus. Vertebral arthrodeses are also widely used in children with scoliosis.²³

According to the update of the 2009 European Consensus for the treatment of spasticity in children with cerebral palsy¹⁶ in relation to orthopedic surgical treatments we have:

Treatment Indication - Established for each severity degree. Surgery: as higher level of GM-FCS is the earlier surgery should be considered.

Objective - To fix the misalignment induced by spasticity involving one or more joints (multilevel) to prevent secondary bone deformities. In case of irreversible bone deformities: reconstruction for functional improvement and facilitating care by reducing the possibility of secondary lesions.

Principle - The experience of the pediatric orthopedic surgeon is critical to the decision-making of the team.

Limitations and controversies - Irreversibility, morbidity, repeated surgeries and lack of scientific evidence.

Physical Interventions

The objectives of a physical training program in the spasticity treatment include: maximize function and minimize secondary complications such as muscle contractures, bone deformities and painful muscle spasms during active or passive movements. ^{28,29}

The foundation of spasticity treatment is rehabilitation. Without the family's cooperation and an appropriate therapy program,

whether at home or in rehabilitation centers, the best therapeutic techniques often fail.²⁹

Usually physiotherapy and occupational therapy begin at early treatment stages along with a good household program done by parents and or caregivers. Adding to the stretching program, parents should be instructed to place children in positions that prevent deformities and promote recovery, without traumatizing or giving noxious stimuli that could stimulate spasticity.³⁰ An appropriate program for positioning prevents trauma and preserves the structure of the joints.²⁸

As important as the orientation described above is the training of family members about technical standards to inhibit synergistic patterns that accompany hypertonicity and to facilitate normal patterns of movement. This can help the child to follow the daily activities and incorporate the affected extremities at work, in games and activities for self-care.²⁸

Spasticity limits mobility. Managing tone is one of the points raised in the understanding of the overall treatment of patients with cerebral palsy. The diagram on figure 3 shows the relation between spasticity and other factors that affect mobility and independence in patients with cerebral palsy.³¹

We must remember that treatment with botulinum toxin opens a therapeutical window within the rehabilitational program where the child with cerebral palsy will have an interval of 3-4 months of drug-induced relaxation. The therapeutic window must be used to its fullest in order to maximize effects of botulinum toxin, and for this one must intensify the physiotherapy and occupational therapy programs during this period. Assure to the patient this opportunity is a fundamental condition that must be discussed with family members prior to the procedure of chemical blocking.³²

The spasticity reduction after botulinum toxin injection associated with physical therapy creates a possibility of acquiring new functional patterns;³³ in addition, the biomechanical changes induced in muscle function by botulinum toxin improves the conditions for strengthening and stretching, and this provides an opportunity to strengthen the antagonistic muscles to the injected ones and thus restore the balance between them.³⁰

Passive stretching

In spastic patients was found that muscle strength decreases progressively to the extent that the member is mobilized. A system of daily stretching is part of any program for the comprehensive treatment of spastic patients.

A technique used to reduce spasticity is the passive stretch where the muscle is mobilized through an arc of motion that simulates the normal range of movement. Another technique is the passive strain used in the treatment of patients with contractures; this involves a sustained and prolonged stretch in the desired positions to the point of maximum muscle stretching tolerance. These techniques reduce spasticity by mechanisms little known and for a few hours. ^{6,7,25} The passive stretching can also be obtained through the use of orthoses

Muscular Strengthening

There are controversies about the value of muscle strengthening in the spasticity treatment. Several therapeutic techniques have been suggested, isokinetic or through special equipment, since it is known that the spastic muscle is usually weak.

The "neuro behavioral" technique was developed after observation of neuro physiological behavior in normal children. Bobath recom-

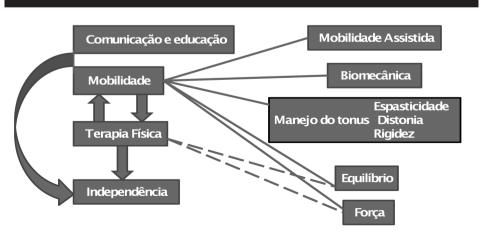


Figure 3 - Relation between spasticity and other factors that affect independence and mobility

mends that the techniques of normal movement and posture correction are impossible in the presence of an abnormal muscle tone and exalted tonic reflexes; its therapeutic intervention principles are based in tonic reflexes inhibition, through inhibitory postures.²⁵

Bobath in particular believes that the counter-resistance strengthening increases muscle tone, which leads to a major objection to the practice of strengthening exercises and exercises with weights in patients with muscle spasticity. In children with cerebral palsy, weight training besides increasing spasticity does not improve function because these patients usually have a lack of muscle selective control at the target muscles. Moreover, despite weakness presence, it does not necessarily contribute to the dysfunction.²⁵

On the other hand, the Kabat and Knott proprioceptive facilitation techniques encourage the training of weak muscles against a therapist resistance with the aim to strengthen muscle groups active in opposing the spastic motion patterns. In spastic adolescents is noted that the isokinetic training results in a strengthening and improvement in gross motor functions. Muscle weakness is an upper motor neuron syndrome component and a strengthening program, encompassing various muscle groups, can contribute to a functional improvement in posture and transfers converging to an overall performance improvement on these patients.^{7,15,25}

According to the 2009 European Consensus for the treatment of spasticity in children with cerebral palsy update¹⁶ in relation to therapy and functional exercise we have:

Treatment Indication - Treatment concomitant to the spasticity treatment performed by qualified therapist.

Objective - To assist the motor development and prevent the development of deformities resulting from spasticity.

Principle - Focusing on the problem related to treatment depending on the disorder's severity: defining goals and targets, perform functional exercises, documenting the changes. Muscle activation immediately after botulinum toxin application, stretch the paretic muscles not injected. Search the muscular balance between agonists and antagonists in all activities of daily life through participation goals. Treatment for when goals are achieved.

Limitations and controversies - Lack of scientific evidence, the concept is only partly based on scientific grounds, there are trends in relation to traditional methods and ideologies.

Miscellaneous physical interventions

Several techniques have been used over the years in the spasticity treatment. *Vibrations* between 100-200Hz applied over the muscles tendons, especially on spastic limbs antagonists, cause a sustained interruption of afferent neurons. For example, vibration applied over the wrist extensors results in a passive contraction of muscles opposing the habitual flexion posture.

The application of local cold and topical anesthetics results in a decrease of spasticity by a decrease in the sensitivity of cutaneous sensory receptors and a decrease in nerve conduction velocity, leading to short periods of increased motor function.

By clinical observation, patient positioning also makes a difference in tone control. The use of weights in the upper extremities in children with cerebral palsy is associated with increased ability to open the hand and standard handgrip improvement. Cranial magnetic stimulation and acupuncture are also described for reducing spasticity.²⁵

Equine therapy

The rehabilitation technique based on the use of horses as therapeutic means. It has been proven useful in the spasticity treatment of children with cerebral palsy, by improving the gross motor function, tone, range of motion, stretching, coordination and balance, reducing the motor impairment degree. Moreover, the equine therapy offers potential cognitive, physical and emotional benefits. 19

Electrostimulation and Biofeedback

Electrostimulation has many uses in neurorehabilitation. It can be used in diagnosis (electromyography) and therapy by reducing the tone or as a form of functional electrical stimulation for muscle contraction when applied on the nerve or motor point.²⁵

The electrical stimulation can also make the effects of botulinum toxin injection more potent.³⁴

The electrical stimulation therapy has been used in reducing severe hypertonicity. The most typical usage is the application on antagonistic muscles, where the stimulation of afferent patterns results in the inhibition of agonist muscles, reducing its tone for a time that can vary from 15 minutes to 3 hours, on average one hour after the procedure. The routine electrical stimulation use is responsible for the contractures decrease and increase in agonist muscles motor activity, besides reducing tone of antagonistic muscles, nonetheless these are short-term effects.

Electrostimulation may also be functionally and dynamically used in assisting muscle strengthening and in the acquisition of functional praxis (eg dorsiflexion stimulation during gait through a functional electrical orthosis – figure 4). The equipment shown in figure 4 consists of a system for dorsiflexor muscle stimulation and a sensor pad with a switch that allows the identification of which moment in gait cycle the member is. It performs a dorsiflexors stimulation during oscillation phase of the gait, allowing easier passage of the limb ahead the trunk; and it goes and off when the calcaneus touches the sensor pad, allowing the relaxation of this muscle group.

Biofeedback

Another form of stimulation that has been used for over 20 years. This type of stimulation consists in the electrical activity of spastic muscles expansion by electronic systems, transforming them into visual or audible signals, which makes easier for the patient to understand the results of contraction and relaxation measurements done during the therapy session. This technique allows an increase of body awareness that results in better control of movement. The positive aspects demonstrated through this technique include improved body image; patients, using this technique, are able to predict how the contraction of inactive muscles would be, improving by that muscle training and providing a better involvement in the rehabilitation program. This type of training, conducted in a regular pattern, may reduce spasticity and occasionally muscle spasms.²⁵Furthermore the involvement and understanding by the patient for this technique is essential. Finally the electrical stimulation (TENS) is a possible tool for the spasticity treatment and for strengthening weak agonist and antagonist muscles. 15,25

Orthoses

Orthoses include all devices applied to members externally that correct abnormal postures caused by gravity or by spastic patterns. There are many reasons for the orthoses use. They can be used simply to compensate paresis of a follow-up member or may also prevent deformities secondary to contractures and reduce pain. Structurally, orthoses can control joint instability; biomechanically, they can change the limb loading to reach the velocity moment below the threshold that must be achieved, before the stretch reflex activity appears in the antagonist muscles. 25

The splints have multiple forms and objectives. They can be designed for active or passive positioning, for inhibition of reflex



Figure 4 - Functional Electrical Orthosis - FES system with sensor pad

patterns, or to produce pressure on specific muscles or tendons areas. They can be static or dynamic. In upper limbs, especially in the region of the wrist and fingers, they can be for palmar or dorsal support. The palmar support is used for positioning and should be observed in order not to produce an increase in flexor spasticity. 15,25

The static orthoses are often effective in keeping the length of the muscles involved, when a muscle is passively stretched and in the elongation process occurs a decrease in sensitivity of stretch reflex, with reduction of spasticity. 15,28

The musculature forced into a passive stretching will undergo anatomical and physiological changes, as an example the adding of sarcomeres to muscle fibers and stretch of the connective tissue elements. The spasticity reduction induced by the use of splints may extend for up to three hours after its removal, allowing the use of the affected limb in functional activities.²⁸

Dynamic orthoses are used to improve the biomechanical aspects, besides helping in stabilizing the posture allowing a functional control of movement.²⁸ This type of bracing for wrist and hand, allows some freedom to the joint. Usually the dynamic orthoses combine palmar and dorsal aspects of the splints.²⁵

The ostheses are commonly used on lower extremities, especially for the needs required in gait. The first short leg braces were made of rigid materials, with a fixed ankle. The latest guidance is that they be constructed with lightweight materials and meet the needs of individual patients according to their characteristics of movement.²⁵

The dynamic lower limb orthoses improve function during gait by allowing some ankle dorsiflexors mobility during different phases of normal gait.^{15,28}

AFO (ankle-foot ortheses) that have floor reaction are preferable to fixed apparatuses on the approach of equinus foot posture, besides facilitating the change from seated to standing

position in children at the pre-gait phase.⁷

According to the 2009 European Consensus for the treatment of spasticity in children with cerebral palsy update¹⁶ for ortheses we have:

Treatment Indication - depends on the national standards, interdisciplinary, continuous cooperation with orthopedic surgeon and infant child orthesist

Objective - To improve function and participation, prevention and / or reduction of muscle contractures (muscle and bone deformities), decrease the incidence of surgery.

Principle - Limbs: functional improvement, maintenance, optimization of the functional reserves. Body: stabilization and support.

Limitations and controversies - Lack of scientific evidence, tack, without international standardization, variability between different concepts in centers rehabilitation at the same country.

Plaster

Plastic plasters can be considered an extension of extremity splints. The theoretical basis for the plaster use in spasticity treatment, lies in the inhibitory response of autogenic Ib afferents fibers from Golgi tendon organs. For example, every time the plaster inhibits the flexor spastic contraction of a region, it can decrease the area's flexor tone. Another theory is that the heat and weight of the cast also contribute to the reduction of tone, however we must be careful with the possibility of neurovascular compromise in this procedure.²⁵ Plasters in spastic patients promote a steady and prolonged muscle stretch that can also be progressive, useful in patients with large static spasticity component.¹⁵

The plaster has been extensively used in children with cerebral palsy as an adjunctive treatment for spasticity. In this group, plaster is used for maintaining the optimal positioning of the joints, facilitating functionality and reducing spasticity. Plasters that reduce the tone are applied to produce stretch in hypertonic muscles while functionally positioning treated extremities.²⁸ Serial casting, with exchange every 7-10 days and repeat 5 times, decrease muscle tone and seem to be more effective in controlling soft tissue contractures resulting from spasticity.²⁵

We must remember that some movement control and spasticity degree decrease should be observed during the use of casts, otherwise the probability of maintaining or improving range of motion afterwards, by braces and splints, will be small. Before using plaster it is necessary to take into account the patient's cognitive function, as well as his/hers skin condition and sensory loss.²⁵ The plaster can also be used in con-

junction with chemical blocks, botulinum toxin and / or phenol. 15

CONCLUSION

Spasticity is a complex clinical manifestation that requires deep understanding of intrinsic mechanisms and pathologies that cause it. An extensive range of therapeutic modalities for its treatment is available. The treatment must be individualized depending on the location, severity and duration of symptoms. The therapeutic goals must be established before starting treatment and must be understood and shared by patients and their families.

On the other hand we must remember the family members of our spastic patients that despite the large range of procedures available for spasticity treatment, the rehabilitational program based in kinesiotherapy cannot be dismissed. Patients who leave the rehabilitative programs do not enjoy the benefits of individual treatments for spasticity control.

When we treat a child with cerebral palsy we must ask ourselves what should and should not be done in childhood in order to preserve mobility and function in the stages of adolescence and adulthood. It is critical that individuals with cerebral palsy have their physical capabilities preserved and enhanced, through tone management, maintenance of motion range, posture correction and judicious use of surgery strategies to preserve muscle strength and incoming sensory stimuli.

Moreover, the application of biomechanical principles, physiological exercises and motor control associated with the current regeneration knowledge, neuronal restoration and transformation changed intervention strategies based on sound science. A more accurate diagnosis and new preventive approaches to primary and secondary aggravating factors, combined with technological advances in rehabilitative apparatus also have great potential to improve the health parameters and most importantly quality of life in patients with cerebral palsy.

REFERENCES

- Jacobs JM. Management options for the child with spastic cerebral palsy. Orthop Nurs. 2001;20(3):53-9.
- Matthews DJ, Balaban B. Management of spasticity in children with cerebral palsy. Acta Orthop Traumatol Turc. 2009;43(2):81-6.

- Chambers HG. Advances in Cerebral Palsy. Curr Opin Orthop. 2002; 13(6): 424-31.
- Téllez de Meneses M, Barbero P, Alvarez-Garijo JA, Mulas F. Intrathecal baclofen and Botulinum toxin in infantile cerebral palsy. Rev Neurol. 2005;40(Suppl 1):S69-73.
- Elovic E. Principles of pharmaceutical management of spastic hypertonia. Phys Med Rehabil Clin N Am. 2001;12(4):793-816, vii.
- Tilton AH. Management of spasticity in children with cerebral palsy. Semin Pediatr Neurol. 2004;11(1):58-65.
- Tilton A. Management of spasticity in children with cerebral palsy. Semin Pediatr Neurol. 2009;16(2):82-9.
- Patel DR, Soyode O. Pharmacologic interventions for reducing spasticity in cerebral palsy. Indian J Pediatr. 2005;72(10):869-72.
- Tilton AH. Therapeutic interventions for tone abnormalities in cerebral palsy. NeuroRx. 2006;3(2):217-24.
- Mooney JF 3rd, Koman LA, Smith BP. Pharmacologic management of spasticity in cerebral palsy. J Pediatr Orthop. 2003;23(5):679-86.
- Flett PJ. Rehabilitation of spasticity and related problems in childhood cerebral palsy. J Paediatr Child Health. 2003;39(1):6-14.
- 12. Kopec K. Cerebral palsy: pharmacologic treatment of spasticity. US Pharm. 2008; 33(1):22-6.
- 13. Brashear A, Lambeth K. Spasticity. Curr Treat Options Neurol. 2009;11(3):153-61.
- Albright AL, Barry MJ, Shafton DH, Ferson SS. Intrathecal baclofen for generalized dystonia. Dev Med Child Neurol. 2001;43(10):652-7.
- Papavasiliou AS. Management of motor problems in cerebral palsy: a critical update for the clinician. Eur J Paediatr Neurol. 2009;13(5):387-96.
- Heinen F, Molenaers G, Fairhurst C, Carr LJ, Desloovere K, Chaleat Valayer E, et al. European consensus table 2006 on botulinum toxin for children with cerebral palsy. Eur J Paediatr Neurol. 2006;10(5-6):215-25.
- Rodda J, Graham HK. Classification of gait patterns in spastic hemiplegia and spastic diplegia: a basis for a management algorithm. Eur J Neurol. 2001;8(Suppl 5):98-108.
- Gormley ME Jr. Treatment of neuromuscular and musculoskeletal problems in cerebral palsy. Pediatr Rehabil. 2001;4(1):5-16.
- Sharan D. Recent advances in management of cerebral palsy. Indian J Pediatr. 2005;72(11):969-73.
- McLaughlin J, Bjornson K, Temkin N, Steinbok P, Wright V, Reiner A, et al. Selective dorsal rhizotomy: meta-analysis of three randomized controlled trials. Dev Med Child Neurol. 2002;44(1):17-25.
- Graham HK; Selber P. Musculoskeletal aspects of cerebral palsy. J Bone Joint Surg, 2003; 85B (2): 157-66.
- Ward AB, Molenaers G, Colosimo C, Berardelli A. Clinical value of botulinum toxin in neurological indications. Eur J Neurol. 2006;13 (Suppl 4):20-6.
- Damiano DL, Alter KE, Chambers H. New clinical and research trends in lower extremity management for ambulatory children with cerebral palsy. Phys Med Rehabil Clin N Am. 2009;20(3):469-91.
- Gormley ME Jr, Krach LE, Piccini L. Spasticity management in the child with spastic quadriplegia. Eur J Neurol. 2001;8 Suppl 5:127-35.
- 25. Jozefczyk PB. The management of focal spasticity. Clin Neuropharmacol. 2002;25(3):158-73.
- Yaggie J, Armstrong W. Spastic diplegic cerebral palsy: a brief introduction to its characteristics, assessment and treatment options. Clin Kinesiol. 2001;55(4):75-80.
- Boyd RN, Morris ME, Graham HK. Management of upper limb dysfunction in children with cerebral palsy: a systematic review. Eur J Neurol. 2001;8 Suppl 5:150-66.
- Ong HT, Chong HN, Yap SSP. Comprehensive management of spasticity in cerebral palsy: role of physical therapy and other adjunctive treatments. Singapore Paediatr J. 2001; 43(4):133-6.
- Tilton AH. Approach to the rehabilitation of spasticity and neuromuscular disorders in children. Neurol Clin. 2003;21(4):853-81, vii.

- 30. Ward AB. Spasticity treatment with botulinum toxins. J Neural Transm. 2008;115(4):607-16.
- 31. Brunstrom JE, Clinical considerations in cerebral palsy and spasticity. J Child Neurol. 2001;16(1):10-5.
- Molenaers G, Desloovere K, Eyssen M, De Cat J, Jonkers I, De Cock P. Botulinum toxin type A treatment of cerebral palsy: an integrated approach. Eur J Neurol. 1999; 6:(Suppl 4): S51-7.
- 33. O'Brien CF. Treatment of spasticity with botulinum toxin. Clin J Pain. 2002;18(6 Suppl):S182-90.
- Yablon SA. Botulinum neurotoxin intramuscular chemodenervation. Role in the management of spastic hypertonia and related motor disorders. Phys Med Rehabil Clin N Am. 2001;12(4):833-74, vii-viii.