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Management of Spasticity and Cerebral Palsy

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1. Introduction

Spasticity was defined by Lance as a “velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex” (Young RR, 1994; Francisco GE, Ivanhoe CB, 1997).

Young further added characteristics of positive and negative symptoms. Positive symptoms consist of exaggerated cutaneous reflexes, including nociceptive and flexor withdrawal reflexes, autonomic hyperreflexia, dystonia, and contractures. Negative symptoms include paresis, lack of dexterity, and fatigability (Young RR, 1994).

Treatment for spasticity was documented as early as the late 19th century, when surgeons Abbe and Bennet discussed decreasing tone in a spastic limb through sensory rhizotomies. Later, in 1898, the scientist Sherrington published experiments in which the sensory roots of spastic cats were severed to relieve spasticity (Abbott R, 1996).

The technique of sensory rhizotomies has been improved on and continues to be used today as a treatment for patients with spasticity as does neuromuscular blockage, a longstanding treatment, which has been used for over 30 years (Koman LA, Mooney JF, Smith BP, 1996).

1.1. Cerebral palsy

Cerebral palsy (CP) is a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior, by epilepsy, and by secondary musculoskeletal problems (Glinac A, Tahirović H, Delalić A, 2013).

The research *Frequency of joined disabilities of children with cerebral palsy in Tuzla canton* covers a total sample of 48 examinees, chronological age from 2-19 years, in Tuzla Canton. Research instrument was a Structural Questionnaire for the parents of children and adolescents with cerebral palsy. Research data were processed by nonparametric statistics method. Basic statistical parameters of frequency and percentages were calculated, and tabular presentation was made. After classification of examinees as per frequency of joined disabilities was done, work results have shown that speech impairment occurred with 35.4 % of children, visual impairment 33.3 %, epilepsy 29.3 %, whereas hearing impairment occurred with 2 % of children (Babajić M, Švraka E, Avdić D, 2013).

Although there are many possible causes of spasticity, this chapter will focus on children with spasticity, most of whom have diagnoses of cerebral palsy; approximately two thirds of all cerebral palsy patients suffer from spasticity (Albright AL, 1996).

A patient with spastic cerebral palsy presents with muscle imbalance, stands with bent knees and legs tightly together, and in severe cases, a scissors-type gait (Frerebeau PH, et al, 1991; Adams RD, Victor M, Ropper AH, 1997). The antigravity muscles are predominantly affected with arms in a flexed and pronated position and legs in an extended and adducted position. When the muscles are at rest they are flaccid to palpation and electromyographically silent.

Spasticity can be associated with cocontraction, clonus and hyperreflexia. Children with spastic cerebral palsy generally have a typical pattern of muscle weakness, impairment in selective motor control and sensory impairment (Mikov A, Dimitrijević L, Sekulić S, Demešič-Drljan Č, Mikov I, Švraka E, Knežević-Pogančev M, 2011).

Many children with more severe spastic CP experience *communication problems* due to disturbed neuromuscular control of speech mechanism, i. e. dysarthria, that diminish the ability of the child to speak intelligibly. However, substantial dysarthria are most often seen in children with severe CP and intellectual disability, while most children with mild and moderate CP and average cognitive level of functioning have normal or near-normal expressive language and articulation skills (Bottcher, 2010).

1.2. Etiology and epidemiology

Spasticity may result from either diffuse or localized pathology of the cerebral cortex, brain stem, or spinal cord. Possible causes of such injuries include traumatic brain injury, stroke, multiple sclerosis, spinal cord trauma, or disease and anoxic insults. The neurologic localization of the lesion causing spasticity may result in different clinical manifestations. Thus, it is important to consider whether the spasticity results from cerebral pathology, whether it is diffuse or localized, or whether it is a result of spinal cord injury.

Diffuse cerebral injury or diseases would include anoxia, toxic, or metabolic encephalopathy, where as *localized cerebral injury* would include tumor, abscess, cyst, arteriovenous malformations, hemorrhage, or trauma.

Spinal cord injury or disease may result as an insult to descending pathways by trauma, inflammatory or demyelinating disease, degenerative disorders, or compression such as is caused by a tumor or cyst (Albright AL, 1996; Frerebeau PH, et al, 1991; Dimitrijevic MR, 1991).

The annual incidence of spinal cord injuries in the United States is estimated to be 30 to 40 new cases per million individuals. About 3% to 5% of cases each year occur in children younger than 15 years of age (Price C, Makintubee S, Herndon W, Istre GR, 1994).

The male-to-female ratio of patients is 4:1 in the general population, but in younger age groups, the ratio is approximately 1.5:1 (Zidek K, Srinivasan R, 2003).

According to the time of insult, causes of *cerebral palsy* can be divided to prenatal (from conception till beginning of delivery), perinatal (started from beginning of labor till end of neonatal period; first 28 days of life) and postnatal (from 29th day of age until two years of age). The majority of international studies indicates that the prevalence of cerebral palsy is about 2-2,5 cases per 1000 born, although there are some reports about lower and higher prevalence rates. Majority of previous research in the world was focused on the prevalence, determination of the motor abilities, and perinatal etiological factors of the cerebral palsy. Evidences indicated that 70-80 % of cerebral palsy is caused by the prenatal factors and that the birth asphyxia has a relatively minor role with the less than 10 % (Švraka E, 2012).

Common causes of *cerebral palsy in children* that may result in spasticity are prolonged second stage labor, fetal distress, cystic degeneration of the brain, prematurity, periventricular encephalomalacia, cortical abnormalities such as porencephaly, or congenital malformations of gyri such as micropolygyria.

Through the last decades, marked improvement in the level of intensive care at Neonatal Intensive Care Units (NICU) which was reflected on an increase in the survival of very low birth-weight (VLBW) and extremely low birth-weight (ELBW) premature newborns. New risk factors have appeared among infants who previously would have died, and the incidence of neurodevelopmental impairments in survivors of NICU is higher than in normal birth-weight newborns. In particular, due to the high risk of interventricular haemorrhage and periventricular leukomalacia, an increasing prevalence of cerebral palsy has occurred in premature, low birth-weight newborns and children born with asphyxia (Švraka E, 2012).

Spasticity is present in about two thirds of cerebral palsy patients, and cerebral palsy affects anywhere from 1.5 to 2.5 per 1000 live births in the United States (Adams RD, Victor M, Ropper AH, 1997).

The number of spastic patients continues to increase due to an increased survival rate of premature births. Males and females are equally affected.

1.3. Pathogenesis and pathophysiology

There are many different types of spasticity. Because of this, more than one mechanism may be responsible for the disturbance in muscle tone and the mechanisms may vary between patients. The neuropathophysiologic processes involved in spasticity are complex and not fully understood, but there is a widely accepted hypothesis that spasticity depends on

hyperexcitability of spinal alpha motor neurons, which is due to the interruption of descending modulatory influences carried by the corticospinal, vestibulospinal, and reticulospinal tracts and other possible tracts (Filloux FM, 1996).

Ia afferent fibers provide segmental input from muscle spindles to alpha motor neuron pools. They synapse on segmental inhibitory interneurons that then inhibit alpha motor neurons innervating antagonist muscles in the Ia reciprocal inhibition pathway. Ib afferents inhibit alpha motor neurons by way of the Golgi tendon organs via the Ib inhibitory interneuron in another pathway known as nonreciprocal inhibition (Young RR, 1994; Filloux FM, 1996).

Increased excitation of these afferents does not seem to be the cause of spasticity. Instead, evidence supports that reduced reciprocal inhibition of antagonist motor neuron pools by Ia afferents, decreased presynaptic inhibition of Ia afferents, and decreased nonreciprocal inhibition by Ib terminals are all possible pathophysiologic mechanisms of spasticity (Young RR, 1994).

The pathophysiology of traumatic brain injury involves a complex combination of forces that has been a subject of substantial debate (Drew LB. and Drew WE, 2004).

On occasion, *autonomic dysreflexia* may occur after an intramuscular injection, although this is relatively rare (Selcuk B, Inanir M, Kurtaran A, Sulubulut N, Akyuz M, 2004).

In some patients, autonomic dysreflexia may occur even if the level of spinal injury is below T6 (Blackmer J, 2003; Krassioukov AV, Furlan JC, Fehlings MG, 2003).

1.4. Diagnostic procedure

Examination should begin with the patient in a relaxed, lying position with the head up and arms resting to the sides because it is easier to determine the extent of spasticity in this position. The examination should include tonic stretch reflexes by manual passive stretches, elicitation of tendon jerks and clonus in a relaxed position, and tonic and phasic stretch reflexes carried out in a sitting position.

The manual passive stretch maneuver is used to assess resistance at different rates. A joint is passively moved while the muscles corresponding to that joint are lengthened and shortened. In cases of mild spasticity, the muscles will only resist when stretched at a high rate, whereas in cases of moderate spasticity, resistance is noticed at a slower rate and the clasp-knife phenomenon may be exhibited. Movement of the muscle may be difficult to impossible in cases of severe spasticity (Dimitrijevic MR, 1991).

Tendon jerks are easier to elicit in spastic patients than in patients with normal muscle tone, and reflex responses can be achieved in muscles without well-defined tendons. Percussion of the tendon reveals hyperactive tendon jerks, especially for the Achilles, patellar, biceps, and triceps tendons (Zidar J, Dimitrijevic MR, 1991).

Measurement of resistance to passive stretch, reduction in the tonic vibration reflex, and reduction of the plantar withdrawal reflex should also be evaluated. Motoneuronal overactivity should also be evaluated because any input to motoneurons produces excessive and

prolonged activity that can be observed in the contractions of many limb muscles (Zidar J, Dimitrijevic MR, 1991).

The amount of function the patient derives from spasticity can be evaluated by having the patient obtain and maintain standing and seated positions. To determine the degree to which the hamstring tone is affecting the alignment of the pelvis and knees, have the child sit with feet straight in front. The patient can sit in a chair to allow the examiner to assess trunk control. The side sit position exhibits a patient's ability to maintain control in an asymmetric position (Abbot R, 1991).

Modified Ashworth Scale (MAS) has been used as a diagnostic test for spasticity. Testing can be done to establish the presence of any lesion or brain or spinal cord injury. The muscle tone is graded according to the *Modified Ashworth scale (MAS)*, a scale ranging from 1 to 5, in which resistance to the passive muscle stretch is measured at various velocities; MAS: 0 = No increase in muscle tone, 1 = Slight increase in the muscle tone, manifested by catch and release or minimal resistance at the end of the range of motion, 2 = more marked increase in the muscle tone through most of the range of motion, but affected parts are easily moved, 3 = considerable increase in the muscle tone, and passive movements are difficult, 4 = affected parts are rigid in flexion or extension (Albright AL, 1996).

MRI of the brain can be performed to rule out periventricular leukomalacia.

On EMG, the jerks show greater amplitudes than are normal and are followed by after-discharge of the motor units that is often slightly longer lasting than normal. The size of tendon jerks can be measured by either EMG response or by recordings of mechanical events.

H-reflex studies are electrically elicited tendon jerks and are restricted mostly to the soleus and flexor carpi radialis muscles in normal adults. In cases of upper motor neuron lesions, the H-reflex may be elicited in muscles where it is not normally seen, such as the intrinsic hand muscles, tibialis anterior, or peroneal muscles (Zidar J, Dimitrijevic MR, 1991).

A baseline EEG to establish underlying seizure activity can also be done as well as basic lab studies. *Neurophysiological studies*, such as the H-reflex study, may be performed in patients with neurodegenerative disease; an enzymatic assay should also be performed.

Video cameras are often helpful during evaluation as the patient's movements can be recorded and compared against movements during and after treatment.

Test	Use
Basic lab studies	Metabolic derangement
Enzymatic assay	Neurodegenerative disease
EEG	Underlying seizure activity
NCS	Neurodegenerative disease (Leukodystrophies)
MRI of the brain	Periventricular leukomalacia

Table 1. Useful Tests for Diagnosis

1.5. Differential diagnosis

Spasticity can be confused with rigidity when a patient is being evaluated. Stretching can distinguish rigidity from spasticity. Rigidity will relax through repeated stretching of a muscle, whereas a spastic muscle will continue to increase in resistance as the velocity of the stretch is increased (Young RR, 1994; Dimitrijevic MR, 1991).

1.6. Quality of life

In families who have children with CP the “constant attendance” of the disease is present, through strict consistent long-term care of family and many other factors, such as services, support and physical aspects of the environment, which all can lead to deterioration of the patient’s quality of life (Glinac A, Tahirović H, Delalić A, 2013).

The study *Family quality of life: adult school children with intellectual disabilities in Bosnia and Herzegovina*, provides initial data for family quality of life in Bosnia and Herzegovina (B&H). It also provides suggestions for improving quality of life for families that have one or more members with intellectual disability (ID). The principle measure used was the *Family Quality of Life Survey 2006 – main caregivers of people with intellectual or developmental disabilities*. The sample consisted of the main caregivers in 35 families that have adult children 18 years and over with ID who attended classes in a specially adapted programme in the Centre for children with ID, autism and cerebral palsy (n = 16), and in the Vocational Secondary School, B&H (n = 19). Regarding diagnosis as reported by main caregivers, 15 sons or daughters had ID of unknown aetiology, eight had cerebral palsy, four had Down syndrome, four had epilepsy and another three had epilepsy as a co-morbidity, two had autism and two had Prader-Willi syndrome. One had a dual diagnosis, ID and mental illness. When asked to rate overall family quality of life, three said ‘excellent’, eight said ‘very good’, 16 said ‘good’, seven said ‘fair’ and one said ‘poor’. Furthermore, when asked to rate their overall satisfaction with their family quality of life, two said ‘very satisfied’, 19 said ‘satisfied’ and 13 said ‘neither satisfied nor dissatisfied’ (Švraka E, Loga S, Brown I, 2011).

Spasticity results in limited functional capacity and increased inactivity. The sequelae of this inactivity may include decubiti, cardiovascular problems, thrombophlebitis, respiratory infections, fixed contractures, osteoporosis, bladder and bowel problems, and social isolation. Ultimately, these consequences of inactivity may lead to a further decrease in strength and function (Francisco GE, Ivanhoe CB, 1997).

The patient’s *quality of life* may be compromised as spasticity has negative impacts on mobility, hygiene, self care, sleeping patterns, self esteem, mood, and sexual function.

It is important to evaluate the advantages and disadvantages that the patient gains from their spasticity so that treatment strategies and goals can be identified. Disadvantages may include interference with activities of daily living, inhibition of good sleep, contractures, dislocations, skin breakdown, bowel and bladder dysfunction, impairment of respiratory function, pain with stretching, and the masking of the return of voluntary movement. However, patients may rely on a certain amount of spasticity to function and the advantages they may receive include

maintaining muscle tone, supporting circulatory function, assisting in activities of daily living, and preventing the formation of deep vein thrombosis.

2. Case study of two children with CP

2.1. Patient A

Patient A was a 5-year-old African-American boy with a history of developmental delay and a diagnosis of cerebral palsy of the spastic-diplegic type. He first presented at 18 months with severe spasticity in both lower extremities. Prior to treatment with botulinum toxin, the patient walked on tip toes and had hip and knee flexion. There was some scissoring of his legs. On *examination*, exaggerated deep tendon reflexes were elicited, as were sustained clonus and bilateral Babinski sign. MRI of the brain showed findings that may be secondary to previous hypoxic injury, compatible with cerebral palsy.

Prior *treatments* included physical therapy, bilateral ankle-foot orthosis, serial casting, and oral baclofen. This boy with spastic-diplegic cerebral palsy walked on tip toes until treatment with botulinum toxin injections.

Following botulinum toxin injection, at the age of 18 months, the patient's gait has improved; he is flat-footed and presently wears bilateral ankle-foot orthosis. His hygiene and positioning have also improved and he returns every 6 months to 9 months for reinjection.

Results of the study *Use of Botulinum toxin type a in children with Spastic Cerebral Palsy*, support the idea that younger children may receive more benefit from multilevel botulinum toxin type A injections, intensive physiotherapy and appropriate orthotic management compared to older children. Younger children might have been able to maintain the functional gains because the motor pattern of very young children provides greater scope for better development and recovery. A younger child has greater potential than older child for increasing the plasticity of the central nervous system. Botulinum toxin type A injections should always be used as an adjunctive treatment to physiotherapy, occupational therapy and orthotic management. In combination with post-injection physiotherapy this treatment could provide long-term benefits (Mikov A, Dimitrijević L, Sekulić S, Demeši-Drljan Č, Mikov I, Švraka E, Knežević-Pogančev M, 2011).

2.2. Patient B

Patient B was a 7-year-old African-American boy with a history of cerebral palsy of the spastic-diplegic type. On primary examination he presented with tightness of both hamstrings and heel cords with the right more involved than the left. The patient had good toe standing, especially on the right side and good sitting balance with a kyphotic sacral-type sitting due to the tight hamstring. He uses a walker to ambulate and walks on tip toes. The EEG was abnormal, indicating the presence of epileptiform activity from the left central parietal head region and diffuse background disorganization, which indicates underlying neuronal dysfunction.

Treatments before *intrathecal baclofen pump* implantation included bilateral ankle-foot orthoses, tendon releases, alcohol block, and botulinum toxin injections. Before treatment with intrathecal baclofen the patient was dependent on a care giver and used a walker to ambulate. With the intrathecal baclofen pump the patient has gained function, does not use a walker to ambulate, and performs activities of daily living independently. With the intrathecal baclofen pump the patient has gained function, does not use a walker to ambulate, and successfully performs activities of daily living.

3. Spasticity management

Traditional treatments for spasticity include physical therapy, occupational therapy and rehabilitation treatments which complete a number of crucial tasks and specific goals in the treatment of patient with CP, this will promote their sensorimotor development, improve their overall posture and position and enhance their control of movements in all their daily activities: a lot of physical therapy approaches were based on different theoretical principles though the main target is the management of abnormal muscle tone and improving the range of motion through neurodevelopment therapy, conductive education, constraint induced movement therapy, etc.

There are other modalities including electrical stimulation and cold temperature (Chiara T, Carlos J Jr, Martin D, Miller R, Nadeau S, 1998; Pease WS, 1998; Scheker LR, Chesher SP, Ramirez S, 1999; Kinnman J, Andersson T, Andersson G, 2000).

Occupational therapy is a client-centered health profession concerned with promoting health and well being through occupation. Possible problems in children with cerebral palsy are motor, sensor, cognitive, intrapersonal, interpersonal, problems of self care, productivity and leisure.

Occupational therapy, in which the patient is stretched anywhere from once daily to several times per day, but this has only a limited effect on the patient's spasticity. Rehabilitation treatment options include casting, orthotics or splints, strengthening, electrical stimulation, practice of functional tasks, sensory integration; muscle stretching, and targeted muscle training (Fetters L, Kluzik J, 1996).

Within the scope of pediatric neurorehabilitation, distinct diseases can produce specific complications. These complications; however, can also occur in association with many disorders. For example, spasticity from injury to the upper motor neuron unit can develop in many neurologic disorders in children. Several of these complications, such as autonomic dysreflexia, deep vein thrombosis, and heterotopic ossification, can be severe and potentially life-threatening (Umphred D, Dewane J, Hall-Thompson M, et al, 2001; Dobkins, BH, 2003; DeLisa JA, Gans BM, Walsh NE, Bockneck WL, Frontera WR, 2004).

3.1. Oral medications

Oral medications can be used to decrease spasticity; however, many have unwanted side effects such as drowsiness, sedation, confusion, and fatigue. Benzodiazepines, such as diazepam, are rarely used because of their strong sedating effects. They result in enhanced presynaptic inhibition, but because they are presumed to enhance the postsynaptic effects of GABA, they can only work if the GABA-mediated process functions. Benzodiazepines have a long half-life and an active metabolite. Benzodiazepine therapy is indicated in spinal cord injury and multiple sclerosis with possible application in traumatic brain injury, cerebral palsy, and cerebrovascular accident. Clinical effects include sedation and reduced anxiety, decreased resistance to passive range of motion, decreased hyperreflexia, and reduction in painful spasms. Side effects of all benzodiazepines include sedation, weakness, hypotension, gastrointestinal symptoms, memory impairment, incoordination, confusion, depression, and ataxia. Also, benzodiazepines are controlled substances with the potential for dependency. Diazepam is the most widely used benzodiazepine for spasticity management. The recommended initial dose is 2 mg 3 times daily with a maximum dose of 60 mg daily (20 mg 3 times daily). If nocturnal spasticity is the presenting problem the patient should be started with a single dose at night.

Like benzodiazepines, baclofen works centrally. Baclofen binds with GABA-B receptors on brain and spinal membranes, restricting calcium influx into presynaptic nerve terminals, thereby reducing spasticity [4]. The use of baclofen is indicated when spasticity is of spinal origin. The clinical effects include decreased resistance to passive range of motion, decrease in hyperreflexia, and reduction in painful spasms and clonus.

Unlike benzodiazepines and baclofen, dantrolene sodium works peripherally at the level of the muscle fiber. It has no effect on neuromuscular transmission, but works by acting directly on the skeletal muscle, hindering the release of calcium from the sarcoplasmic reticulum, thereby preventing the excitation-contraction coupling mechanism. This affects both intrafusal and extrafusal fibers by decreasing the force of muscle contraction. However, this mechanism is not selective for muscles with increased tone, and the resulting generalized muscle weakness may weaken respiratory muscles. The use of dantrolene sodium is indicated in treating spasticity secondary to cerebrovascular accident, cerebral palsy, and has possible applications for traumatic brain injury, spinal cord injury, and multiple sclerosis. Clinical effects of dantrolene sodium include decreased resistance to passive range of motion, decrease in hyperreflexia and tone, and reduction in spasms and clonus.

Another group of oral medications used in spasticity management includes clonidine and tizanidine, which are alpha 2 noradrenergic receptor agonists that release excitatory neurotransmitters and inhibit supraspinal facilitatory pathways (Young RR, 1994; Francisco GE, Ivanhoe CB, 1997).

Tizanidine is a new oral antispasticity agent that is selective in decreasing tone and spasm frequency in only spastic muscles, eliminating the unwanted side effect of generalized muscle weakness. Tizanidine is reported to have reduced symptoms of spasticity in patients with multiple sclerosis or spinal cord injury and is well tolerated in most patients. It is an imida-

zoline derivative similar to clonidine but without the cardiovascular effects when appropriately titrated. Tizanidine results in a direct reduction of excitatory amino acid release from spinal interneurons and inhibits facilitatory caeruleospinal pathways. Its peak effect occurs 1 to 2 hours following administration and its half-life is 2.5 hours. The clinical effects of tizanidine include reduced muscle tone, spasm frequency, and hyperreflexia. Animal studies with tizanidine demonstrate antinociceptive activity under specific conditions with increased dose titration (McCarthy RJ, Kroin JS, Lubenow TR, Penn RD, Ivankovich AD, 1990).

As with other antispasticity medications, the potential side effects of tizanidine are dose related and may be mitigated by dosage titration. The potential side effects include drowsiness, dry mouth, and dizziness. Literature suggests that tizanidine may be better tolerated than other antispasticity agents as measured by the global tolerance rating scale (Lataste X, Emre M, Davis C, Groves L, 1994).

In placebo-controlled studies, tizanidine has been shown to be effective in multiple sclerosis and spinal cord injury. It is also useful for spasticity of spinal pathology when weakness is of concern. Tizanidine may also prove effective in managing spasticity of cerebral origin (Medici M, Pebet M, Ciblis D, 1989).

Secondary oral and systemic agents include tiagabine, cyproheptadine, clonidine, lamotrigine, gabapentin and carbidopa-levodopa (Gracies JM, Nance P, Elovic E, et al, 1997).

Multiple medications have been recommended, of which the most recent addition is gabapentin (Zidek K, Srinivasan R, 2003).

The use of antihypertensive pharmacologic agents in treating spasticity is unclear because randomized trials have not been performed. Nifedipine has been used in a bit-and-swallow technique; more recently, captopril also has been found to be of benefit (Esmail Z, Shalansky KF, Sunderji R, Anton H, Chambers K, Fish W, 2002).

3.2. Chemo-denervation

Chemo-denervation such as using botulinum toxin type A, has proved easier, more effective, and less painful for patients. First clinically introduced in the United States in the early 1980s, botulinum toxin is a potent neurotoxin derived from the anaerobic bacteria *Clostridium botulinum*, but when used in treatment, no serious systemic toxin effects have been reported (Francisco GE, Ivanhoe CB, 1997).

The medication is more costly than alcohol or phenol but the cost is offset by less physician time and the lack of anesthesia. The formation of antibodies has been a concern, but this can be prevented by allowing 2 months to 3 months between injections. Botulinum toxin works by acting in the neuromuscular junction, preventing the release of acetylcholine, which results in functional denervation. It can be given without EMG and anesthesia, does not cause dysesthesias, and is no more painful than an injection of saline solution. Effects are local and last 3 months to 4 months, or longer. It is contraindicated during pregnancy, lactation, in individuals with neuromuscular disorders (such as myasthenia gravis), in patients taking aminoglycosides, or in those who have a known allergy to the drug. Adverse effects are not

common and are usually associated with the site of injection, such as bleeding, bruising, and soreness or redness at the injection site, or diffusion to nearby muscle groups. In patients that do not respond to botulinum toxin, possible reasons should be considered before labeling the patient as unresponsive. Reasons could be related to injection technique, improper toxin storage, or the patient's individual characteristics. Overall, botulinum toxin has proven clinically to be effective, safe, and less painful than other invasive therapies (Francisco GE, Ivanhoe CB, 1997; Keam SJ, Muir VJ, Deeks ED, 2011).

Botulinum toxin is available in serotypes A and B, which have different unit potencies, side-effect profiles, and dilution schedules. Both have been used in children with cerebral palsy, although serotype A has been used more extensively. Dosing guidelines have been suggested for botulinum toxin A for adult and pediatric patients. Adult recommendations are available for botulinum toxin B, but studies are ongoing for pediatric patients (Tilton AH, 2003; Schwerin A, Berweck S, Fietzek UM, Heinen F, 2004; Sanger TD, Kukke SN, Sherman-Levine S, 2007).

Some results suggest that botulinum toxin type A can be effective in reducing muscle tone over a longer period, but not in preventing development of contractures in spastic muscles. Mechanical and functional alterations can arise from the muscle tissue itself even though the nervous system is the site of the primary lesion. The gross mechanical changes occur in skeletal muscle secondary to spasticity and during development of contracture. Muscle stiffness can change for a variety of structural reasons, only one of which is altered fiber length. There is currently no evidence in the literature that muscle fiber length is shortened in contracture or in spastic skeletal muscle. Contracture formation results from inappropriate architectural adaptation of extremity muscles in response to upper motor neuron lesion (Mikov A, Dimitrijević L, Sekulić S, Demeši-Drljan Č, Mikov I, Švraka E, Knežević-Pogačev M, 2011).

Several studies have reported the successful use of botulinum toxin A for the treatment of drooling in children with cerebral palsy, using injection into the submandibular or parotid glands alone or in combination with other agents. In some studies, the beneficial effects have lasted for up to 4 months without serious side effects or disturbances of oral function (Jongerius PH, van den Hoogen F, van Limbeek J, Gabreels FJ, van Hulst K, Rotteveel JJ, 2004; Bothwell JE, Clarke K, Dooley JM, et al, 2002; Suskind DL, Tilton A, 2002).

Other treatments include *chemical neurolysis*, in which the nerve conduction is impaired through the use of chemical agents and therapeutic nerve block using phenol or alcohol. The goals of these treatments are to prevent muscle contractures and improve the patient's function. A common side effect is that after the nerve is injected, alcohol levels measure above the legal limit in children. Other side effects include damage to sensory and motor nerves, pain at injection site, scarring, and dysesthesias. To ensure the correct site, injection must be made using an electrical stimulator (Albright AL, 1996; Francisco GE, Ivanhoe CB, 1997).

3.3. Neurosurgical approaches

Another treatment used alleviate spasticity in children with cerebral palsy is *rhizotomy*. Studies have shown that performing selective dorsal rhizotomy at a young age can reduce the need for orthopedic surgery (Chicoine MR, Park TS, Kaufman BA, 1997).

Goals of rhizotomy are decreased tone, increased mobility, and the facilitation of care for the patient, however, the reduction in spasticity cannot be predicted and sometimes results in excessive hypotonia (Im D, McDonald CM, 1997).

The procedure is very meticulous, requiring general anesthesia and a neurophysiologist who must be present to identify which nerve is to be severed.

Other *neurosurgical approaches* include peripheral neurectomy, myelotomy, and dorsal column electrical stimulation.

It has been established that oral baclofen does not cross the blood-brain barrier effectively and that higher doses of the medication result in serious side effects (Francisco GE, Ivanhoe CB, 1997).

Intrathecal baclofen results in a greater decrease in spasticity by allowing higher concentrations of baclofen in the cerebrospinal fluid at about 1% the daily oral dosage (Im D, McDonald CM, 1997).

To be considered for intrathecal baclofen pump placement, the patient must have severe lower limb spasticity that does not respond to other less-invasive treatments. The patient must first be given a trial of 50 µg baclofen through a lumbar puncture or spinal catheter. If unresponsive, 75 µg can be tried after 24 hours and a third trial of 100 µg can be tried 24 hours after that, after which if the patient is still unresponsive he or she must be excluded from the treatment (Francisco GE, Ivanhoe CB, 1997).

Implantation lasts 1 to 2 hours and the pump is easy to refill subcutaneously. It is programmed by a computer-controlled radiotelemetry programmer that is linked to the pump's internal computer and that selects the rate and pattern of baclofen administration. Complications to intrathecal baclofen include hypersensitivity to baclofen, intolerance to the side effects of baclofen including drug tolerance, cerebrospinal fluid leakage, pump pocket seroma, hematoma, infection, and soft tissue erosion. The objective of intrathecal baclofen is to individualize the patient's dose and infusion so that the lowest dose that yields the greatest response can be achieved (Young RR, 1994; Francisco GE, Ivanhoe CB, 1997).

In comparison, intrathecal baclofen has less complications and side effects than other treatments and more generalized results in both cerebral and spinal spasticity, making intrathecal baclofen the most effective current tool for the treatment of spasticity in non-ambulant individuals. A recent systematic review showed that there was no evidence to support the clinical use of intrathecal baclofen in ambulant individuals with hypertonicity without further rigorous longitudinal studies (Pin TW, McCartney L, Lewis J, Waugh MC, 2011).

As a precaution, families are prescribed diazepam or diazepam rectal as well as oral baclofen to have at home. If there is evidence of withdrawal, one of these medications is administered, and the patient is instructed to go immediately to the emergency department. Although aggressive use of benzodiazepines and oral baclofen may be helpful, recognition and return to appropriate intrathecal baclofen dosage is essential for rapid recovery (Alden TD, Lytle RA, Park TS, Notzel MJ, Ojemann JG, 2002).

3.4. Orthopedic procedures

Orthopedic procedures are the most frequently performed operations for spasticity. The targets of these operations are muscles, tendons, or bones. Muscles may be denervated and tendons and muscles may be released, lengthened, or transferred. The goals of surgery may include reducing spasticity, increasing range of motion, improving access for hygiene, improving the ability to tolerate braces, or reducing pain. Orthopedic problems that may result from a spastic limb include cubital or carpal tunnel syndrome, spontaneous fracture, dislocation of the hip or knee, and heterotopic ossification.

The most common orthopedic procedure for the treatment of spasticity is a *contracture release*. In this procedure, the tendon of a muscle that has a contracture is partially or completely cut. The joint is then positioned at a more normal angle, and a cast is applied. Regrowth of the tendon to a new length occurs over several weeks. Serial casting may be used to gradually extend the joint. Following cast removal, physical therapy is used to strengthen the muscles and improve range of motion.

Spastic muscles in the shoulder, elbow, forearm, hands, and legs may all be treated with tendon or muscle lengthening. Spasticity in the shoulder muscles may cause abduction or adduction and internal rotation of the shoulder. Abduction results in difficulties with balance, which then affects walking and transferring, and adduction causes problems when reaching for an object or with hygiene and personal care. An operation known as a slide procedure may be used to lengthen the supraspinatus muscle in an abducted spastic shoulder. With adducted shoulders, the surgeon can perform a release of all 4 muscles that typically cause this deformity.

In an operation known as a tendon transfer, the orthopedic surgeon moves a tendon from the spot at which it attaches to the spastic muscle. With the tendon transferred to a different site, the muscle can no longer pull the joint into a deformed position. In some situations, the transfer allows improved function. In others, the joint retains passive but not active function. Ankle-balancing procedures are among the most effective interventions.

The goal of surgical-orthopedic treatment which is basically symptomatic improve or facilitate the movement to solve the functional or fixed contractures preventing further rehabilitation, to solve the deformation that reduces or prevents movement, sitting, causing pain as in the cases of hip luxation, or threaten respiration as in cases of severe scoliosis. Subluxation and dislocations of the hip in children with CP are most common in children and adolescents who do not walk. We must bear in mind the saying that every child and adolescent with CP has a hip disorder until proven otherwise. The occurrence of dislocation of the hips makes furniture, hygiene and often causes pain. Requires regular radiological controls hips once or twice a year in the course of growth, to hip dislocation discovered at an early stage. Subluxation and luxation of the hips treated surgically. The decision about surgery should bring those involved in the treatment of patients, carefully weighing hopper performs coarse benefits and harms of surgery. Surgery is necessary to balance the muscle forces around the hip and normalize abnormal anatomic relationships (Đapić T, Šmigovec I, Kovač-Đapić N, Polovina S, 2012).

Osteotomy and arthrodesis involves operations on the bones and are usually accompanied by operations to lengthen or split tendons to allow for fuller correction of the joint deformity.

Osteotomy can be used to correct a deformity that cannot be fixed with other procedures. In an osteotomy, a small wedge is removed from a bone to allow it to be repositioned or reshaped. A cast is applied while the bone heals in a more natural position. Osteotomy procedures are most commonly used to correct hip displacements and foot deformities. Arthrodesis is a fusing together of bones that normally move independently. This fusion limits the ability of a spastic muscle to pull the joint into an abnormal position. Arthrodesis procedures are performed most often on the bones in the ankle and foot. In triple arthrodesis, the 3 joints of the foot are exposed, the cartilage is removed, and screws are inserted into the bones, fixing the joints into position. With a short walking cast in place for 6 weeks or until the bones have fully healed, the patient may bear weight immediately after the operation (http://wemove.org/spa/spa_oss.html 2007).

The risks of developing a structural spinal deformity ranges from 24% to 36% for scoliosis and is 50% for lordosis for an average of 4 to 11 years after selective dorsal rhizotomy (Turi M, Kalen V, 2000; Johnson M, Goldstein L, Thomas SS, Piatt J, Aiona M, Sussman M, 2004).

Other principals include single event, multilevel surgery; surgery is delayed as long as possible (more than 6 years). Spasticity management is used as an adjunct to surgical intervention (Boyd R, Graham J, Natras G, Graham K, 1999).

3.5. New treatments in spasticity management

Acupuncture and homeopathic approaches (Guo Z, Zhou M, Chen X, Wang R, 1997), herbs and hyperbaric oxygen [41-45], constraint induced training [46, 47], the Adeli suit [48], conductive education, craniosacral, and manipulation and patterning.

Context therapy is a new intervention approach that focuses on changing the task and the environment rather than children's impairments. It can be a viable treatment to achieve parent-identified functional goals for children with cerebral palsy (Darrah J, Law MC, Pollock N, et al, 2011).

A summary of management in spasticity is provided in Table 2.

Therapeutic intervention	Mechanisms	Major points
Nonpharmacologic treatments		
Physical therapy	Neurodevelopmental therapy (NDT)	Different techniques are tailored depending on the individual goals
Occupational therapy	Constraint-induced movement therapy (CIMT)	
	Neurophysiologically based therapy/ Vojta	Training of both manual and fine motor skills of the paretic side through activity limitation of the healthy side
	Manual medicine	
	Training of the muscular strength	Reflex locomotion to encourage motor development through repetitive triggering reflex creeping and reflex turning
	Treadmill therapy	
	Conductive education	
	Hand-arm bimanual intensive therapy (HABIT)	Encouraging motor learning through active and passive mobilization, soft tissue release and manipulations
	Sensory integration therapy/Ayres	Encouraging locomotion and posture through specific training of certain muscle groups

Therapeutic intervention	Mechanisms	Major points
		Gait training through walking on treadmill, with body weight support Systematic, intensive training of small learning steps in the motor, linguistic and cognitive domains Motivation for bimanual activity of the paretic and nonparetic side with specified tasks Everyday tasks training for coordination and sensory information enhancement
	Splints, strengthening, electrical stimulation, practice of functional tasks, muscle stretching, and targeted muscle training	Mainstays and cornerstones in spasticity management; complications, such as autonomic dysreflexia, deep vein thrombosis, and heterotropic ossification, can be severe and potentially life-threatening
Casting and orthosis	Extend joint range diminished by hypertonicity; reduce an abnormal pattern by positioning	Temporary effect
Selective posterior rhizotomy	Balancing spinal cord-mediated facilitatory and inhibitory control	Permanent effect; sometimes results in excessive hypotonia
Orthopedic surgery	Corrects deformity induced by muscle overactivity involving muscles, tendons, or bones	In moderate to severe spasticity, permanent effect
Pharmacological treatments, oral medications		
Benzodiazepine	Increases the affinity of GABA for GABA-A receptors; inhibitory effect at both the spinal cord and supraspinal levels	Short-term treatment; strong sedating effects
Dantrolene sodium	Inhibits release of calcium from sarcoplasmic reticulum in muscle; works peripherally at the muscle fibers	Serious side effects; hepatotoxicity in 1% patients, respiratory muscle weakness
Baclofen	GABA agonist; binds at the GABA-B receptor; restricts calcium influx into presynaptic nerve terminals in the spinal cord	Rapidly absorbed after oral administration; levels in the CSF are low because of low lipid solubility
Tizanidine	Centrally acting alpha-2 noradrenergic agonist; inhibits release of excitatory neurotransmitters in the spinal cord and supraspinally	Drowsiness, dry mouth, and dizziness; monitor liver function
Pharmacological treatments, chemodenervation		
Alcohol/Phenol block	Nonselective proteolytic agents; selective denervation when injecting into motor nerves or muscles	Damage to sensory and motor nerves, painful dysesthesias
Botulinum toxin injection	High affinity and specificity to the presynaptic membranes of cholinergic motor neurons	Recommended as effective treatment; no sensory disturbance
Pharmacological treatments, other		
Intrathecal baclofen pump	Using a programmable implanted pump, baclofen can be delivered intrathecally	Severe, generalized spasticity; less complications and side effects

Table 2. Management in Spasticity

Pregnancy

The patient with spasticity may expect to have a difficult pregnancy and delivery as well as difficulty managing and caring for an infant.

Anesthesia

Not applicable.

4. Conclusion

To prevent cerebral palsy in infants and, thus, the resulting spasticity, it is important that mothers receive prenatal care during pregnancy, that measures are taken to avoid premature labor, and that special consideration is given to pregnancies involving multiple gestations.

Early detection and treatment of neurodegenerative diseases may prevent the development of spasticity as well as detect the underlying diseases that could result in brain injury. If children have conditions that make them susceptible to brain or spinal cord injury or both, safety measures should be taken (i.e., helmets for patients who have frequent seizures).

The goals of and benefits to the patient are important when considering the path of treatment. In some cases, function will not return, but treatment can result in pain reduction and allow easier management of patient care. Common goals are to decrease pain, prevent or decrease contractures, improve ambulation, facilitate activities of daily living, facilitate rehabilitation participation, save caregiver's time, improve the ease of care, and increase safety. Appropriate management choices are based on therapeutic objectives. Physical and occupational therapists can play a key role in identifying these objectives. Treatments with the fewest side effects are usually given priority. Both the patient's and the caregiver's goals must be considered.

Rehabilitation multidisciplinary team could be good connection with Management. There are different approaches in rehabilitation treatment of persons with cerebral palsy, especially children and adolescents. The treatment of children with spastic cerebral palsy is a combination of intensive sensorimotor stimuli, physical therapy, occupational therapy, Vojta therapy, orthopedic procedures and/or botulinum toxin applications. It is child/family-centered management.

The ICF can guide management but does not give sufficient detail of the "hows and whys" of the child activities to enable a specific treatment plan.

5. Summary

Spasticity may result from either diffuse or localized pathology of the cerebral cortex, brain stem, or spinal cord. Possible causes of such injuries include traumatic brain injury, stroke, multiple sclerosis, spinal cord trauma, or disease and anoxic insults. The neurologic localiza-

tion of the lesion causing spasticity may result in different clinical manifestations. Thus, it is important to consider whether the spasticity results from cerebral pathology, whether it is diffuse or localized, or whether it is a result of spinal cord injury.

Cerebral palsy (CP) is a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior, by epilepsy, and by secondary musculoskeletal problems

Spasticity can be associated with cocontraction, clonus and hyperreflexia. Children with spastic cerebral palsy generally have a typical pattern of muscle weakness, impairment in selective motor control and sensory impairment.

It is important to evaluate the advantages and disadvantages that the patient gains from their spasticity so that treatment strategies and goals can be identified. Disadvantages may include interference with activities of daily living, inhibition of good sleep, contractures, dislocations, skin breakdown, bowel and bladder dysfunction, impairment of respiratory function, pain with stretching, and the masking of the return of voluntary movement.

There are many different types of spasticity. Because of this, more than one mechanism may be responsible for the disturbance in muscle tone and the mechanisms may vary between patients. The neuropathophysiologic processes involved in spasticity are complex and not fully understood, but there is a widely accepted hypothesis that spasticity depends on hyperexcitability of spinal alpha motor neurons, which is due to the interruption of descending modulatory influences carried by the corticospinal, vestibulospinal, and reticulospinal tracts and other possible tracts.

Traditional treatments for spasticity include physical therapy, occupational therapy and rehabilitation treatments which complete a number of crucial tasks and specific goals in the treatment of patient with CP, this will promote their sensorimotor development, improve their overall posture and position and enhance their control of movements in all their daily activities: a lot of physical therapy approaches were based on different theoretical principles though the main target is the management of abnormal muscle tone and improving the range of motion through neurodevelopment therapy, conductive education, constraint induced movement therapy, etc.

Oral medications can be used to decrease spasticity; however, many have unwanted side effects such as drowsiness, sedation, confusion, and fatigue. Benzodiazepines, such as diazepam, are rarely used because of their strong sedating effects. They result in enhanced presynaptic inhibition, but because they are presumed to enhance the postsynaptic effects of GABA, they can only work if the GABA-mediated process functions.

Chemo-denervation such as using botulinum toxin type A, has proved easier, more effective, and less painful for patients. First clinically introduced in the United States in the early 1980s, botulinum toxin is a potent neurotoxin derived from the anaerobic bacteria *Clostridium botulinum*, but when used in treatment, no serious systemic toxin effects have been reported.

Another treatment used to alleviate spasticity in children with cerebral palsy is *rhizotomy*. Studies have shown that performing selective dorsal rhizotomy at a young age can reduce the need for orthopedic surgery. Goals of rhizotomy are decreased tone, increased mobility, and the facilitation of care for the patient, however, the reduction in spasticity cannot be predicted and sometimes results in excessive hypotonia.

Other *neurosurgical approaches* include peripheral neurectomy, myelotomy, and dorsal column electrical stimulation.

Orthopedic procedures are the most frequently performed operations for spasticity. The targets of these operations are muscles, tendons, or bones. Muscles may be denervated and tendons and muscles may be released, lengthened, or transferred. The goals of surgery may include reducing spasticity, increasing range of motion, improving access for hygiene, improving the ability to tolerate braces, or reducing pain. Orthopedic problems that may result from a spastic limb include cubital or carpal tunnel syndrome, spontaneous fracture, dislocation of the hip or knee, and heterotopic ossification.

Abbreviations

EEG: electroencephalogram

EMG: electromyography

MRI: Magnetic Resonance Imaging

ICD codes

ICD-9:

Abnormal involuntary movements: 781.0

ICD-10:

Other and unspecified abnormal involuntary movements: R25.8

Associated disorders

Adrenoleukodystrophy

Anoxia

Cerebral palsy

Multiple sclerosis

Neurodegenerative disease

Spinal cord injury

Stroke

Traumatic brain injury

Major keyword descriptors

bent knees

gait disturbances

muscle imbalance

poor hygiene

poor positioning

scissors-type gait

stretch reflexes

tendon jerks

Minor keyword descriptors

bladder problems

bowel problems

cardiovascular problems

fixed contractures

osteoporosis

pain

respiratory infections

thrombophlebitis

Glossary

Adrenoleukodystrophy: demyelination of nerve cells in the brain and progressive dysfunction of the adrenal gland.

Anoxia: diminished supply of oxygen to an organ's tissues.

Cerebral palsy: Nonprogressive disorder of movement and posture that can occur anywhere from 0 to 5 years of age, caused by a brain lesion.

Clasp-knife phenomenon: characterized by a free interval of movement of the limb, followed by a sudden stop and increase in muscle resistance which melts away as the passive stretching of the limb continues.

Multiple Sclerosis: plaques form from inflammation of the white matter of the central nervous system, causing destruction of the myelin sheath, resulting in diminished or lost function.

Permuted topics, synonyms, variants

Spasticity

Related topics

Acupuncture

Autosomal dominant inherited ataxias

Baclofen

Cerebral palsy

Childhood ataxia with central nervous system hypomyelination

Childhood movement disorders

Neurodegeneration with brain iron accumulation

Hyperargininemia

Hyperbaric oxygenation for the treatment of stroke

Machado-Joseph disease

Multiple sclerosis

Nonautosomal dominant inherited ataxias

Sjogren-Larsson syndrome

Differential diagnosis

Rigidity

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References

- [1] Abbott, R. (1996) Sensory rhizotomy for the treatment of childhood spasticity. *J Child Neurol* 11 (suppl1): S36-42.

- [2] Abbott, R. (1991) Childhood spasticity assessment. In: Sindou M, Abbott R, and Kera-vel Y, editors. *Neurosurgery for spasticity: a multidisciplinary approach*. Wien; New York: Springer-rlag:51-6.
- [3] Adams, RD; Victor, M. & Ropper, AH. (1997) Motor paralysis: cardinal manifesta-tions of neurologic disease. In: Adams RD, Victor M, Ropper AH, editors. *Principles of neurology*. Vol 6. New York: McGraw Hill:54-6.
- [4] Albright, AL. (1996) Spasticity and movement disorders in cerebral palsy. *J Child Neurol*;11(suppl 1):S1-4.
- [5] Akman, MN; Loubser, PG; Fife, CE, et al. (1994) Hyperbaric oxygen therapy: implica-tions for spinal cord injury patients with intrathecal baclofen infusion pumps. *Para-plegia*; 32:281-4.
- [6] Alden, TD; Lytle, RA; Park, TS; Notzel, MJ. & Ojemann, JG. (2002) Intrathecal baclo-fen withdrawal: a case report & review of the literature. *Child Nerv Syst*; 18(9-10): 522-5.
- [7] Babajić, M; Švraka, E. & Avdić, D. (2013) Frequency of joined disabilities of children with cerebral palsy in Tuzla canton. *Journal of Health Sciences*;3(3): 222-226
- [8] Blackmer, J. (2003) Rehabilitation medicine: I. Autonomic dysreflexia. *CMAJ*; 169:931-5.
- [9] Bothwell, JE; Clarke, K; Dooley, JM, et al. (2002) Botulinum toxin A as a treatment for excessive drooling in children. *Pediatr Neurol*;27(1):18-22.
- [10] Bottcher, L. (2010). Children with spastic cerebral palsy, their cognitive functioning, and social participation: a review. *Child Neuropsychology*, 16: 209-228.
- [11] Boyd, R; Graham, J; Nattras, G. & Graham, K. (1999) Medium-term response charac-terization and risk factor Analysis of botulinum toxin type A in the management of spasticity in children with cerebral palsy. *Eur J Neur (Suppl 4)*:S37-45.
- [12] Chiara, T; Carlos, J Jr; Martin, D; Miller, R. & Nadeau, S. (1998) Cold effect on oxygen uptake, perceived exertion, and spasticity on patients with multiple sclerosis. *Arch Phys Med Rehabil*; 79:523-8.
- [13] Chicoín, MR; Park, TS. & Kaufman, BA. (1997) Selective dorsal rhizotomy and rates of orthopedic surgery in children with spastic cerebral palsy. *J Neurosurg* ;86:34-9.
- [14] Chung, CY; Chen, CL. & Wong, AM. (2011) Pharmacotherapy of spasticity in chil-dren with cerebral palsy. *J Formos Med Assoc*;110(4):215-22.
- [15] Collet, JP; Vanasse, M; Marois, P, et al. (2001) Hyperbaric oxygen for children with cerebral palsy: a multicenter, placebo controlled, randomized clinical trial. *Lancet*; 357:582-6.

- [16] Crocker, MD; MacKay-Lyons, M. & McDonnell, E. (1997) Forced use of the upper extremity in cerebral palsy: a single-case design. *Am J Occup Ther*; 51:824-33.
- [17] Darrah, J; Law, MC; Pollock, N. et al. (2011) Context therapy: a new intervention approach for children with cerebral palsy. *Dev Med Child Neurol*; 53(7):615-20.
- [18] Dimitrijevic, MR. (1991) Clinical assessment of spasticity. In: *Neurosurgery for spasticity: a multidisciplinary approach*. New York: Springer-Verlag: 33-7.
- [19] DeLisa, JA; Gans, BM; Walsh, NE; Bockneck, WL. & Frontera, WR. (2004) *Physical medicine and rehabilitation: principles and practice*. Philadelphia: Lippincott Williams & Wilkins.
- [20] Dobkins, BH. (2003) *The clinical science of neurologic rehabilitation*. London: Oxford University Press.
- [21] Drew, LB. & Drew, WE. (2004) The contrecoup-coup phenomenon: A new understanding of the mechanism of closed head injury. *Neurocrit Care*; 1(3):385-90.
- [22] Đapić, T; Šmigovec, I; Kovač-Đapić, N. & Polovina, S. (2012) Surgery of cerebral palsy with special reference to treatment spastic luxation of the hip. *Paediatrics Today*; 8 (Suppl 2) : 20-30 ISSN 1840-0914 (Print) ISSN 1840-2968 (Online)
- [23] Esmail, Z; Shalansky, KF; Sunderji, R; Anton, H; Chambers, K. & Fish, W. (2002) Evaluation of captopril for the management of hypertension in autonomic dysreflexia: a pilot study. *Arch Phys Med Rehab*; 83(5):604-8.
- [24] Fetters, L. & Kluzik, J. (1996) The effects of neurodevelopmental treatment vs practice on reaching of children with spastic cerebral palsy. *Phys Ther*; 76:346-58.
- [25] Filloux, FM. (1996) Neuropathophysiology of movement disorders in cerebral palsy. *J Child Neurol*; (suppl 1):S5-12.
- [26] Francisco, GE. & Ivanhoe, CB. (1997) Pharmacologic management of spasticity in adults with brain injury. In: Kraft GH, Horn LJ, editors. *Physical medicine and rehabilitation 8:4*. Philadelphia: WB Saunders Company: 707-31.
- [27] Frerebeau, PH. et al. (1991) Clinical feature of spasticity. In: *Neurosurgery for spasticity: a multidisciplinary approach*. New York: Springer-Verlag: 29-32.
- [28] Glinac, A; Tahirović, H. & Delalić, A. (2013) Family socioeconomic status and health-related quality of life in children with cerebral palsy: assessing differences between clinical and healthy samples. *Paediatrics Today*; 9(2):183-191. DOI 10.5457/p2005-114.74
- [29] Gracies, JM; Nance, P; Elovic, E. et al. (1997) Traditional pharmacological treatments for spasticity part II: general and regional treatments. *Muscle Nerve*; 20(suppl 6):S92-120.

- [30] Guo, Z; Zhou, M; Chen, X. & Wang, R. (1997) Acupuncture methods for hemiplegic spasm. *J Tradit Chin Med*; 17(4):284-8.
- [31] Im, D. & McDonald, CM. (1997) New approaches to managing spasticity in children with cerebral palsy. *West J Med*; 166(4):271.
- [32] Johnson, M; Goldstein, L; Thomas, SS; Piatt, J; Aiona, M. & Sussman, M. (2004) Spinal deformity after selective dorsal rhizotomy in ambulatory patients with cerebral palsy. *J Pediatr Orthop*; 24(5):529-36.
- [33] Jongerius, PH; van den Hoogen, F; van Limbeek, J; Gabreels, FJ; van Hulst, K. & Rotteveel, JJ. (2004) Effect of botulinum toxin in the treatment of drooling: a controlled clinical trial. *Pediatrics*; 114(3):620-7.
- [34] Keam, SJ; Muir, VJ. & Deeks, ED. (2001) Botulinum toxin A (Dysport®): in dystonias and focal spasticity. *Drugs*; 71(8):1043-58.
- [35] Koman, LA; Mooney, JF. & Smith, BP. (1996) Neuromuscular blockage in the management of cerebral palsy. *J Child Neurol* ; 11(suppl1):S23-8.
- [36] Kinnman, J; Andersson, T. & Andersson, G. (2000) Effect of cooling suit treatment in patients with multiple sclerosis evaluated by evoked potentials. *Scand J Rehabil Med*; 32:16-9.
- [37] Kluger, J. (2001) The root of tranquility: is extract of kava a natural substitute for valium—or just alternative medicine's newest herb du jour? | {website:Time Website}{webURL:<http://www.time.com/time/>} |
- [38] Krassioukov, AV; Furlan, JC. & Fehlings, MG. (2003) Autonomic dysreflexia in acute spinal cord injury: an under-recognized clinical entity. *J Neurotrauma*; 20(8):707-16.
- [39] Lataste, X; Emre, M; Davis, C. & Groves, L. (1994) Comparative profile of tizanidine in the management of spasticity. *Neurology*; 44(suppl 9):53-9.
- [40] McCarthy, RJ; Kroin, JS; Lubenow, TR; Penn, RD. & Ivankovich, AD. (1990) Effect of intrathecal tizanidine on antinociception and blood pressure in the rat. *Pain*; 40(3):333-8.
- [41] Medici, M; Pebet, M. & Ciblis, D. (1989) A double-blind, long-term study of tizanidine ('Sirdalud') in spasticity due to cerebrovascular lesions. *Curr Med Res Opin*; 11(6):398-407.
- [42] Mikov, A; Dimitrijević, L; Sekulić, S; Demeši-Drljan, Č; Mikov, I; Švraka, E. & Knežević-Pogančev M. (2011) Use of Botulinum toxin type a in children with Spastic Cerebral Palsy. *HealthMED, Journal of Society for development of teaching and business processes in new net environment in B&H. Published by DRUNPP, Sarajevo. Vol.5, No 4, p. 922-928 ISSN 1840-2291*

- [43] Montgomery, D; Goldberg, J; Amar, M. et al. (1999) Effects of hyperbaric oxygen therapy on children with spastic diplegic cerebral palsy: a pilot project. *Undersea Hyperb Med*; 26:235-42.
- [44] Panteliadis, CP. (2011) *Cerebral Palsy, A multidisciplinary approach*. ISBN: 978-3-87185-403-3
- [45] Pease, WS. (1998) Therapeutic electrical stimulation for spasticity: quantitative gait analysis. *Am J Phys Med Rehabil*; 77:351-5.
- [46] Pin, TW; McCartney, L; Lewis, J. & Waugh, MC. (2011) Use of intrathecal baclofen therapy in ambulant children and adolescents with spasticity and dystonia of cerebral origin: a systematic review. *Dev Med Child Neurol*; 53(10):885-95.
- [47] Pittler, MH. & Ernst, E. (2000) Efficacy of kava extract for treating anxiety: systematic review and meta-analysis. *J Clin Psychopharmacol*; 20:84-9.
- [48] Price, C; Makintubee, S; Herndon, W. & Istre, GR. (1994) Epidemiology of traumatic spinal cord injury and acute hospitalization and rehabilitation charges for spinal cord injuries in Oklahoma, 1988-1990. *Am J Epidemiol*; 139(1):37-47.
- [49] Sanger, TD; Kukke, SN. & Sherman-Levine, S. (2007) Botulinum toxin type B improves the speed of reaching in children with cerebral palsy and arm dystonia: an open-label, dose-escalation pilot study. *J Child Neurol*; 22(1):116-22.
- [50] Scheker, LR; Chesher, SP. & Ramirez, S. (1999) Neuromuscular electrical stimulation and dynamic bracing as a treatment for upper-extremity spasticity in children with cerebral palsy. *J Hand Surg [Br]*; 24:226-32.
- [51] Schwerin, A; Berweck, S; Fietzek, UM. & Heinen, F. (2004) Botulinum toxin B treatment in children with spastic movement disorders: a pilot study. *Pediatr Neurol*; 31(2):109-13.
- [52] Selcuk, B; Inanir, M; Kurtaran, A; Sulubulut, N. & Akyuz, M. (2004) Autonomic dysreflexia after intramuscular injection in traumatic tetraplegia: a case report. *Am J Phys Med Rehabil*; 83(1):61-4.
- [53] Stephens, K, editor. (2000) Poland: space suit technology offers new hope for cerebral palsy rehab. | {Website: Orthotic and Prosthetic Business News} {webURL: <http://www.oandpbiznews.com>} | [serial online]. Spring 1998;1(2). Accessed December 15, 2000.
- [54] Suskind, DL. & Tilton, A. (2002) Clinical study of botulinum-A toxin in the treatment of sialorrhea in children with cerebral palsy. *Laryngoscope*; 112(1):73-81.
- [55] Švraka, E; Loga, S. & Brown, I. (2011) Family Quality of Life: Adult school children with intellectual disabilities in Bosnia and Herzegovina. *Journal of Intellectual Disability Research*. The Foremost International Journal on Intellectual Disability. Volume

55 part twelve. Special Issue Part One: Family Quality of Life. Edited by Ralph Kober and Mian Wang. ISSN 0964-2633 (Print) ISSN 1365-2788 (Online)

- [56] Švraka, E. (2012). Children with Cerebral Palsy and Epilepsy, Epilepsy - Histological, Electroencephalographic and Psychological Aspects, Dr. Dejan Stevanovic (Ed.), ISBN: 978-953-51-0082-9, InTech, Available from: <http://www.intechopen.com/books/epilepsy-histological-electroencephalographic-andpsychological-aspects/children-with-cerebral-palsy-and-epilepsy>.
- [57] Taub, E; Miller, NE; Novack, TA. et al. (1993) Technique to improve chronic motor deficit after stroke. Arch Phys Med Rehabil; 74:347-53.
- [58] Tilton, AH. (2003) Injectable neuromuscular blockade in the treatment of spasticity and movement disorders. J Child Neurol; 18:S50-66.
- [59] Turi, M. & Kalen, V. (2000) The risk of spinal deformity after selective dorsal rhizotomy. J Pediatr Orthop; 20(1):104-7.
- [60] Umphred, D; Dewane, J; Hall-Thompson, M. et al. (2001) RMU model for neurological rehabilitation, Provo, UT.
- [61] WE MOVE: Worldwide Education and Awareness for Movement Disorders. Orthopedic operations. Available at: http://wemove.org/spa/spa_oss.html. Accessed October 11, 2007.
- [62] Young, RR. (1994) Spasticity: a review. Neurology; 44 (suppl 9):S12-20.
- [63] Zidar, J. & Dimitrijevic, MR. (1991) In: Neurosurgery for spasticity: a multidisciplinary approach. New York: Springer-Verlag: 39-46.
- [64] Zidek, K. & Srinivasan, R. (2003) Rehabilitation of a child with spinal cord injury. Semin Pediatr Neurol 2003;10(2):140-50.

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