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Spasticity: A general outlook of pathogenesis and current management

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Abstract

Spasticity is an upper motor neuron disorder demonstrated by a velocity-dependent increased muscle tone, clonus, spastic dystonia or tonic stretch hypertonia. It is seen in conditions, such as traumatic brain and spine injury, stroke, hypoxic brain damage multiple sclerosis, degenerative diseases etc. The incidence of spasticity remains variable amongst different conditions. For example, in stroke patients, about 38-40% of patients would develop some degree of spasticity with about 16% requiring treatment. Incidence varies from between 27% in acute-subacute phase to about 42.6% per month in the chronic phase (> 3 months). Furthermore, there is an estimated 40%patients with spinal cord injury living with spasticity with some data estimating a higher figure (78%). Management of this condition which impact patients and caregivers are therefore of essence. Currently there are various treatment modalities available for management. The aim of this review is to discuss the pathophysiology, experimental studies of spasticity and to highlight various therapeutic strategies for management of spasticity.

Keywords: Spasticity; Cerebral palsy; Selective dorsal rhizotomy; Physiotherapy

1. Introduction

Spasticity is an upper motor neuron disorder demonstrated by a velocity-dependent increased muscle tone, clonus, spastic dystonia or tonic stretch hypertonia.[1, 2]. In certain neurological conditions, such as traumatic brain and spine injury, stroke, hypoxic brain damage multiple sclerosis, degenerative diseases etc., spasticity remains a finding (Table. 1). The impact of spasticity on the daily life of patients are massive due to loss of functionality and constant dependency on others for normal daily activities. Furthermore, the economic burden of managing this condition is equally huge with some patients being on pharmacological treatment for long periods of time coupled with routine episodes of rehabilitation. The incidence and prevalence of spasticity remains variable amongst different conditions. For instance, in stroke patients, about 38-40% of patients would develop some degree of spasticity[3, 4] with about 16% requiring treatment. Incidence varies from between 27% in acute-subacute phase to about 42.6% per month in the chronic phase (> 3 months)[5]. Furthermore ,there is an estimated 40%patients with spinal cord injury living with spasticity with some data estimating a higher figure (78%)[6]. The management of spasticity still remains a challenge, with currently no accepted standardized treatment protocol. The aim of this preview is to discuss the pathophysiology, experimental studies of spasticity and to highlight various therapeutic strategies for management of spasticity[7].

Spasticity may be caused by any of the following:

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Table 1 Common causes of spasticity

Cerebral palsy
Head injury
Suffocation
Multiple sclerosis
Cervical spinal stenosis
Infections of brain and spinal cord
Neurogenerative illness
Tumors of brain and spinal cord
Phenylketonuria
Traumatic brain and spinal cord injuries
Stroke
Vitamin or mineral deficiency (Vitamin B12, Vitamin E, copper)
Toxins

2. Pathophysiology of Spasticity

Spasticity occurs when there is disorder in upper motor neuron (UMN). Upper motor neurons are the largest pyramidal cells in the motor regions of the cerebral cortex that relays information to activate interneurons and lower motor neurons. Lesions of the UMN therefore reduces the inhibitory drive in the corticospinal tract causing misbalance between excitatory and inhibitory input resulting in spasticity . Spasticity is generally evident as excitability of muscle spindles, but with the involvement of the central nervous system[8]. UMN lesions inhibit information from brain to the spinal cord, resulting in a state of net disinhibition of the spinal reflexes.. There are many interrelated feedback mechanisms that can account for spasticity [9, 10]. For example, UMN lesions reduces the inhibitory feedback in the corticospinal tract, which results in continuous excitability of alpha-motor neuron causing sustained muscle contraction, especially in flexor muscles. Furthermore, there is increased excitability of spinal neurons in the brainstem motor tracts [9, 10]. Disruption of inhibitory response of interneuron of the antagonist muscle or prolonged action potentials in the sensory neurons of muscle spindles greatly contributes to hypertonia[9, 10]. Based on the pathophysiology spasticity can be classified into 3 types:

- Intrinsic Tonic Spasticity: Exaggeration of the tonic component of the stretch reflex (manifesting as increased tone),
- Intrinsic Phasic Spasticity: Exaggeration of the phasic component of the stretch reflex (manifesting as tendon hyper-reflexia and clonus), and
- Extrinsic Spasticity: Exaggeration of extrinsic flexion or extension spinal reflexes

3. History and Clinical presentation

Patient may present at the clinic with (1) new-onset of spasticity as the initial complaint from an underlying neurological condition like TBI, stroke or SCI or (2) worsening of pre- existing spasticity from previously diagnosed chronic neurologic disease e.g.MS over years or CP from infancy [11]. History of newer symptoms as well as progression of symptoms should be attained in patient who present with newer onset of spasticity. This includes but not limited to any weakness in motor functions, altered bowel and/or bladder function, altered sensation, pain, and sexual dysfunction. Furthermore, family, travel, diet history should be ascertained as well as any condition that compromises patients immunity. Patient with worsening chronic spasticity must be evaluated for possibility of a newer disease onset , or for triggers and for disease progression. These triggers may be drug-related, exposures to the skin, visceral, , or device-related. Spasticity can also be worsened by stress or other noxious stimuli such as injury, infections,, deep vein thromboses (DVT)[12]. The physical exam should include a thorough neurological exams including muscle tone, muscle power, reflexes, and sensation.

Patients may present with upper extremities findings such as “thumb in palm” deformity, with clinched fist resulting from persistent flexion of fingers flexion and thumb adduction. Spasticity in the legs might present with increased muscle tone especially hip adductors, flexors and extensors of the knee, and plantar flexors and ankle invertors. Patients may report with difficulty with wearing footwear if spasticity involves the extensor hallucis longus or long toe flexors. Physical examination may reveal some degree of resistance with motion , with increasing resistance directly related to increased speed of motion. This resistance is said to be a catch and persist with repeated movement till a sudden give felt which is termed the “clasp-knife phenomenon” [13] (Figure 1). Other examination findings are clonus, spastic dystonia or spastic co-contractions. Clonus is said to occur when there is simultaneous contraction and relaxation of both agonist and antagonist muscles groups. Spastic dystonia occurs when there is muscle contraction at rest, leading to a clinical posture that is very sensitive to stretch. Spastic co-contractions on the other hand refers to abnormal antagonist contractions that happens at the same time during voluntary agonist movement. The modified Ashworth scale remains one of the frequently used grading system for spasticity , which grades spasticity from 0 to 4 [12, 14]. Other scales include the Penn spasm frequency scale or Tardieu scale [14, 15]. (Table 2)

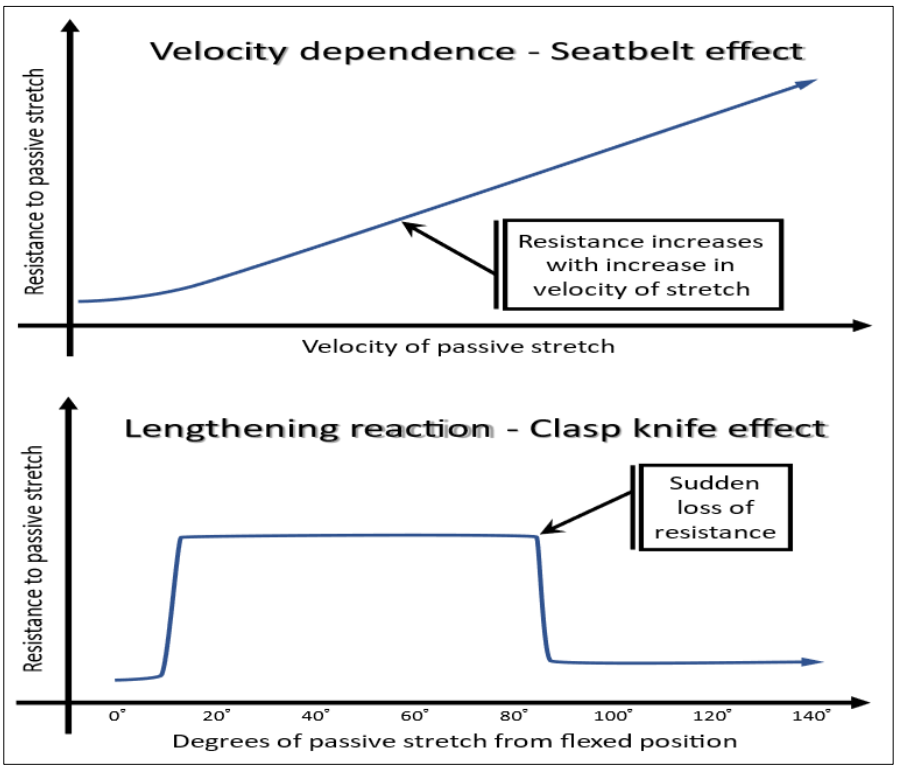

Figure 1 Characteristics of spasticity [16]

Table 2 Evaluation of spastic patient [17]

Assessment components	**Assessment scale criteria**	**Clinical assessment**
Passive motion range	Modified Ashworth scale (MAS), Tardieu Scale, motion analysis, goniometer	Assessment of length and extensibility of soft tissue. This is assessed by fast passive stretch of muscle. The MAS is used to assess stiffness and Tardieu Scale to assess spasticity.. Treatment goal: To restore range of motion[17]
Active motion range		Assesses neurological function from the net result of agonist recruitment minus the sum of passive soft tissue stiffness and spastic co-contraction muscle resistance. Treatment goal: Preservation of active movement ability of patient[18]

Rapid alternating movement		Assesses routine coordination and fatigue. Patient is asked to perform repetitive movement as fast as possible and fatigue due to spasticity assessed [18]
Function of Upper limb	Modified Frenchay Scale, Wolf Motor Function Test; Action Research Arm Test	Patients assessed based duration of task and quality of the movement. Alternatively, assessment of upper limb function via 10 daily routines, for example grasp and release of a cup
Function of Lower limb	Gait assessment	Assesses patient speed and strategies to compensate abnormal gait. Patient is allowed to walk for 10 m or 2 min while assessing gait.

Table 3 Grading of Spasticity

Modified Ashworth Scale

- Zero is defined as no increase in tone.
- 1 is a "catch and release," or minimal resistance towards end-of-range of motion (ROM).
- 1+ is similar to a grade of 1, but with a catch that is followed by resistance through less than half of ROM.
- 2 is increased muscle tone through the majority of ROM while still able to move the affected part.
- 3 is difficult passive movement throughout the majority of ROM.
- 4 presents with the affected part in rigid flexion or extension.

Penn Spasm Frequency Scale

- 0 is no spasms.
- 1 is no spontaneous spasms; only elicited through vigorous sensory and motor stimulation.
- 2 is occasional spontaneous spasms and easily induced spasms, occurring less than once per hour.
- 3 is spasms occurring between 1 and 10 times per hour.
- 4 is more than 10 spasms per hour.

Tardieu Scale

- Velocity to stretch is graded from V1 (as slow as possible) to V3 (as fast as possible).
- Muscle reaction is graded from 0 (no resistance through passive movement) to 5 (joint being immobile).
- The spasticity angle is graded as R1 (angle of catch at velocity V2 or 3) or R2 (full range of motion when the muscle is at rest and tested at V1 velocity).

3.1. Animal Models of Spasticity

Spasticity a common problem following neurological insult, has been difficult to reproduce in experimental models, cat [19-21] or rat [22-24]. This is because unlike humans, animals are able to recover motor function during incomplete spinal transections, a situation that may lead to chronic spasticity in humans. However, simulating complete transection in animals may results in bowel and bladder dysfunction which requires daily assistance and other complications such as such skin and gastric lesions, autonomic dysreflexia, cystitis, and autotomy [25-28]. Modeling contusional injuries [24], hemisection transections [23], or other injuries similar to cat model [19-21, 29] has been tried in rat models. However, only complete spinal transection models seem to simulate that which is found in humans after SCI, but with challenges in managing the long-term bowel and bladder dysfunctions and gait abnormalities.

Ritz et al developed the first animal model to successfully simulated complete spinal cord transection at Ca1 level whiles retaining normal bowel and bladder function, reflexes and hind limb gait [30]. In this model, there was decreased tail muscle function with spasticity, and abnormal ventral flexion of the tail at the midline. The spasticity characterized by hypertonia, clonus, and hyperreflexia, remained for about 3 years duration. Even though, this model offers simulation closer to larger animals and also easily reproducible, it is not suitable for studying limb movements by spinal control an important phenomenon in paralyzed humans. In the quest to attain a more suitable model, another trial for spasticity was done in a rat, but this time with cord transection at the level of S2. This resulted in dysfunction of tail muscle whiles leaving bowel, bladder, or hindlimb function intact. After the spinal cord transection, there was paralysis of the tail for two weeks, followed by hypertonia, clonus, and hyperreflexia which remain permanent after several weeks. Stretching of muscle or stimulation of the tail was marked with spasms and increased muscle tone, visible on electromyographic (EMG) recordings. The surrounding skin and hair, also developed sensory disturbances including thermal hyperalgesia

and tactile allodynia a phenomenon common in humans with spinal injury [31]. Such models have been used to examine intrinsic excitability in motorneurons both in vitro and in vivo providing considerable knowledge on the nature of the differences between inhibitory and excitatory, control of motor neurons in intact or spinal cord insults[32].

4. Management of Spasticity

4.1. Non-Pharmacological

4.1.1. Physiotherapy

Physiotherapy for spasticity can be categorized under 5 groups: exercise therapy, electrical stimulation, vibration, standing therapy, and radial shock wave therapy (RSWT).

- Exercise therapy:This includes passive mobility, stretches, tilt table standing and splinting. Traditionally there are 2 approaches; gait-related therapy and generic/ rehabilitation exercises. Gait-related therapies includes assisted standing, robotic-assisted gait exercises, and treadmill exercises with/without overhead suspension and body-weight support. Generic exercises and/or rehabilitation regimen includes balancing, strengthening and fitness routines with or without somatosensory or electrical stimulation. The exercise interventions are either at the outpatient or in patient with session and assessments determine progress of exercise programs. Some studies have shown that one session of unloaded lower limb exercise or Bobath's could improve ankle MAS and tibial nerve H-reflex excitability significantly[33-35]. A meta-analytical study of outpatient exercise routines showed significant improvement in muscle tone and spasticity. These exercises included routine stretches, balance and stability exercises , endurance and mobilization [36-39]. Inpatient exercise routine include multidisciplinary approach with inpatient rehabilitation, education, exercise instructions, and home training [40, 41]. Other studies using robot-assisted exercise protocols and body weight supported treadmill training [42, 43] also showed improvement of spasticity and ankle range of motion with resulting improvement in MAS[44].
- Electrotherapy: These involves the use of electrical stimulation (ES), transcutaneous electric nerve stimulation (TENS), somatosensory stimulation, ultrasound and other types of electrotherapy[45]. Electrical stimulation is either functional electrical stimulation (FES) or TENS. Some studies have reported significant improvement in MAS after some sessions of FES[46, 47]. Other studies compared the outcomes of different durations of TENS (1 hr/d or 8 hrs/d) on improvement of MAS . Results showed no improvement in quadriceps, patellar, or ankle tendon reflexes or clonus in both groups. However there was some significant improvement in Penn Spasm Frequency Score after 8hours of TENS. The use of TENS has been reported to significantly improve EMG activity and muscular tone of gastronomes while also significantly improving MAS of the ankle[48-50].
- Vibration:Vibration therapy has been shown to be instrumental in the treatment of spasticity. Therapy includes whole-body or focal muscle vibration (FMV) .To ascertain the effect of FMV on MAS, two studies have found insignificant improvement in MAS [37, 51]. However, FMV resulted in improvement of gait and time for double support [51].
- Standing Therapy: Of the studies that discussed therapeutic standing on Oswestry standing frame, the results showed no improvement in MAS or Penn Spasm Score but showed considerable improvement in hip and range motion [52].

Radial Shock Wave Therapy (RSWT)

A single study showed that four sessions of RSWT around planter extensor muscles might improve MAS of the ankle but not H-reflex excitability. Other study however showed no benefits of RSWT in spasticity[52].

4.1.2. Acupuncture

Acupuncture is another simple, less expensive, and safe treatment option for spasticity. Various studies have proven its efficacy in improving motor, sensory, speech, spasticity and other neurological functions in Chinese and Korean patients following stroke episode[53-56]. The efficacy of acupuncture is explained by the following mechanisms.

Breaking pain-spasm-pain cycle : The mechanism underlining the efficacy of acupuncture in spasticity still remains unclear. The widely accepted idea is however that acupuncture reduces pain, thereby breaking the pain-spasm-pain cycle causing muscle relaxation [45][57]. The threshold of pain receptor is increased after Acupuncture through the release of CNS opioid peptides, thereby reducing pain and hence relaxing the muscles [58].

Regulating activity of spinal motor neurons

Brain or spinal cord injury results in hyperexcitability of spinal motor neurons due to decreased supraspinal inhibitory control. There is also an increase in alpha-motor and gamma-motor neuron excitability and reduced inhibition of specific interneurons [10]. Acupuncture is therefore seen to regulate spinal motor neurons activity by down regulating both alpha and gamma motor neurons and/or upregulating interneurons inhibition. Similarly Fink et al. identified that acupuncture decreases the ratio of H-reflex amplitude to compound muscle action potential amplitude (H/M ratios) in comparison to controls [59, 60]. This decrease is means reduced excitability of motor neurons. Another study by Yu et al. also [61] found that acupuncture prolongs the recovery time of H-reflex in spastic muscle thereby increasing interneurons inhibition and reducing spasticity.

Regulation of neurochemicals

The role of acupuncture in spasticity was studied by Sun et. al in a rat stroke model. Like other studies their findings revealed that acupuncture decreases spasticity, but via enhancement of GABA, KCC2, and GABAAγ2 in the rat brainstem[62]. The elevated GABA levels results in inhibition GABA-mediated pre-synapses, thereby decreasing release of excitatory neurotransmitters hence indirectly reducing spasticity. Qi et al also discovered that acupuncture reduces muscle spasm via inhibition of inflammatory markers hence reducing spasticity [63].

4.2. Pharmacological management

Spasticity is mostly associated with other symptoms like pain, feeding disorders, sleep difficulty and difficulties with personal care among others [64]. It is worth-nothing that, not all spasticity are considered disabling and warrant management. It is therefore prudent to define which spasticity is considered disabling hence required pharmacological treatment (Table 4). Various pharmacological agents either acting centrally or peripherally on the central nervous system have been used in the treatment of spasticity.

Table 4 Definitions of disabling spasticity and location used in the current paper [65]

Disabling spasticity	
Term	**Definition**
Disabling spasticity	Spasticity which is perceived by the individual or caregivers as hindering body function, activities, and/or participation.
	This definition is based on clinical expertise and conceptually incorporates the domains of the International Classification of Functioning, Disability and Health (ICF).
Disabling spasticity location	
Term	Definition
Focal spasticity	Spasticity limited to muscles in a close anatomical region, including only 1 or 2 joints (excluding finger and toe joints, e.g. hand and forearm or foot and ankle) [66].
Segmental spasticity	Spasticity limited to several adjacent anatomical regions (e.g. hand, forearm, elbow and/or shoulder) [66].
Multi-segmental spasticity	Spasticity distributed to anatomically separate and distant sites and affecting at least 2 limbs, including the trunk (e.g. arm and leg, leg and trunk, or arm and trunk) [66].
Generalized spasticity	Spasticity diffused in more than 2 limbs.
Multi-focal spasticity*	Spasticity affecting multiple joints that are not adjacent (e.g. ankle and hip or wrist and shoulder).

4.2.1. Centrally Acting Drugs

- **Baclofen:** This function as agonist of gamma aminobutylic acid (GABA) B receptors at the spinal level [67] causing hyperpolarization of receptors. This leads to restriction of calcium influx thereby (1) restricting the release of presynaptic endogenous excitatory neurotransmitters (2) inhibiting spinal reflexes [68]. Baclofen is very useful in the management MS or TBI induced spasticity. However ,sudden withdrawal of baclofen treatment may result in rebound of the spasticity, hallucinations or seizures, hyperthermia, renal failure, and

even death. Thus, gradual tapering of medication is recommended in view of discontinuation. Dosing of baclofen should starts from 5 mg two or three per day, and with gradual increase by 5 mg in every 3 to 5 days if result not achieved up to 80 mg/day, the maximum dose recommended by FDA [69].

Alpha-2 Agonists

Clonidine an alpha-2 agonist has been studied to decrease spasticity by inhibiting afferent sensory impulses below the level of injury [70]. In the past Clonidine was used in patient with spasticity from SCIs and also as a blood pressure medication [70]. The use of clonidine in spastic patient has shown variable outcomes ,however due to the unwarranted side effect of bradycardia, hypotension,and drowsiness, clonidine has fallen out of use [71][72]. Tizanidine is another alpha-2 adrenergic agonist chemically similar to clonidine and also enhances presynaptic inhibition of the spinal reflex. Like clonidine, Tizanidine causes drowsiness and sedation in about 50% of patients. Although can also cause hypotension, liver damage, dry mouth, bradycardia, and dizziness some trials have indicated better tolerability than baclofen or diazepam[73]. Dosing for tizanidine starts at 2 to 4 mg/day, typically at night, and could reach a maximum dose of 36 mg/day divided 3 to 4 times/day[74].

Anticonvulsants

- **Diazepam:** Diazepam exerts an inhibitory functions on GABA receptors, leading to increased neuron hyperpolarization and reduced neuron firing. The result is reduction in nerve reflexes and hence spasticity especially in MS and SCI[68, 75]**.** Side effects include sedation, impaired memory, disrupted REM sleep [76],and may cause withdrawal symptoms if stopped abruptly [77]. Diazepam is started at a dose of 4 mg at night or 2 mg two times a day, with maximum adose of 60mg in the day. Gabapentin another anticonvulsant is also given for the treatment of spasticity but mostly as an adjunct [71]. It has similar structure to GABA but has no binding properties to the receptors[76]. The mechanism of action is thought to be the inhibition of calcium channel via alpha-$2_{\delta 1}$ subunit of voltage-dependent calcium channels. Although gabapentin is not the first line drug for spasticity, Gruenthal et al. [78] showed that gabapentin alone reduced the Ashworth scale and spasticity in comparison to placebo. Side effects include includes drowsiness, nystagmus and tremor [76].

4.2.2. Peripherally Acting Drugs

Dantrolene Sodium unlike other spasticity drugs acts by blocking calcium release from sarcoplasmic reticulum of peripheral muscle [79]. This results in decrease in muscle spindle sensitivity and fiber contraction. Side effect consist of effect on cardiac muscle, liver toxicity, sedation ,drowsiness, fatigue , weakness, paresthesias, nausea, diarrhea, and vomiting. Dantrolene is mostly preferred for spasticities in CP or head injury[67]. It is also used as a treatment for malignant hyperthermia, neuroleptic malignant syndrome, and hyperthermia from baclofen withdrawal. Initial dosing is from 25 mg twice daily to maximum dose of 400 mg daily, in two or three divided doses per day.

4.3. Interventional Treatments

- **Diagnostic Nerve Blocks:** This treatment procedure involves using electrostimulation or ultrasound guidance to localize spastic nerve and temporarily blocking nerve conduction with local anesthetics. This allows for assessment of patient to plan for more definite management since the temporary reduction in spasticity is an indication patient might likely benefit from definite permanent management. Frequently used agents includes lidocaine or bupivacaine[80].
- **Chemoneurolysis:** Injection of neurolytic agents has been found useful in management of spasticity with effect lasting for months[81]. These agents which includes phenol and ethyl alcohol are injected under electrostimulation or EMG guidance **[68, 82]**. Usually a phenol concentrations of 2% to 7% is used to achieve axonolysis (demyelination and denaturation of protein the axons) lasting about 6 months or more. Ethyl alcohol when used has optimum efficacy at concentration of between 45% to 100%. Although is less toxic than phenol common side effects includes dysesthesias, muscle weakness, transient swelling of muscle, DVT, etc. Phenol, on the other hand could cause CNS depression, convulsions, and cardiovascular failure. Dosage of phenol is normally below 8.5 grams and limited to 20-30 mL of 5% concentration. Ethyl alcohol has minimal systemic side effects if injected intravascularly[83].
- **Chemodenervation with Botulinum Toxin:** .There are currently 4 approved botulinum toxins used clinically: onabotulinumtoxin A, abobotulinumtoxin A, incobotulinumtoxin A, and B toxin, rimabotuinumtoxin B [82]. Onabotulinumtoxin A is currently being used in the treatment of both upper and lower limb spasticity whiles incobotulinumtoxin A is mostly for upper limb. All serotypes however have effect on the neuromuscular junction by blocking release of acetylcholine. The toxin which is produced by *Clostridium botulinum* is released in a form containing a light and heavy chain. When is taken up by the nerve terminal the light chain which is

the active part is taken up by the host cytoplasm cleaves to the SNARE complex and its components (SNAP-25,synaptobrevin, and syntaxin) thereby preventing release of acetylcholine leading to chemical denervation. Common side effects include respiratory, flu-like symptoms, depression or dysphagia and injection site pain[84, 85], hence care should therefore be taken in patients with myasthenia gravis, amyotrophic lateral sclerosis (ALS), peripheral motor neuropathic disease or Lambert-Eaton syndrome. Botulinum toxin should also be used with caution in patient undergoing treatment with spectinomycin or aminoglycoside antibiotics. Effect of botulinum injection is dose specific with higher doses causing significant muscle weakness, hence it is recommended that electrical stimulation ,ultrasound or EMG be used for localization of the specific spastic muscle [86]. Dose of Onabotulinumtoxin A is generally from 25 to 200 units per muscle but relatively dependent on the degree of spasticity, muscle size, function and patient weight. Typically, there is the rule of 3s that describe effect of injection i.e. takes 3 days to have initial effect, peak effect in 3 weeks, and lasts for about 3 months.

- **Intrathecal Baclofen Pump:** The intrathecal baclofen pump is another management option. This device is usually implanted at the T6-7 or T11-12 intrathecal space and delivers baclofen directly into the cerebrospinal fluid (CSF), allowing patient to receive necessary concentration while decreasing related side effects associated with oral doses [87]. For instance dose concentration ratio of baclofen in the spinal cord between the intrathecal and oral dosage is 100:1 [88].The device components include a catheter implanted into the intrathecal space and a programmable battery powered pump-reservoir system that is implanted subcutaneously in the abdominal wall. The pump could be programmed to deliver different dosage of baclofen at a programmed time and refilled intermittently when the programmable alarm indicates reservoir is empty or pump malfunction[69]**.** Side effect of baclofen overdose including somnolence, respiratory depression, hypotonia or severe muscle weakness, nausea/vomiting, hypotension, and seizures are more severe in these management group. ITB pump is useful in patients with generalized spasticity [89], however percutaneous trial injection is done to establish benefit before implantation is considered. Patients who demonstrates significant decrease in spasticity, are considered good candidate for the procedure. A double dose of the efficacious trial percutaneous dose is considered the initial dose for 24hours after pump is implanted. Care is taken to follow strict compliance and monitoring to prevent side effect and reservoir running out [68].

4.4. Surgical management

Selective dorsal rhizotomy (SDR) is a procedure performed to permanently reduce spasticity. This procedure is suitable for CP children with bilateral lower limb spasticity [90]. It involve surgically disrupting monosynaptic stretch reflex at the lumbosacral level [90]. Various studies which compared patient who underwent this procedure to those who underwent only physiotherapy indicated marked improvement in spasticity, gross muscle strength, and function, gait and kinematics in SDR group [91-94]. Currently, there are 2 main ideologies regarding SDR, the “Peacock-school” and the “Park-school”, with the former introduced by Warwick Peacock [95] and later a modification by Dr. Tae Sung Park (who modified Peacock’s procedure and limited exposure to conus) [96]. With Peacock’s approach laminectomy of L2 to L5 is done and SDR of L2 to S1 done under electromyographical (EMG) monitoring according to Fasano’s principles [95, 97-99]. The dorsal root is divided into smaller radicles and bundles with abnormal EMG reading sectioned in order to preserve sensory and sphincter functions [90]. Peacock would always spare S2 when there is anal sphincter function, even when there is abnormal S2 EMG findings. Over time Marc Sindou developed the keyhole approach i.e. Keyhole interlaminar dorsal rhizotomy (KIDr) technique to minimize the extent of the laminectomy and reduced ligamentous disruption [100]. A key factor identified to play a major role in the outcome of SDR is intraoperative neuromonitoring [101, 102]. For example a study showed that sectioning of S2 nerve using EMG and compound muscle action potentials (CMAP) monitoring resulted in improvement in ankle spasticity [102]. Also Nishida et al. have argued that neurophysiological monitoring has positive impact on outcomes. Similar view was shared by Fukuhara and colleagues who studied micro-architecture of spastic nerve highlighted the role of stimulation to identify and section roots causing abnormal spasticity[103, 104]. On the contrary Steinbok argued that there was still about 90% improvement in lower limb function in procedure done without monitoring [105] [106]. Furthermore Warf et al. also stated that SDR could be done with no neuromonitoring or fascicular dissection providing similar out come and hence effective to practice especially in lower resource centers [107].

4.5. Future directions

An enormous amount of work has been done on the usefulness of neuromonitoring for SDR surgery but there is still the lack of proper understanding in the physiology of spasticity and how surgical intervention addresses the cause and not only managing symptoms. Rigorous research is therefore encouraged to the effect. Furthermore, research into other modalities of treatment like stem cell transplant is warranted[108, 109]. Although there are some studies on combine treatment modalities, there is still vast gap in our understanding of the efficacy of combine treatment approaches. For example some studies have shown improvement of gait and ankle flexibility when electrical stimulation was added to

after botulinum toxin A injection ankle [110]. Another study shows improvement of spastic foot drop after a stroke in adults[111, 112] when of Botox was done with ITB [113]. Further research is therefore needed for the advancement of spasticity treatment.

5. Conclusion

The management of spasticity presents with variable outcome from patient to patient. Hence there is diversity in opinions as to the management, with no common consensus or treatment guideline for management. As it stands prognosis is generally considered good in patients that respond to a particular treatment modality. There is the need to further understanding into the various treatment available and to draw up an algorithm for management. The standard algorithm can also serve as a basis for future prospective research or randomized control trials to scientifically measure the efficacy of the algorithm and the various treatment modalities.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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