Spasticity: A Comprehensive Review

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Spasticity is an important problem among patients with neurologic disorders,[1,2] such as traumatic brain injury, spinal cord injury, stroke, cerebral palsy or multiple sclerosis. Spasticity is always caused by upper motor neuron lesion resulting in either intermittent or persistent involuntary muscle contraction.[1-3]

Fundamentally, spasticity can be widely classified into harmful and useful spasticity. The impacts of harmful spasticity to patients include handicap, abnormal posture, spasticity-related pain, and struggle in movement, ambulation, self-care and caregiving.[4,5] Furthermore, harmful spasticity leads to adverse emotional status, poor body image and loss of motivation in patients.[6] On the other hand, useful spasticity is, interestingly, occasionally beneficial to some patients, for example, by increasing regional blood circulation, and aiding maintenance of standing and walking.[4,6] Therefore, the selection of patients who need treatment is vitally essential.

The present article is a comprehensive review of spasticity with regard to aspects of pathophysiology, clinical manifestation, pretherapeutic assessment, nursing care and multi-modalities of management.

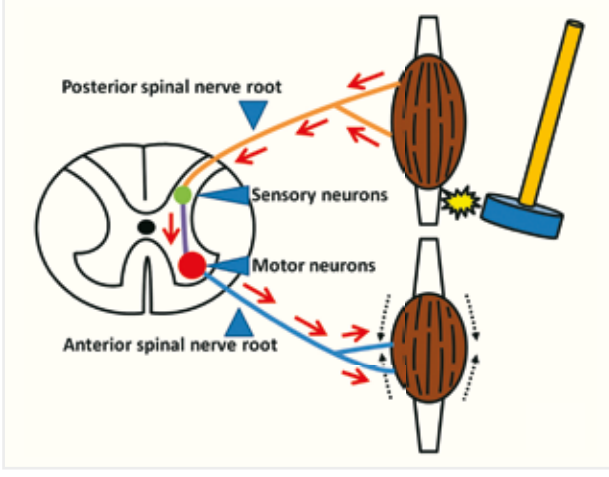

Fig 1. The circuit of the spinal reflex arc. A percussion of a tendon creates a nerve impulse (red arrow). The nerve impulse is ordinarily transmitted through the posterior spinal nerve root, sensory neurons, motor neurons, anterior spinal nerve root and motor nerve causing abrupt muscle contraction.

Pathophysiology

Basically, in order to understand the pathophysiology of spasticity, the circuit of the spinal reflex arc should be mentioned first. In normal circumstances, a complete spinal reflex arc occurs as the following sequence. Firstly, a rapid stimulation of a muscle group creates a nerve impulse inwards to the posterior (dorsal) horn neurons of the spinal cord through the posterior (dorsal) spinal nerve root. This is defined as "the afferent spinal pathway". After that, the nerve impulse is immediately transmitted to the motor neurons in the anterior (ventral) horn of the spinal cord and it is eventually sent outwards from the spinal cord or "the efferent spinal pathway" through the anterior (ventral) nerve root, and then a motor nerve to the muscle resulting in a rapid muscle contraction (Fig 1).[7] In a normal individual, the intensity of the muscle contraction should be normal (normoreflexia). A common instance of the spinal reflex arc is the patellar reflex (knee jerk). A percussion on the patellar tendon promptly causes a contraction of the quadriceps femoris muscle resulting in an extension of the knee. The muscle contraction is abruptly elicited following a percussion on the tendon.

In a normal situation, motor neurons in the spinal cord are controlled by the higher central nervous system (CNS) or so called supraspinal control through descending inhibitory tracts, particularly the dorsal reticulospinal tract which runs closely adjacent to the corticospinal tract.[8] In the presence of a CNS lesion, including brain or spinal cord pathology or a combination of them, disruption of the inhibitory tract causes a loss of the supraspinal control. Subsequently, the motor neurons become hyperactive[1,3,8] resulting in an emergence of spasticity. Interestingly, Boulenguez and colleagues found a downregulation of the potassium-chloride co-transporter-2 (KCC2) of the motor neuron cell membrane. The change of KCC2 makes the membrane potential of chloride more positive affecting the neuronal function to be more excitable.[9,10]

Prolonged spasticity leads to secondary biomechanical changes within the spastic muscles and soft tissues. Skeletal muscle plasticity in spasticity consists of increased variability in muscle size, increased numbers of abnormal

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muscle fibers (round fibers, moth-eaten fibers), increased extracellular matrix, reduced muscle fiber length and reduced numbers of sarcomeres. Additionally, increased muscle spindle cell stiffness and decreased mechanical properties of the extracellular matrix often occur.[11] These changes usually cause stiffness and contracture which impede the normal range of movement and function of the patients.[8]

Clinical manifestation

Clinical features of spasticity are varied ranging from a mild to a severe degree. Spasticity-related pain or spastic paralysis can be found.[2] The clinical manifestation is chiefly categorized into positive and negative features. The positive characteristics include muscle hyperactivity, muscle spasm, hyperreflexia, clonus, abnormal plantar reflex, flexor and extensor co-contraction, abnormal body posture and spasticity-associated pain. On the contrary, the negative attributes are comprised of weakness, paralysis, early hypotonia, muscle fatigability, loss of dexterity and abnormal coordination.[1,2,7,8,12]

Tendon shortening or joint contracture may occur following long-lasting spasticity. Psychiatric problems are also found, such as depressive disorder, stress disorder, insomnia or impaired cognitive function. Furthermore, pressure ulcer and recurrent infection are common in patients suffering from spasticity.[7]

Clinical assessment

Useful spasticity does not require treatment, whereas harmful spasticity should be appropriately managed. Therefore, clinical assessment of the patients is essential for suitable management and it can be used for evaluation following treatment.

Ordinarily, clinical assessment of spasticity includes the following.

1. Severity of spasticity
2. Range of motion (ROM) of related joints
3. Tendon shortening or joint contracture
4. Quality of life (QOL)

Assessment of severity of spasticity

Originally, the Ashworth Scale was proposed to use for assessment of spasticity in multiple sclerosis.[13] Presently, the Modified Ashworth Scale (Table 1) is a commonly used tool in assessment of severity of spasticity due to various etiologies.[14-17] A higher level of the score indicates increased severity of spasticity.

Furthermore, spasticity can be categorized into mild, moderate and severe spasticity. Mild spasticity (mild increase in tone, no or minimal loss of ROM) is sometimes beneficial to the patient and may not require any intervention. Moderate spasticity (moderate increase in tone, loss of ROM, possible contracture) usually requires management, particularly are habilitation program. Severe spasticity (marked increase in tone, loss of ROM, probable contracture) absolutely needs long-term continuous multimodalities of treatment to prevent orthopedic sequelae.[1] Surgical intervention should be considered in the refractory cases.[18]

Assessment of ROM

Goniometry (the measurement of ROM in a joint) is a proper clinical assessment of the ROM.[2, 16] It should be performed by using a standard goniometer. A difference of measurable degree between each extreme direction of joint movement is defined as a ROM. ROM can be measured during either active manner (joint is moved by the patient) or passive manner (joint is moved by the examiner).

Assessment of tendon shortening or joint contracture

Clinical differentiation between spasticity and tendon shortening or joint contracture is very important because management of both conditions is significantly different. Two major strategies for the clinical distinction are the following.

1. Local peripheral nerve block

The principle of the method is temporary inhibition of motor nerve function[19] by using injected local anesthetic agent, especially xylocaine. After the injection, normal ROM indicates only spasticity, whereas limited ROM relates to associated tendon shortening or joint contracture.[20]

2. General anesthesia

Examination of range of motion whilst a patient undergoes general anesthesia is a strategy used immediately before operative treatment. The interpretations of this method resemble those of a local nerve block (Fig 3).

Assessment of QOL

QOL is popularly used in the evaluation of therapeutic outcome. Additional to reduced spasticity, improved voluntary movement and decreased pain, and a higher level of QOL are the main indicators of improved outcome of management.[16] There are various methods in the assessment of QOL, e.g. 36-item Short-Form Healthy Survey (SF-36)[21] and Satisfaction With Life Scale (SWLS)[22]

SF-36 is a questionnaire utilized to evaluate physical health and mental health. The evaluated physical aspect consists of physical functioning, role-physical, bodily pain and general health. The assessed mental health includes vitality, social functioning, role-emotional and mental health.[21]

SWLS is an easily used questionnaire for assessment of satisfaction with life (Table 2). Additional to application in patients with spasticity, it may be used in adults with spinal cord injury since childhood.[23] The higher score relates to the better QOL.

Management and nursing care

The steps of management and nursing care for

TABLE 1. Modified Ashworth Scale[14]

Grade	Description
0	No increase in muscle tone
1	Slight increase in tone manifested by a catch and release at the end of the range of motion
1+	Slight increase in tone manifested by a catch, followed by minimal resistance in remainder of range
2	More marked increase in muscle tone through most of range
3	Considerable increase in tone, passive movement difficult
4	Affected parts rigid in flexion or extension

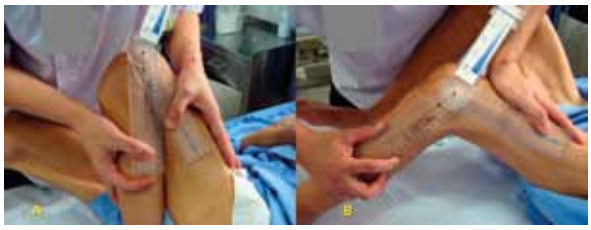

Fig 2. Goniometry by using goniometer. Measurement of angle of the joint during full flexion (A) and full extension (B). The difference of the angles is the ROM.

patients with spasticity are the following, respectively (Fig 4).[1,24-26]

- Discrimination between useful and harmful spasticity.
- Consideration of necessity of treatment.
- Settlement of goals of treatment, plans of management and nursing care.
- Categorization into focal and generalized spasticity.
- Appropriate management.
- Consideration of operative management in cases of failure of non-operative treatment.
- Major plans of nursing care includes the following.[1,6,26,27]
- Interval evaluation before and after treatment.
- Placement of spastic patients in suitable positions.
- Practice in standing and walking.
- Rehabilitation and physical therapy.
- Use of orthotic devices.
- Detection and correction of precipitating factors of worsening spasticity, for example occult infection, urinary retention, constipation, poor physical posture, inappropriate clothes or orthosis.
- Provide knowledge to patients and caregivers.

Importantly, the management of patients with spasticity should be a multidisciplinary approach[1,7,24,26,28,29] and always requires regular long-term follow-up.

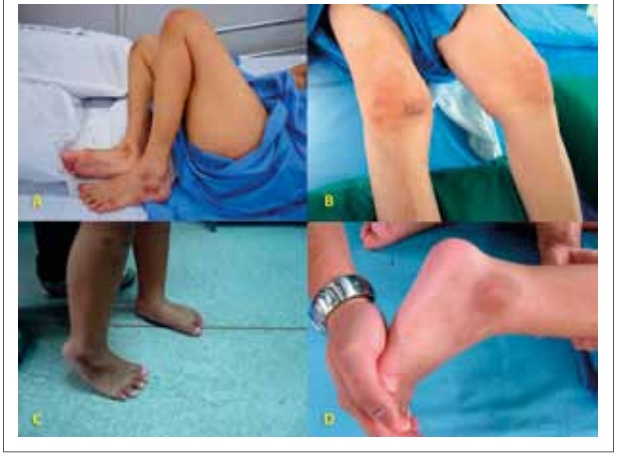

Fig 3. Discrimination between spasticity and tendon shortening or contracture of the knee (A & B) and ankle (C & D). A: Before general anesthesia, the patient is in flexed knee posture. B: After general anesthesia, fully extended knee indicates pure spasticity without orthopedic complication. C: Another patient has right equinus foot prior to general anesthesia. D. The same posture of the right ankle is elicited during general anesthesia. This finding indicates orthopedic sequelae which may be shortening of tendo achilles or ankle contracture or a combination of both.

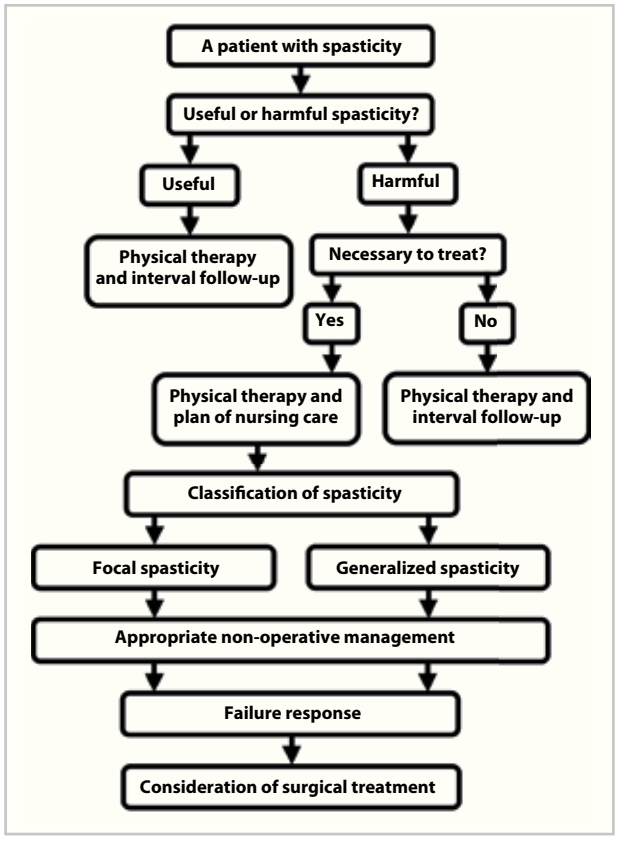

Fig 4. The algorithm of management and nursing care for spasticity[1, 24-26]

The following are major therapeutic options for spasticity.[3,7,24,26,29]

1. Non-operative treatment
   - Oral antispasmodic agents
   - Botulinum toxin injection
   - Physical therapy
   - Orthotic devices
2. Operative treatment
   - Neurosurgical procedures
   - Orthopedic procedures
   - Combined neurosurgical and orthopedic procedures

Oral antispasmodic agents

Utilization of oral antispasmodic agents is usually the first line management for spasticity.[30] Theoretically, this option is proper for patients who are suffering from regional or generalized spasticity. The common oral drugs used for treatment of spasticity include baclofen and tizanidine.[1,24,29] Baclofen is a common antispasmodic drug for patients with spasticity of various etiologies. The drug stimulates the inhibitory neural pathways resulting in reduced spasticity.[3,31] It is less effective in spasticity of the cerebral origin than that of the spinal origin.[30] Never-

TABLE 2. Satisfaction With Life Scale (SWLS)[22]

Score	Description
1	In most ways my life is close to my ideal
2	The conditions of my life are excellent
3	I am satisfied with my life
4	So far I have gotten the important things I want in life
5	If I could live my life over, I would change almost nothing

theless, it is still the most widely used drug in spasticity reduction. The major adverse effects include somnolence, confusion, dizziness and unsteady gait.[1,31]

Tizanidine is a popular drug used for spasticity of the spinal origin.[3,29,31] It can be given in cases with cerebral origin spasticity as well. The drug acts mainly by reduction of neural impulses in the central nervous system leading to diminished spasticity.[1,3,29,31] Tizanidine is an effective antispasmodic agent confirmed by a few randomized studies.[30] The common side effects are somnolence, dizziness, dry mouth and muscle weakness.[1,3,31]

Botulinum toxin injection

This strategy is the treatment of choice for spasticity revealing in focal form.[3,32] Type A botulinum toxin[3] is directly injected into spastic muscles.[32] The injected drug acts by inhibition of neural transmission at the neuromuscular junction.[1,33] However, the effects of the drug are always impermanent.[1] The effects often vanish within 3 months,[29] so repeated injections are compulsory. Many large series confirmed the efficacy of botulinum toxin in the treatment of spasticity.[34-39] The adverse effects are mainly divided into local and systemic effects. The local adverse effects consist of focal muscle weakness and pain around the injected site, whereas the systemic side effects include flu-like symptoms, dry mouth and, extremely rarely, generalized paralysis.[3,29,33]

Physical therapy

Physical therapy is one of the most important modalities which should be incorporated into the management of most spastic patients. The main objective is to improve motor function and prevent orthopedic complications.[24,29] Furthermore, it plays a major role in positioning and seating.[40] The appropriate time of the therapy should be at least 2 hours daily.[29]

Orthotic devices

This option is a major supplementary treatment in spastic patients with abnormal posture or impaired motor function. Selection of the devices relies on various factors, including postural characteristics, residual motor function and the ability of patients.[29] Ankle foot orthosis (AFO) is a markedly useful orthosis among patients with ankle spasticity or who have gait problem, whereas hand splint is distinctly beneficial in patients with spastic wrist and fingers (Fig 5). A large meta-analysis revealed that AFO could immediately improve speed and step length of walking, and standing balance in patients after stroke and non-progressive cerebral lesions.[41]

Neurosurgical procedures

In fact, neurosurgical treatment is obligatorily the last alternative in the management of spasticity. The surgery must be considered in patients who do not respond to the non-operative management.[18,24] Fundamentally, the neurosurgical intervention is divided into two categories[42] including

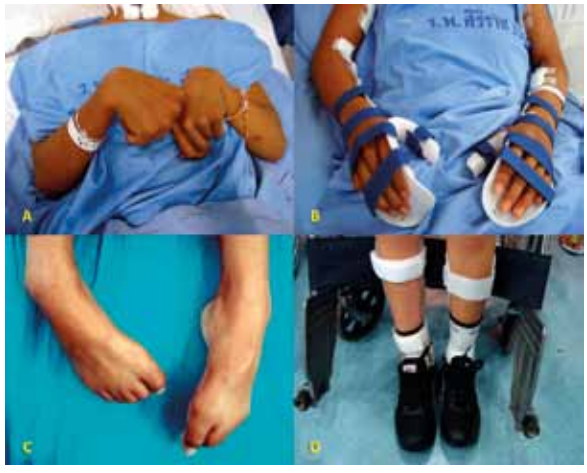

Fig 5. Utilization of orthotic devices. A: The patient with severe spastic wrist and fingers of the both sides. B: Application of hand splint following neurosurgical treatment for the spasticity ensuing the dramatically improved posture. C: Another patient with severe bilateral ankle spasticity and tendon shortening on the right side. D: After a combined neurosurgical and orthopedic operation, AFO is used to improve the posture and gait function.

1. Neuroablative procedures

The principle of all operative procedures in this category is diminution of the hyperactive spinal reflex arch in either the afferent and / or efferent pathway.[43] The reduction of excessive spinal reflex arc function brings about apparently diminished spasticity. The surgery can be performed on various neural components, including dorsal spinal nerve root, dorsal root entry zone (DREZ) of the spinal cord, ventral spinal nerve root, and peripheral nerve.[43-46] Numerous studies have shown the efficacy of neuroablative procedures on severe intractable spasticity.[47-52] The marked advantage of the operations is being free of hardware or the necessity for refill of intrathecal drugs, whereas the major disadvantages are irreversible effects and surgical complications, e.g. weakness and sensory deficit.[7,42,43,53]

2. Neuromodulations[42]

Implantation of a device for intrathecal drug delivery or neurostimulator is the hallmark of this procedures. The operations modulate the function of the nervous system resulting in decrease of spasticity. The preference of the procedures is non-ablative and reversible, while the major disadvantages include the high cost of hardware and intrathecal baclofen and hardware complications such as hardware malfunction, infection or expired power source.[7] Nevertheless, intrathecal baclofen therapy have recently become the most popular neuromodulative procedure for

TABLE 3. The neurosurgical treatment of spasticity[7,42-44,46,47,49,53,54,59]

Neuroablative procedures		Neuromodulations	
Peripheral procedures	**Central procedures**	**Intrathecal drug delivery**	**Electrical stimulation**
Selective dorsal rhizotomy	Dorsal root entry zone (DREZ)	Intrathecal baclofen therapy	Spinal cord stimulation
Selective peripheral neurotomy	lesion or DREZotomy		Cerebellar stimulation
	Longitudinal myelotomy		
	Thalamotomy		
	Dentatotomy		

patients who are suffering from severe intractable generalized or regional spasticity. The operation could effectively reduce spasticity in many series.[54-58]

An overall summary of the neurosurgical treatments of spasticity is shown in Table 3.

Orthopedic procedures

These types of surgery are sometimes considered a treatment of spasticity, particularly in cases with tendon shortening or joint contracture. The surgery aims to reposition joints and extremities ensuing in increased ROM and improved posture.[40] Reduction of muscle tension, for example by using tendon lengthening leads to decrease spasticity.[42] Furthermore, osteotomy can be used to correct existent fixed bone and joint deformities.[25]

Combined neurosurgical and orthopedic procedures

The combined surgery is indicated in patients with severe intractable spasticity with significant orthopedic sequelae.[60] Enhanced therapeutic effect is the crucial purpose of the combined procedures. However, both types of operation can be performed either separately or in the same session.

CONCLUSION

Spasticity is a major problem in patients with neurologic abnormalities. It impedes voluntary movement, daily living and care giving. Pretherapeutic assessment of spasticity is essential in the management. A multidisciplinary approach is clearly useful and leads to better treatment outcomes. The selection of the various therapeutic options depends on an individual. Surgical intervention should be an alternative for the refractory cases.

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Correction

Tanawan Kummalue, et al. would like to make a correction of the original article on "Antiproliferative and antimicrobial activities of endophytic fungus isolated from *Erycibe elliptilimba*" [Siriraj Med J 2010;62:237-240]. The family name of the second author should change from "Fungladda" to "Foongladda."