SPASTICITY

An overview of the term.

Tsintou Magdalini*

Medical School, University of Thessaly.

*Editor in Chief, reviewer, webmaster.

Abstract

The attempt of a precise definition of the term spasticity, scientifically documented and clinically adequate, is constant in recent years. Numerous of clinical signs that may be contained in the term, a large number of pathologies exhibiting clinically spasticity and the practical weaknesses of objective measurements for spasticity, render the term ambiguous.

This article there is an attempt to describe this clinical entity, a historical overview of the definitions which have occasionally given as well its differential diagnosis and its diagnostic approach.

Keywords: motor neuron, pyramidal tract, hypertonia, clonus, catch.

1. Introduction.

Spasticity is due to a failure of the pyramidal system and it is characterized by hypertonia. The simultaneous violent and involuntary contraction of both the agonist and
antagonist muscles participating in a movement is responsible for an asynchronously motion. The spasticity is accompanied by leg clonus and increased tendon reflexes.

In spasticity, an increased resistance to the passive muscle movement is also observed because of the myotatic reflex superaction. The resistance occurs at the beginning of the passive movement and then a sharp relaxation follows (jack-knife phenomenon). Spasticity more strongly affects certain muscles, the so-called anti-gravity muscles i.e. the muscles working against gravity (the forearm flexors and leg extensors) causing characteristic postures. The abnormal patterns of both movements and postures cause deformities of the spine (scoliosis, kyphosis), the hips, the knees and the ankles. The spastic muscles exhibit muscle weakness and a limited orbit range of the articulation joints.

But if diagnosed early, through a targeted diagnostic strategy by means of the physical examination and weighted scales and also objective measures, the prognosis is very good. Although a medical intervention is not often required, given that the functionality of the patient is affected to varying degrees depending of the case, it is important to have a clear picture of this nosological entity and of its differential diagnosis in order to avoid subdiagnosis, delaying the proper treatment of the patient, encumbering the future treatments and reducing the possibility of a full recovery.

2. Definition

2.1 The history of the term

The term "spasticity" was originally associated with a slight resistance towards the end of the passive movement with increased tendon reflexes [1]. Since then, this situation has been further studied and its pathophysiology understanding associated with spasticity has evolved much. Many definitions for the term of spasticity have been proposed but eventually even up to date the scientific community has not settled on a definition.
The first formal definition was given in 1980 by Lance [2-4] and it suggested that spasticity should be defined as "a movement disorder characterized by a motion speed dependent hypertonia and increased tendon reflexes arising from the suspension weakness of the tendon reflexes and it occurs as a component of the upper motor neuron syndrome".

However, the definition has been challenged in recent years for two different reasons. Firstly, many different clinical signs are referred to as spasticity such as increased muscle tone, clonus, seizures, and increased reflexes, but it is clear that these signs may occur independently of one another and they do not necessarily have a common pathophysiology. The definition of Lance is narrower than the clinical use of the term but also even more explicit. Some authors have decided to use more clinically oriented definitions involving clonus, seizures, and increased reflexes [5.6], whereas others have decided to use a stricter definition which is hypertonia oriented [7.8]. In addition, many studies have demonstrated that many candidates can be found that they have spasticity by the clinical examination but they do not exhibit any sign of increased reflexes [9.10]. The increased muscle tone in such patients appears to be caused by structural changes of the muscles associated with the contractions [11-14]. So a debate has arisen about whether the spasticity may also be explained by changes in the muscle properties rather than the increased reflexes and changes in the central processing of the incoming sensory stimuli to the spinal column. The most likely explanation for why many studies have failed to find increased reflexes in patients in whom spasticity was clinically found, is that it is difficult to clinically distinguish the increased hypertonia caused by passive changes in the muscles than the active ones. This is even more difficult in the lower limb. This problem may also explain the relatively low reliability of the Ashworth scale in the spasticity measurement as it relies on the ability of the examiner to distinguish the resistance depending on the motion speed [15-17].
We briefly mention only a few broad knowledge of the pathophysiological view of the spasticity because it's something that has influenced the subsequent definitions which were given. The observed then abnormal increased activity of the tendon reflexes may also arise from changes in the membrane properties of the α-motor neuron and / or changes in the activation threshold of the neuron in question [18]. The latter is influenced by a variety of pathways: Ia Group of presynaptic inhibition, Ia group of reciprocal inhibition (by antagonist), of recurrent Ib inhibition, group II of stimuli afferents, group III and IV of the cutaneous stimuli afferents and the reduced recurrent inhibition Renshaw. Both Denny-Brown and Lance seems to suggest that the hyper-excited deep tendon reflexes are a prominent spasticity feature [15-19]. Current evidence suggests that this may not be true and that the variability of the reflexes in humans with spasticity is great [20.21] and it may not differ from that of the general population without spasticity.

Trying to be more accurate, the North American Task Force on Childhood Motor Disorders proposed the revising of the spasticity definition, defining it as "a motion speed-dependent increase of hypertonia with a resistance sense (catch) after overcoming a threshold [22].

But unfortunately, with these definitions, is considered as a given condition that the increase of the tendon reflexes can be reliably measured through the assessment of the muscle tone (rigidity) during the passive movement of the muscles. The indirect measurement of the muscle activation, however, is flawed because it involves confusing factors. Factors that may act as confusing of rigidity are the mechanical properties of the musculoskeletal structures which are extended, the compliance of the patient (ie, the ability to relax) and finally the muscular activity at rest. These factors may lead to significant variances between the cases. An additional confusing factor in modeling of the muscle activity effects in stiffness refers to the modeling of the productive power during an eccentric contraction [22]. To solely attribute
the increased motion speed-dependent resistance on an externally imposed movement to spasticity, is therefore probably inaccurate. The muscle-tendon complex behaves as a glioelastic material and it will inherently exhibit the same motion speed-dependent behavior in the absence of any muscle activation. An important part of the literature, ignoring Lance definition, defines spasticity as an increase in muscle tone (ie an increase in resistance to an externally imposed passive movement). Although it seems a fairly clear definition, there is also a potential source of ambiguity in this definition. The word "tone" may also be defined as a state of readiness for action / contraction (innervation status) [23].

To infer which of the two definitions have been used in studies of adult spasticity is usually easy. However, this may be difficult in studies of cerebral palsy. Using the same logic as mentioned earlier, the validity of the use of the increased stiffness as an indication for spasticity existance is little. The North American Task on Childhood Motor Disorders is trying to make more accurate Lance definition, by adding additional details [19]. This modification incorporated a new term to the previous definition (it is described as a 'catch' that is a sense of resistance to the passive movement) and also set a condition (the «catch» occurs only above a threshold). The key element for differentiating spasticity, according to this definition is the appearance of «catch» when exceeding an arbitrary threshold. Therefore, we could say that this modification did not significantly contribute to the original definition of Lance.

More recently, members of the SPASM cooperative debated their arguments on the insufficiency of the previous definitions in clinical practice because of their narrow limits and thus they proposed the extension of the term to "a disturbed sensory-mobility control, derived from the upper motor neuron syndrome, presented as an intermittent or persistent activation of the muscles [19]. This definition seems to be shifting the definition focus to the inclusion of the modern pathophysiological spasticity view and the clinical practice. Thus the term
"spasticity" can now be used to describe the more "positive elements" associated with the upper motor neuron syndrome.

However, this definition may exclude pathological movement patterns arising during voluntary movements and to exclude all the "negative elements" associated with the upper motor neuron syndrome. (Note: the combined responses phenomenon may also occur in neurologically healthy people when trying to activities that involve maximum voluntary contractions. Therefore, it is not clear whether the resulting muscle activity should be faced as something undesirable and unintended). Whereas this definition may be more relevant to the clinical practice, the term may lose its usefulness if the researchers fail to identify what specific aspect of the spasticity is measured or studied.

There are two broad approaches to the definition of spasticity. The majority of the efforts are directed to providing a close and accurate description of the spasticity term. Although this practice is probably the most reliable, it seemed to be inconsistent with the common clinical cases so it was not as effective as expected.

The second type of definitions follows an opposed approach, with an attempt to define spasticity through listings of “umbrella” type including a broad scope of all possible variable manifestations of the phenomenon. Although the latter type of definitions is scientifically weaker, it provides the framework for the development of yet closely defined and highly accurate definitions. Consequently, the scientific community should take a decision on whether it should continue to support the narrower and precise definitions or it should reconsider by extending the boundaries of the definitions.

Briefly, it is reasonable to conclude that there is an adequate definition of the spasticity phenomenon. Through the currently available definitions, the SPASM cooperative
proposed a wider framework for spasticity definition, providing a starting point for the development of the future clinically usable definitions.

In conclusion we could say that the most popular definition of spasticity is based on the hyper-excited reflexes arising as a result of the upper motor neuron syndrome:

«Spasticity is a motor disorder characterized by a motion speed-dependent hypertonia and increased tendon reflexes arising from the suspension failure of the tendon reflexes and occurs as a component of the upper motor neuron syndrome» (Lance, 1980).

However, in the clinical practice, spasticity is often interpreted as a single entity which may include enhanced reflexes, rigidity, resistance to the passive movement, etc. Thus, in spasticity neural or no neural elements may be included.

Therefore, given the complexity of the term, the SPASM cooperative tried to create a broader more functional definition of spasticity:

«Assuming that all the involuntary activities involve the reflexes, then spasticity is the intermittent or persistent involuntary hyperactivation of the skeletal muscles associated with the upper motor neuron syndrome».

But whether this new definition of spasticity is more accurate should be determined in the future.

3. **Differential Diagnosis**

3.1 **The most common causes**

The knowledge of neuroanatomy is extremely useful for developing a differential diagnosis. We therefore examine the possible causes of spasticity regionally:
Spinal cord: We will consider here space-occupying lesions of the spinal cord, amyotrophic lateral sclerosis, Friedreich's ataxia, transverse myelitis, neurosyphilis, multiple sclerosis and anterior spinal artery occlusion. Advanced syringomyelia may also be a cause.

Brainstem: Common causes of spasticity here are stem tumors, cerebral hemorrhage, basilar artery thrombosis, multiple sclerosis, encephalomyelitis and neurosyphilis.

Cerebral hemispheres: Once again, the space-occupying lesions are still important, as well as the cerebral hemorrhage, embolism and thrombosis. For the children it is wise to consider the cerebral palsy, encephalitis and Schilder’s disease. There are many degenerative diseases of the cerebellum, leading to spasticity but until they are diagnosed the disease has progressed. The same occurs to multiple sclerosis affecting the cerebral cortex.

Miscellaneous: Stiffman syndrome is associated with muscle stiffness in the neck, torso and limbs. The location of the lesion is unknown.

In overal, spasticity is usually caused by lesions to a part of the brain or the spinal cord that controls the voluntary movements. This may result from spinal cord lesion, multiple sclerosis, cerebral palsy, anoxic brain injuries, brain injury or cranial trauma, stroke, toxoplasmosis, vitamin B12 deficiency and metabolic diseases such as Adrenoleukodystrophy, Amyotrophic lateral sclerosis (Lou Gehrig's disease), and phenylketonuria.

A more complete list of the possible causes is listed below.

4. **Other causes probably related to spasticity:**

   • 3-Methylglutaconic aciduria type 1
   • 3-Methylglutaconic aciduria type 3
   • 3-methylglutaconic aciduria, type 4 - progressive spasticity
• Acidic dry cell batteries inhalation poisoning - spasticity
• ADANE - spasticity
• Adrenoleukodystrophy
• Agyria - spasticity
• Agyria-pachgyria type 1 - spasticity
• Aicardi Goutieres syndrome
• Aicardi-Goutieres syndrome 1 - spasticity
• Aicardi-Goutieres syndrome 2 - spasticity
• Aicardi-Goutieres syndrome 3 - spasticity
• Aicardi-Goutieres syndrome 4 - spasticity
• Aicardi-Goutieres syndrome 5 - spasticity
• Akesson syndrome - spasticity
• Alexander disease
• Alexander Syndrome - spasticity
• Alpers Syndrome - spasticity
• Alpha-ketoglutarate dehydrogenase deficiency - spasticity
• Amyloidosis, oculoleptomeningeal - spasticity
• Amyotrophic lateral sclerosis - spasticity
• Amyotrophic lateral sclerosis 2, juvenile - spastic gait
• Amyotrophic lateral sclerosis 3 - spasticity
• Amyotrophic lateral sclerosis 4, juvenile - spasticity
• Amyotrophic lateral sclerosis 5 - spasticity
• Amyotrophic lateral sclerosis 6 - spasticity
• Amyotrophic lateral sclerosis 7 - spasticity
• Amyotrophic lateral sclerosis 8 - spasticity
• Amyotrophic lateral sclerosis, 11 - spasticity
• Amyotrophic lateral sclerosis, type 6 - spasticity
• Angiokeratoma -- mental retardation -- coarse face - spasticity
• Anoxic brain damage
• Anoxic brain injury
• Arginase deficiency - spasticity
• Arthritis -- short stature -- deafness - spasticity
• Aspartoacylase deficiency
• Aspartylglucosaminidase deficiency - spasticity
• Aspartylglucosaminuria - spasticity
• Ataxia spastic congenital miosis - spasticity
• Ataxia, Hereditary, Autosomal Dominant - spasticity
• Ataxia, spastic, 3, autosomal recessive - spasticity
• Bahemuka Brown syndrome - spasticity
• Basal Ganglia Disease, Adult-Onset - spasticity
• Bd syndrome - spasticity
• Bonneman-Meinecke-Reich syndrome - spasticity
• Brain -- bone -- fat - spasticity
• Brain malformations
• Brain trauma
• Bruyn-Scheltens syndrome - spasticity
• CACH syndrome - spasticity
• CAMFAK syndrome - spasticity
• Cerebral Autosomal Recessive Arteriopathy with Subcortical Infarcts and Leukoencephalopathy - spasticity
• Cerebral palsy
• Cerebral Palsy, Spastic Quadriplegic, 1 - spasticity
• Cerebral Palsy, Spastic Quadriplegic, 2 - spasticity
• Cerebral Palsy, Spastic Quadriplegic, 3 - spasticity
• Cerebrorenodigital syndrome - spasticity
• Cerebrotendinous Xanthomatosus - spasticity
• Cerebrovascular accident
• Ceroid lipofuscinoses, neuronal 2, late infantile type - spasticity
• Ceroid lipofuscinoses, neuronal 4 - spasticity
• Charlevoix-Saguenay spastic ataxia
• Chemical poisoning -- Acidic dry cell batteries - spasticity
• Chondrodysplasia Punctata, Rhizomelic type - spasticity
• Chromosome 1, monosomy 1q25 q32 - spasticity
• Chromosome 11q partial deletion - spasticity
• Chromosome 18 trisomy syndrome
• Chromosome 18, Tetrasomy 18p - spasticity
• Chromosome 2p deletion syndrome - spasticity
• Chromosome 7, monosomy 7q3 - spasticity
• Chylomicron retention disease with Marinesco-Sjogren syndrome - spasticity
• Coenzyme Q 10 (CoQ10), deficiency - spasticity
• Coffin syndrome 1 - spasticity
• Congenital Gigantism with Skeletal Dysplasia - spasticity
• Corneal cerebellar syndrome - spasticity
• Corpus callosum dysgenesis X-linked recessive - spasticity
• Cree leukoencephalopathy - spasticity
• Creutzfeldt-Jakob disease
• Cutis verticis gyrata mental deficiency - spasticity
• Cystinuria -- lysinuria - spasticity
• Dandy-Walker malformation with mental retardation, basal ganglia disease, and seizures - spasticity
• De Sanctis-Cacchione syndrome - spasticity
• Degenerative motor system disease - spasticity
• Del (3) (pter-25.3) - spasticity
• Dementia, familial British - spasticity
• Dihydropyrimidine dehydrogenase deficiency
• Dystonia-Parkinsonism, Adult-Onset - spasticity
• Encephalo cranio cutaneous lipomatosi s - spasticity
• Encephalocele anterior - spasticity
• Encephalocele frontal - spasticity
• Encephalopathy -- intracranial calcification -- growth hormone deficiency -- microcephaly -- retinal degeneration - spasticity
• Encephalopathy due to sulphite oxidase deficiency - spasticity
• Epileptic encephalopathy, early infantile, 1 - spasticity
• Epileptic encephalopathy, early infantile, 3 - spasticity
• Fahr's Syndrome - spasticity
• Familial British dementia
• Fischer Syndrome - spasticity
• Franek-Bocker-Kahlen syndrome - spasticity
• Fucosidosis - spasticity
• Gangliosidosis GM1 type 3 - spasticity
• Gangliosidosis, generalized GM1 type 3 - spasticity
• Gaucher disease type 2 - progressive spasticity
• Glucose transport defect, blood-brain barrier - spasticity
• Glut-1 Deficiency Syndrome - Spasticity
• GM1 gangliosidosis, type 2
• Graeck-Imerslund disease - Spasticity
• Grasbeck-Imerslund Disease - spasticity
• Griscelli syndrome type 2
• Grix-Blankenship-Peterson syndrome - spasticity
• Gustavson syndrome - spasticity
• Hallervorden-Spatz disease - spasticity
• Hallervorden-Spatz Syndrome - Spasticity
• HARP syndrome
• Head injury
• Head trauma
• Hereditary spastic paraparesis
• Herpes, Neonatal - spasticity
• Herpes, Neonatal -- Central Nervous System Infection - spasticity
• Holoprosencephaly deletion 2p - spasticity
• Homocarnosinase deficiency
• Howard-Young syndrome - spasticity
• Hyperexplexia
• Hyperglycinemia - spasticity
• Hyperornithinemia-hyperammonemia-homocitrullinuria syndrome - spasticity
• Hypomyelination -- hypogonadotropic hypogonadism -- hypodontia - spasticity
• Hypoxic episode
• IBIDS syndrome - spasticity
• Ichthyosis mental retardation asymptomatic spasticity - spasticity
• Inborn errors of thyroid hormone synthesis related to hypothyroidism - spasticity
• Incontinentia Pigmenti - spasticity
• Infantile sialic acid storage disorder - spasticity
• Isaacs-Mertens syndrome
• Ischemic episode
• Jakob-Creutzfeldt Disease
• Johnston Aarons Schelley syndrome - spasticity
• Kernicterus - spasticity
• Koone-Rizzo-Elias syndrome - spasticity
• Krabbe disease
• Krabbe leukodystrophy - spasticity
• Kuf Disease - Spasticity
• Laurence-Moon syndrome
• Lesch-Nyhan syndrome - spasticity
• Leukoencephalopathy -- palmoplantar keratoderma - spasticity
• Lindstrom syndrome - spasticity
• Lison syndrome
• Lissencephaly -- immunodeficiency - spasticity
• Lissencephaly type 1, due to LIS 1 anomalies - spasticity
• Lubs X-linked mental retardation syndrome - spasticity
• Lupine poisoning - spasticity
• MacDermot-Winter syndrome - spasticity
• Machado-Joseph Disease - spasticity
• Malaria (malignant tertian)
• Malformations in neuronal migration - spasticity
• Marinesco-Sjogren syndrome - Spasticity
• Massa-Casaer-Beulemans syndrome - spasticity
• Medulloblastoma - spasticity
• Megalencephalic leukoencephalopathy with subcortical cysts - spasticity
• Megaloblastic Anemia 1 - spasticity
• Meningoencephalitis
• Mental retardation -- spasticity -- ectroductyly - spasticity
• Mental retardation athetosis microphthalmia - spasticity
• Mental retardation progressive spasticity - progressive spasticity
• Mental retardation progressive spasticity, X-linked - progressive spasticity
• Mental retardation, X-linked -- Dandy Walker malformation -- Basal ganglia disease -
  - Seizures - spasticity
• Mental retardation, X-linked -- hypotonia -- recurrent Infections - spasticity
• Mercury -- Teratogenic Agent - spasticity
• Methylmalonic aciduria -- microcephaly -- cataract - spasticity
• Methylmercury -- Teratogenic Agent - spasticity
• MGA 4 - progressive spasticity
• Microcephaly -- mental retardation -- spasticity -- epilepsy - spasticity
• Microcephaly brain defect spasticity hypernatremia - spasticity
• Microlissencephaly -- micromelia - spasticity
• Micophthalmia -- brain atrophy - progressive spasticity
• Micophthalmia -- mental deficiency - spasticity
• Micophthalmia and mental deficiency - spasticity
• Micophthalmia syndromic, type 10 - spasticity
• Mild citrullinemia - spasticity
• Miller-Dieker syndrome - spasticity
• Mixed Cerebral Palsy - spasticity
• Motor and cognitive disorder due to sepiapterin reductase deficiency - spasticity
• Motor neuron disease
• Mucopolysaccharidosis type 2 Hunter syndrome- severe form - spasticity
• Multiple sclerosis
• N syndrome - spasticity
• N-acetyl-alpha-D-galactosaminidase - spasticity
• Near-drowning
• Nephronophthisis familial, adult -- spastic quadriplegia - spasticity
• Nerve sheath neoplasm - spasticity
• Neuroaxonal dystrophy -- renal tubular acidosis - spasticity
• Neurodegeneration With Brain Iron Accumulation 2 - spasticity
• Neurodegenerative syndrome, X-linked, Hamel type - spasticity
• Neuroferritinopathy - spasticity
• Neuroferritinopathy (adult-onset basal ganglia disease) - spasticity
• Neuromyelitis Optica - spasticity
• Neuronal intranuclear inclusion disease - spasticity
• Neuropathy sensory spastic paraplegia - spasticity
• Niemann-Pick disease - spasticity
• Niemann-Pick Disease, Type C
• Niemann-Pick disease, type D - spasticity
• Non-ketotic hyperglycinemia - spasticity
• Nonbullous congenital ichthyosiform erythroderma
• Nyssen-Van Bogaert-Meyer syndrome - spasticity
• Oculopatocerebral syndrome - spasticity
• Oral-facial-digital syndrome type 3
• Ossification of the posterior longitudinal ligament of the spine - spastic gait
• Ovarioleukodystrophy - spastic gait
• Parkinson disease 9 (PARK9) - spasticity
• Parkinsonism, early onset with mental retardation - spasticity
• Pelizaeus-Merzbacher brain sclerosis - spasticity
• Pelizaeus-Merzbacher disease - spasticity
• Pelizaeus-Merzbacher disease, adult onset - progressive spasticity
• Perinatal asphyxia
• Periodic hyperlysinemia - spasticity
• Periventricular leukomalacia
• Pernicious anemia - spasticity
• Phenylketonuria - spasticity
• PIBIDS syndrome - spasticity
• Pilo dento unguar dysplasia -- microcephaly - spasticity
• Polynephopathy, Hearing Loss, Ataxia, Retinitis Pigmentosa and Cataract - spasticity
• Pontocerebellar hypoplasia with infantile spinal muscular atrophy - spasticity
• Powell-Venencie-Gordon syndrome - spasticity
• Premature chromosome condensation with microcephaly and mental retardation - spasticity
• Prenatal utero-placental insufficiency
• Primary Lateral Sclerosis - spasticity
• Primary lateral sclerosis, adult - spastic gait
• Progressive Rubella Panencephalitis - progressive spasticity
• Proud-Levine-Carpenter syndrome - spasticity
• Pseudo-torch syndrome - spasticity
• Purine nucleoside phosphorylase (PNP) deficiency
• Pyruvate dehydrogenase phosphatase deficiency - spasticity
• Renier-Gabreels-Jasper syndrome - spasticity
• Retinis pigmentosa -- deafness -- hypogenitalism - spasticity
• Rett-like syndrome - spasticity
• Rhizomelic chondrodysplasia punctata, type 1 - spasticity
• Rhizomelic chondrodysplasia punctata, type 3 - spasticity
• Richards-Rundle syndrome - spasticity
• Rubella panencephalitis - progressive spasticity
• Schimke, X-linked, mental retardation syndrome - spasticity
• Schindler disease - spasticity
• Schindler disease, type 1 - spasticity
• Schinzel Giedion Syndrome - spasticity
• Segawa Syndrome - spasticity
• Selective Vitamin B12 malabsorption with Proteinuria - spasticity
• Severe head injury
• Sialuria, Finnish type - spasticity
• Sjogren-Larsson syndrome
• Space occupying lesion
- Spastic paraparesis - spasticity
- Spastic paraplegia 10, autosomal dominant - spastic gait
- Spastic paraplegia 14, autosomal recessive - spastic gait
- Spastic paraplegia 17 - spastic gait
- Spastic paraplegia 2, X-linked - spastic gait
- Spastic paraplegia 23 - spastic gait
- Spastic paraplegia 3, autosomal dominant - spastic gait
- Spastic paraplegia 31, autosomal dominant - spastic gait
- Spastic paraplegia 4, autosomal dominant - spastic gait
- Spastic Paraplegia 42, Autosomal Dominant - spastic gait
- Spastic paraplegia 7, autosomal recessive - spastic gait
- Spastic paresis -- glaucoma -- mental retardation - spasticity
- Spastic quadriplegia -- retinitis pigmentosa -- mental retardation - spasticity
- Spasticity -- mental retardation - spasticity
- Spasticity -- mental retardation -- epilepsy, X-linked - spasticity
- Spasticity -- multiple exostoses - spasticity
- Sphingolipidosis - spasticity
- Spina bifida
- Spinal atrophy -- ophthalmoplegia -- pyramidal syndrome - spasticity
- Spinal cord injury, acute
- Spinal cord injury, chronic phase
- Spinal tumor
- Spinocerebellar ataxia 3 - spasticity
- Spinocerebellar ataxia, autosomal dominant - spasticity
- Spinocerebellar ataxia, autosomal recessive 5 - spasticity
- Spinocerebellar ataxia, Machado-Joseph type I - spasticity
- Spinocerebellar ataxia, Machado-Joseph type II - spasticity
- Spinocerebellar ataxia, Machado-Joseph type III - spasticity
- Spinocerebellar ataxia, Machado-Joseph type V - spasticity
- Spinocerebellar ataxia, X-linked, 2 - spasticity
- Spinocerebellar degenerescence, book type - spasticity
• Stratton-Parker syndrome - spasticity
• Stroke
• Sturge-Weber syndrome
• Subacute Sclerosing Panencephalitis - spasticity
• Suffocation
• Superficial siderosis of the central nervous system - spasticity
• Symmetrical thalamic calcifications - spasticity
• Syringomelia - spasticity
• Syringomyelia, lumbar lesion - spasticity
• Thalamic degeneration symmetrical infantile - spasticity
• The Methylmalonic Acidemias - spasticity
• Thompson-Baraitser syndrome - spasticity
• Thrombocytopenia -- cerebellar hypoplasia -- short stature - spasticity
• Tome-Brune-Fardeau syndrome - spasticity
• Trauma
• Triose phosphate-isomerase deficiency - spasticity
• Troyer syndrome
• Tumor
• Upper motor neuron weakness - spasticity
• Upper motor neurone lesion
• Van Bogaert disease - spasticity
• Van Bogaert-Scherer-Epstein Disease - spasticity
• Vanishing white matter leukodystrophy - spasticity
• Variant CJD - spasticity
• Viral encephalitis
• Vocal cord dysfunction familial - spasticity
• Weaver Syndrome - progressive spasticity
• Wells Jankovic syndrome - Spasticity
• Woods Black Norbury syndrome - Spasticity
• X-linked alpha thalassemia mental retardation syndrome (ATR-X) - spasticity
• X-linked hydrocephalus spectrum - spasticity
5. The differential diagnostic approach

After having established the level of the lesion, an MRI or a CT scan of the region can be conducted. A lumbar puncture may be useful for determining the diagnosis of multiple sclerosis, encephalitis and neurosyphilis, if an invasive lesion is excluded.

5.1 Other useful tests

1. MRA (vascular involvement)

2. Evoked potentials (VEP), brainstem evoked potentials (BSEP) (multiple sclerosis)

3. Carotid Artery Duplex Scan (carotid stenosis or occlusion)

4. Four-vessel cerebral angiography

5. CBC, measurement of serum vitamin B12 (pernicious anemia)

6. Conclusion

Spasticity is a chronic disorder with muscular stiffness, muscle control and functionality reduction as a result of a variety of central nervous system lesions including
trauma, stroke, multiple sclerosis and cerebral palsy. With the proper neurological, physiotherapeutic, surgical, psychosocial interventions it can be treated by greatly enhancing the quality of patient’s life.

References


