# Spastic movement disorder: impaired reflex function and altered muscle mechanics

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**In clinical practice, signs of exaggerated tendon tap reflexes associated with muscle hypertonia are generally thought to be responsible for spastic movement disorders. Most antispastic treatments are, therefore, directed at the reduction of reflex activity. In recent years, however, researchers have noticed a discrepancy between spasticity as measured in the clinic and functional spastic movement disorders, which is primarily due to the different roles of reflexes in passive and active states, respectively. We now know that central motor lesions are associated with loss of supraspinal drive and defective use of afferent input with impaired behaviour of short-latency and long-latency reflexes. These changes lead to paresis and maladaptation of the movement pattern. Secondary changes in mechanical muscle fibre, collagen tissue, and tendon properties (eg, loss of sarcomeres, subclinical contractures) result in spastic muscle tone, which in part compensates for paresis and allows functional movements on a simpler level of organisation. Antispastic drugs can accentuate paresis and therefore should be applied with caution in mobile patients.**

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## Introduction

Spasticity is a well known syndrome, most commonly arising after stroke, multiple sclerosis, spinal cord injury, some traumatic brain injuries, and other CNS lesions. Many patients with a spinal or cerebral lesion have a spastic movement disorder, with slowing of stepping and of voluntary limb movements. Clinical diagnosis of spasticity is based on the combination of physical signs in relaxed patients—ie, exaggerated tendon reflexes and muscle hypertonia defined as a velocity-dependent resistance of a muscle to stretching.[1] In this review, we relate the above definition of spasticity to the knowledge of the mechanisms underlying the associated movement disorder.

Descending overactivity causing exaggerated reflexes might be responsible for muscle hypertonia, which then leads to spastic movement disorder.[2–6] This view is supported by experiments on decerebrate cats:[7] muscle tone during stretching is substantially reduced after severing the nerves involved in the stretch-reflex loop. Therefore, the intention of most treatment approaches is to attenuate or abolish reflex activity and thereby to reduce muscle tone.[2,8] However, this dominant view does not take into account four important points. First, exaggerated tendon reflexes are only a small part of the reflex mechanisms involved in the control of functional movement, such as walking. Second, most studies on the effect of antispastic drugs are focused on isolated clinical signs, such as reflex activity, and not on the spastic movement disorder that hampers patients. Third, without the development of spastic muscle tone (eg, after stroke), some patients would be unable to walk because of the paresis. Last, rigid muscle tone occurs immediately after decerebration of cats, whereas human spasticity develops over weeks after acute lesions.

No animal model exists for human spasticity, perhaps because the pathophysiology of spasticity is multifactorial. Any changes in the neuronal or biomechanical systems, because of, for example, differences in the site and duration of a central lesion, are of importance in determining which neural control mechanisms are deficient and contribute to the movement disorder.[9] Furthermore, such deviations might already be secondary and compensatory to the primary dysfunction of the motor system. There are differences in the appearance of spasticity between spinal and supraspinal lesions and lesions of different origin (eg, inflammatory or traumatic). However, these factors have little influence on the impairment of function.

Research on functional movement in recent years indicates that the clinical signs of spasticity are little related to the functional spastic movement disorder, which hampers patients and should be the focus of any treatment. For example, exaggerated reflexes, a dominant sign in clinical assessment, have little effect on the movement disorder. In this review, we describe the state of reflex behaviour and muscle mechanics in patients with spasticity and the resulting muscle tone during three conditions: passive (clinical), active non-functional (laboratory setting), and functional (walking).

## Clinical signs: passive condition

In a clinical setting, muscle tone and tendon tap reflexes are routinely examined in relaxed patients. Exaggerated tendon tap reflexes and an increased resistance of a muscle to stretching indicate the presence of spasticity caused by a central motor lesion.

### Short-latency stretch reflex

The nature and mechanisms underlying exaggerated tendon reflex activity (monosynaptic or oligosynaptic segmental reflexes) have been the focus of many studies in patients with spasticity. This short-latency reflex activity is mediated by fast conducting group Ia nerve fibres from the muscle spindles to the spinal cord. A severe acute central lesion is associated with a loss of tendon tap reflexes followed by hyper-reflexia due to neuronal reorganisation in both cats[10] and human beings.[11] New connections can cause changes in the strength of reflex excitability and denervation can cause hypersensitivity.[10]

Exaggerated reflexes might result from hyperactivity of fusimotoneurons[12,13] (also called gamma motor neurons), which corrrespond to the alpha motor neurons innervating normal muscle fibres, although only indirect approaches have been applied, and this has not been proven convincingly.[14–16] Furthermore, after a central lesion, increased electromyographic activity is not likely to be caused by either reduced recurrent inhibition of motor neurons via Renshaw cell activity[17,18] or intraspinal nerve sprouting.[19]

However, there is evidence for reduced presynaptic inhibition of Ia afferent fibres in the legs of paraplegic but not hemiplegic people.[20,21] Reduced Ia inhibition in the arms seems to be present on the hemiplegic side.[22] There is no association between decreased presynaptic inhibition of Ia afferents and the degree of muscle hypertonia as assessed by the clinical Ashworth scale.[21] In addition, deficient disynaptic reciprocal inhibition,[23] increased excitability of reciprocal Ia inhibitory pathways,[24–26] changed postactivation depression,[27] and disinhibition of group II pathways[28–30] might lead to hyper-reflexia in spasticity of spinal and supraspinal origin. Other mechanisms are probably also involved.[21]

A severe central motor lesion can be followed by flaccid paresis with loss of tendon tap reflexes. The H-reflex (an electrically elicited short-latency reflex excluding muscle spindles) is already present during spinal shock when tendon reflexes cannot be elicited.[31] After 1–2 weeks, tendon reflexes and muscle tone reappear. At later stages (4–6 weeks) clinical signs of spasticity (ie, exaggerated reflexes and increased muscle tone) become established. The loss of reflexes is attributed to reduced excitability of alpha and gamma motor neurons due to the sudden loss of input from supraspinal centres. When spasticity has developed, the threshold of the soleus stretch reflex is decreased in patients with hemiparetic spasticity,[32,33] possibly due to an increase in motor-neuron excitability.[34] However, repetitive, clonic muscle contractions are more likely to be associated with impaired interaction of central and peripheral mechanisms than with a recurrent stretch reflex activity.[35]

## Flexor reflex

The flexor reflex is a polysynaptic spinal reflex that might be connected with spinal locomotor centres.[36] The dominant view is that flexor reflexes are exaggerated after a central nervous lesion and cause muscle spasms after severe spinal cord injury.[37] Also, a spontaneous firing of motor neurons during rest might lead to muscle spasms,[38] initially caused by receptor upregulation and later by neuronal sprouting.[39,40]

The increase of flexor reflexes in patients with chronic spinal cord injury might represent a marker for neuronal plateau potentials.[41] Furthermore, it seems that the sites where flexor reflexes can be elicited become expanded in patients with a spinal or supraspinal lesion.[42,43] Otherwise, a great variability of flexion reflex responses exists in patients with spinal cord injury.[44]

After acute, complete spinal cord injury, flexor reflex excitability and spastic muscle tone develop in parallel.[31] However, after a few months, there is a divergent course in which the severity and occurrence of muscle spasms increase, whereas flexor reflex amplitude decreases.[31] In line with this, patients with complete chronic spinal cord injury have a low incidence of the early component of the flexor reflex[44,45] and flexion reflexes produce smaller leg joint torques than those in healthy people.[46] These observations suggest that the activity of flexor reflexes is little related to the occurrence of muscle spasms in spasticity of spinal origin.

## Muscle tone

Muscle hypertonia is clinically assessed using the Ashworth scale, and is defined as a velocity-dependent resistance to stretch. This is particularly true for the leg extensor[47,48] and arm flexor muscles[34,49] (ie, the antigravity muscles). In patients with chronic stroke, spastic muscle hypertonia (clinically defined as an increased resistance of a muscle to stretch) is associated with muscle activity measured by electromyography, which largely exceeds that seen in healthy people.[50,51] Thus, muscle hypertonia in clinical testing reflects a combination of intrinsic and reflex-mediated muscle stiffness. Also, the tone of the muscles on the non-affected side of patients with stroke are not completely normal; they show some increase in tone compared with the muscles of healthy controls.[52] Despite the extra electromyographic activity, which exceeds that observed in healthy subjects after muscle stretch, passive stiffness (eg, muscle contracture) at the ankle joint is also increased and contributes to clinically defined spastic muscle hypertonia after stroke.[53–55] In studies that have used a more complete analysis looking at all contributing factors, it becomes evident that the abnormal stretch reflex activity is insufficient to explain increased muscle tone in people with stroke or multiple sclerosis.[51,56–58] Reflex-mediated stiffness in the ankle plantar flexors[58] and elbow flexor muscles[34,50,59] in patients with stroke and spasticity is within the range of healthy controls and seems to be only slightly increased in patients with spinal cord injury.[60]

More recent studies indicate an increase in passive stiffness of a muscle to stretch in patients with stroke and spasticity due to changes in collagen tissue and tendons,[51,54,58] an enhancement of intrinsic stiffness of muscle fibres,[61] and a loss of sarcomeres,[62] leading to subclinical contractures. In addition, morphometric and histochemical investigations show changes in mechanical muscle-fibre properties[63–65] that might contribute to spastic muscle tone. Consequently, clinical muscle hypertonia in patients with stroke seems to be associated with subclinical muscle contracture rather than with reflex hyperexcitability.[57,62,66] Changes in biomechanical conditions of a muscle might also have

an important effect on the stretch reflex behaviour (possibly via group III/IV muscle afferents) in people with stroke.[67,68]

Exaggerated stretch or flexor reflexes elicited in passive muscles, as seen in clinical bedside examinations, are not solely responsible for the increased resistance of a spastic muscle to stretch. Secondary changes in intrinsic and extrinsic muscle properties contribute to spastic muscle tone. This interpretation is based mainly on observations made in patients with stroke. Corresponding results are, however, also reported for central motor lesions of different origin (eg, traumatic spinal cord injury and multiple sclerosis).[8]

## Non-functional movement: active muscle

Active muscle function in normal and impaired motor control is commonly investigated in a laboratory setting in which people can exert a controlled level of voluntary contraction. This method allows insight into the neuronal mechanisms underlying muscle tone regulation compared with the passive condition.

Voluntary elbow movements in patients with stroke are more disturbed by paresis than by antagonistic muscle hypertonia, even in those with marked spasticity—ie, increased muscle tone.[50,69] When background contractions are matched to normal levels in patients with spasticity, little evidence exists for exaggerated reflex activity.[58,61,70,71] However, during isotonic leg muscle contractions, modulation and inhibition of Ib afferents (innervating the force-sensitive Golgi tendon organs) is reduced[72] and some co-contraction of antagonistic arm muscles can occur.[73,74]

Studies that apply joint displacements in voluntarily activated limb muscles show different results from those obtained in the passive muscle. Most of these studies are done during isometric muscle contractions or isotonic movements of arms[50,59] and legs[60,75–77] with matched background electromyographic activity of corresponding muscles of the spastic and non-affected sides of patients with hemiplegic stroke. The studies show a uniform pattern of compensatory electromyographic responses to the displacements. In the unaffected muscles, the short-latency reflex is followed by a long-latency reflex response,[78,79] which never appears in a passive muscle condition—long-latency or polysynaptic reflexes are assumed to be mediated mainly by group II fibres on a spinal level (eg, during locomotion) and group I fibres on a supraspinal level (eg, hand movements). Compared with the short-latency reflexes they represent flexible, functionally essential reflex mechanisms; for details about the possible mechanisms and pathways underlying the long-latency reflexes, see elsewhere.[80] On the spastic-paretic side, this long-latency component is reduced or absent.[50,59,77] Nevertheless, the automatic resistance to the joint displacement is of similar amplitude on the affected and unaffected sides.

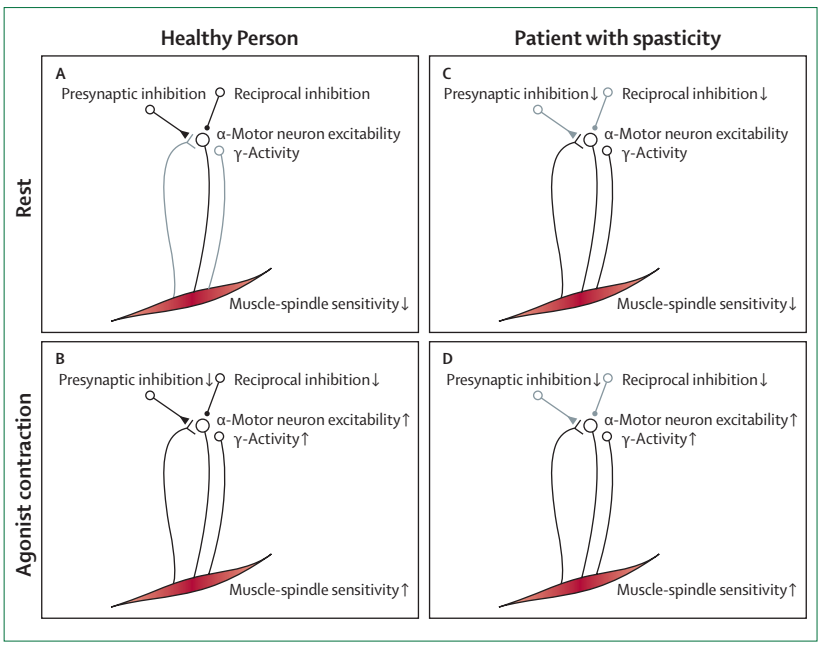


***Figure 1*: Short-latency reflex behaviour in passive and active muscle**
In healthy people, the stretch reflex activity is low at rest (A), which is explained by low excitability of spinal motor neurons, low muscle-spindle sensitivity, low discharge rate of Ia afferents, and pronounced presynaptic inhibition (Ib and Ia reciprocal inhibition). During voluntary contraction of the muscle (B) motor neuron excitability, spindle sensitivity, and Ia afferent discharge increase, whereas presynaptic inhibition (Ib inhibition and Ia inhibition) decreases. Stretch reflex activity is consequently high. In spasticity, presynaptic Ib and Ia inhibition is already decreased at rest (C) and stretch reflex activity is high already. During voluntary contraction (D) there is little change in these parameters and the stretch reflex activity is not very different from that at rest. The arrows designate whether the mechanism is decreased or increased during contraction compared with rest.[9]

During muscle contractions in healthy people, different inhibitory mechanisms on short-latency reflexes are removed.[9] By contrast, in spasticity, presynaptic inhibition, postactivation depression, and reciprocal inhibition do not further decrease during contraction (figure 1). Therefore, short-latency stretch reflexes in patients with spasticity are less different in size between the relaxed and active conditions compared with those in healthy people.[9,50] These reflexes are still prominent but show no task-dependent modulation on the spastic-paretic side compared with the unaffected side of patients with hemiparetic stroke.[50] This behaviour mainly concerns arm flexor[50] and leg extensor[75] muscles. In the ankle dorsiflexor[77] and arm extensor[50] muscles compensatory electromyographic responses are reduced or absent without a preceding short-latency reflex.

In the voluntarily contracted (non-functional) muscle of healthy people, reflex behaviour differs from that in the passive (clinical) condition. By contrast, in patients with spasticity the excitability state remains roughly unchanged in the passive and voluntarily activated muscles. In a non-functional perturbation task, the overall electromyographic response is usually reduced on the spastic side despite exaggerated short-latency stretch reflexes due to the loss of functionally important longer latency reflex components.

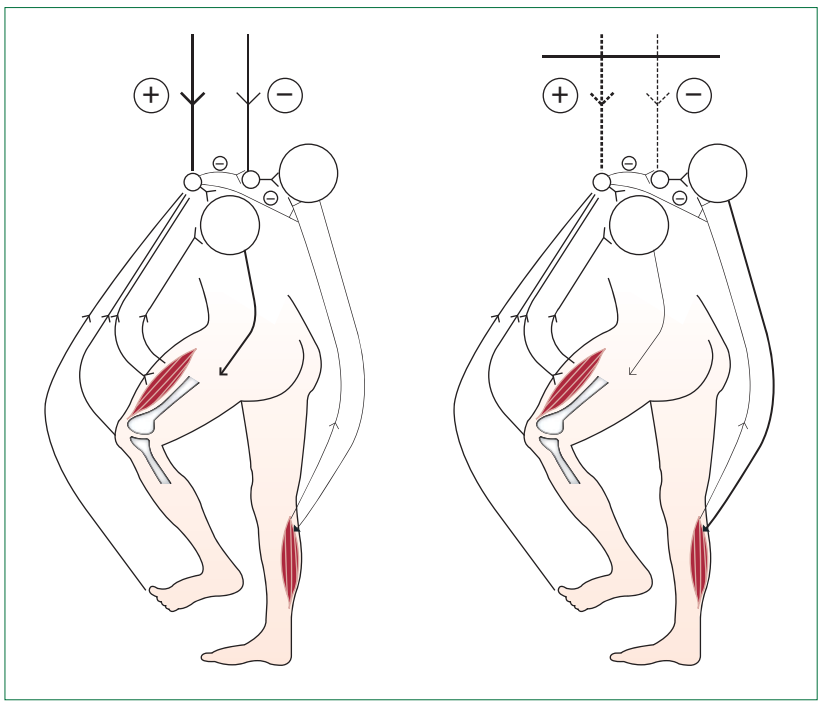

*Figure 2:* **Reflex behaviour during human gait**
Left: In the healthy physiological condition, long-latency reflex activity is facilitated by supraspinal drive and becomes significantly involved in leg muscle activation to adapt the locomotor pattern to the ground conditions. Ia afferent-mediated inputs are inhibited. Right: after a spinal or supraspinal lesion, the functionally essential activity of long-latency reflexes is impaired owing to the loss of supraspinal input.[87]

## Functional movement: walking

After central motor lesions, patients have movement disorder. To achieve adequate treatment, it is crucial to address the mechanisms underlying the impaired function. Several studies indicate that the clinical signs of spasticity are not related to the movement disorder. In this section, we discuss some of the mechanisms underlying the impaired movement.

### Pattern of leg muscle activation

During a functional movement, such as locomotion, patients with spastic hemiparesis or paraparesis have typical patterns of leg muscle activation recorded with electromyography. Spastic gait is associated with a low level of leg muscle activity compared with that in the unaffected side of hemiparetic patients or in healthy people.[75,76,79] The reduction depends on the severity of paresis. Furthermore, after stroke, gait recovery during rehabilitation is not associated with changes in walking pattern.[81] The timing of the pattern (ie, the reciprocal activation of antagonistic leg muscles) is preserved in spasticity of spinal and supraspinal origin.[79,82,83] Only rarely does some coactivation of antagonistic leg muscles occur during the stance phase.[84–86] Premature leg extensor activation during the stance phase of gait[85] depends on the plantar-flexed position of the spastic-paretic foot. Premature leg extensor activation in the early stance phase, or even before impact, also occurs when healthy people walk by voluntarily tip-toeing (ie, the extensor activation depends on the foot position before impact). Furthermore, coactivation of antagonistic leg muscles can be recorded in healthy people when they are walking with slightly flexed knees (Dietz V, unpublished).

In a few patients with spasticity, the impact of the forefoot is associated with the appearance of stretch-reflex potentials.[84] The leg extensor amplitude modulation in electromyography, which healthy people typically have during the stance phase, is reduced or lacking[87] (figure 2). In line with this, the contribution of afferent feedback to the ongoing locomotor soleus activity is low in people with spasticity.[88]

Overall, evidence gained from studies on functional movements shows that our clinical spasticity measures do not relate to problems in walking after stroke.[89] Equilibrium control during upright standing is similarly little affected by monosynaptic reflex hyperexcitability, but more by reduced long-latency reflex components.[90]

### Reflex behaviour

In healthy people, group Ia afferent input to the spinal cord becomes suppressed during the stance phase of gait.[78,87] Because of reduced Ia suppression in spasticity, short-latency stretch reflexes commonly appear in the leg extensor muscles during the transition from the swing to the stance phase of gait, which is rarely the case in healthy people or in the unaffected side of patients with spastic hemiparesis. Furthermore, the inability to suppress reflex excitability during the swing phase of gait might contribute to impaired walking.[87,91–96]

During walking in healthy people, the H reflex and short-latency stretch reflex (both mediated by group Ia afferents) in leg muscles become modulated in a specific way.[91,92] In people with spastic paresis, this physiological reflex modulation is impaired.[92–96] Also, the modulation of cutaneous reflexes is reduced during gait.[94] In line with this, the fast regulation of motor-neuron discharge, which characterises functional muscle activation, is absent in spasticity.[63,97] The quadriceps-tendon jerk-reflex depression, which is present in healthy people, is absent in patients with spinal lesions and is associated with a loss of modulation during the step cycle. These changes are less pronounced in patients with cerebral lesions;[92] besides this, there are no other known qualitative differences in reflex behaviour between spasticity of cerebral origin and that of spinal origin,[92] although direct comparisons are rare.

During perturbations of gait (eg, short acceleration impulses of the treadmill during the stance phase of stepping) in the unaffected leg, short-latency stretch-reflex components are followed by large compensatory long-latency (or polysynaptic) reflexes in extensor[80,87,98] and dorsiflexor muscles.[99] By contrast, in the spastic leg, short-latency reflexes are isolated without a significant long-latency electromyographic component.[75,100] After stance displacements associated with a stretch of the leg

flexor muscles, the amplitude of the compensatory tibialis anterior electromyographic response is smaller on the spastic side than on the unaffected side without a preceding short-latency reflex potential.[99,101] Hence, there is similar reflex behaviour during displacements applied to activated limb muscles in both non-functional and functional conditions. These findings might result from impaired use of afferent input by spinal neuronal circuits after central lesions. The consequence is reduced adaptation of muscle activity to the ground conditions,[88] which, together with the reduced capacity to modulate reflex activity over the normal range, might contribute to the spastic movement disorder.[70,87]

### Tension development

Muscle tone, as defined clinically, cannot be examined during movement. However, tension development at the Achilles tendon, resulting from a combination of muscle stiffness and electromyographic activity, can be recorded. Tension development differs between the affected and unaffected legs in patients with stroke and spastic hemiparesis.[75] On the unaffected side, changes in tension at the Achilles tendon parallel the amplitude of triceps surae electromyographic activity. On the spastic side, the tension development is associated with a stretching of the triceps surae during the stance phase of gait. During this period, the leg extensor muscles are tonically activated with low electromyographic amplitude.[75] This is interpreted as tension development on a simpler level of organisation on the spastic side due to changes in mechanical properties of the leg extensor muscles. The possible mechanisms underlying these changes are outlined above. Thus, secondary to a cerebral or spinal lesion, there is a major alteration in the normal muscle–joint relationships[62,102,103] that allow for support of the body during stepping movements.

Recent studies on spastic movement disorder provide evidence that the central pattern of leg muscle activation is largely preserved after a central lesion and the clinically dominant hyper-reflexia is little involved in spastic movement disorder. Impaired function and attenuation of long-latency (polysynaptic) reflexes hamper walking. Secondary to a central lesion, changes in muscle, ligament, and tendon properties occur. No qualitative difference exists between spasticity of cerebral and spinal origin. The obvious consequence is the regulation of muscle tone on a simpler level. This behaviour of the spastic muscle allows for the support of the body during walking. Therefore, such changes should not be considered as pathological, but rather as adaptive to a primary disorder. They may even be viewed as optimum for a given state of the system of movement production.[104] Knowledge about the nature of these changes in muscle mechanics is still rudimentary.

## Cerebral palsy

Children with perinatal lesions of the central motor system share some characteristics of spasticity with adults. However, owing to the early onset of the damage, impaired motor system development influences the mechanisms contributing to spasticity.

Although neurophysiological studies indicate non-homogeneous states of muscle tone in children with cerebral palsy,[105] typical features exist during walking. The leg muscle activity underlying the walking of children with congenital cerebral palsy has characteristic signs of impaired maturation of the normal gait pattern—ie, it closely resembles that of stepping in newborn infants.[106,107] The electromyographic pattern recorded in young adults with cerebral palsy consists of a coactivation of antagonistic leg muscles with a reduced and tonic mode of electromyographic activity and the appearance of isolated electromyographic potentials mainly in the leg extensor muscles after ground contact.[108,109] Also, a short-latency reflex irradiation, usually observed in healthy infants of less than 2 years of age[110–112] is present in children with cerebral palsy, which suggests that the early infant stepping pattern persists in these children.[113]

When the cerebral lesion is acquired at a late stage and the reciprocal mode of leg muscle activity is already established (ie, at around the age of 4 years), reciprocal activation of antagonistic leg muscles remains preserved during spastic gait, similar to that in stroke patients.[75] As in adults with spasticity, there is no correlation between the clinical signs of exaggerated stretch reflexes and spastic muscle tone.[114] Studies indicate abnormalities in the viscoelastic properties of muscles with intramuscular contractures at an early stage,[105,108] as in adult patients. These changes might result in a gait equinus, which might be due to a paresis of the foot flexors but is postulated to be a contracture of the extensor muscle.[115]

In conclusion, children with cerebral palsy share some clinical signs and mechanisms underlying movement disorder with adults with spasticity. Impaired corticospinal input during development, associated with a deficient modulation of spinal interneuronal circuits, might lead to abnormal reciprocal inhibition in children with cerebral palsy during walking. Such a mechanism might contribute to the coactivation pattern.

## Therapeutic consequences

Any treatment of spasticity should focus on the specific movement disorders of individual patients. In most cases, the physical signs obtained during the clinical examination are an epiphenomenon rather than the cause of the functional condition. Recent studies have shown that functional movements involve essential reflex mechanisms that are not assessed with clinical tests (figure 3). Nevertheless, site, origin, and severity of a central motor lesion can influence the clinical appearance of spasticity and have to be taken into account for the appropriate treatment of individual patients.

The dominant view is that treatment of spasticity should be directed towards a reduction of stretch reflex

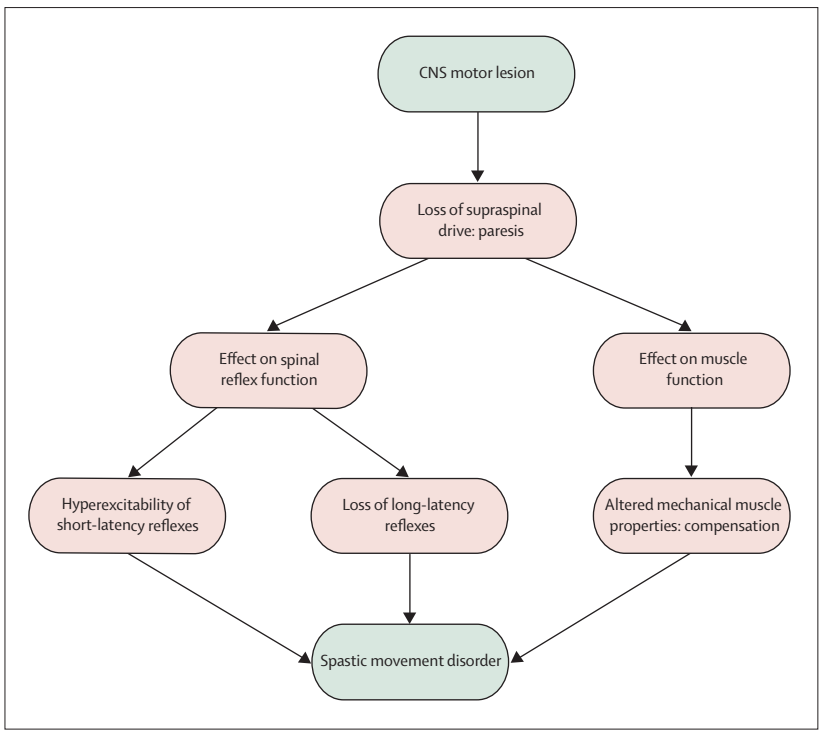


***Figure 3:*** **Mechanisms involved in spastic movement disorder**
A central motor lesion leads to changes in the excitability of spinal reflexes and a loss of supraspinal drive. As a consequence, changes in muscle function occur and lead to altered mechanical muscle properties. The combination of all sequelae of the primary lesion leads to the spastic movement disorder.[87]

activity. This treatment approach is primarily based on studies of muscle tone and reflex activity under passive conditions (although treatment with Botulinum toxin type A is commonly based on electromyographic recordings made during active movements).

Investigations of functional leg and arm movements show no causal relationship between exaggerated reflexes and movement disorder following a spinal or supraspinal lesion. Impaired walking might be mainly caused by disabling paresis and impaired use of afferent input by spinal neuronal circuits. As a result, antispastic medications that are directed to reduce clinical signs of spasticity, such as exaggerated reflexes and muscle tone, do not improve the movement disorder.[116–120] Medication can even increase weakness,[118,121,122] which might interfere with functional movements, such as walking. By contrast, cannabinoids improve mobility in patients with multiple sclerosis but have no effect on spastic muscle tone.[123] In children with spastic diplegia, selective dorsal rhizotomy (which reduces afferent input to the spinal cord) combined with physiotherapy results in an improvement in mobility similar to that observed in those not receiving the procedure.[124,125] However, some changes in gait mechanics were reported after treatment.[126] Similarly, Botulinum toxin type A is assumed to result in a largely cosmetic effect on spastic signs commonly without functional improvement,[121,127] although this toxin might reduce the activity of the intrafusal fibres.[128,129] Intrathecal baclofen might also reduce hyperactive reflexes without producing significant weakness.[130–132]

In conclusion, therapeutic interventions in patients with spastic paresis of either spinal or cerebral origin should be focused on the training, relearning and activation of residual motor function,[133,134] and the prevention of secondary complications, such as muscle contractures.[135] There have been few controlled studies documenting the positive effect of a functional training programme in cerebral palsy.[125,136]

Antispastic drug therapy might predominantly benefit immobilised patients by reducing muscle tone and relieving muscle spasms,[137] which might in turn improve nursing care for these patients.

### Search strategy and selection criteria

References for this review were identified through searches of MEDLINE between 1990 and April 2007 with the search terms "spasticity", "spastic movement disorder", "exaggerated reflexes", "muscle hypertonia", and "central motor lesion". References were also identified through searches of the references of relevant articles and the authors' own files. More recent publications were preferred. Only papers published in English were reviewed. The final list was generated on the basis of originality and relevance.

## Conclusion

This review describes the differential roles of background and reflex activity as well as muscle fibre function in passive, active, and functional movement disorders after a central motor lesion. According to research, exaggerated reflexes have a minor role and secondary changes in mechanical muscle fibre properties have a major role in spastic movement disorder as suggested by the results of clinical assessment. In functional movements, changes in muscle fibre properties leading to spastic muscle tone are needed to compensate for the loss of neuronal drive. Further studies are needed to understand the regulation and importance of spinal and descending control mechanisms during movement and to detail the intracellular and extracellular modifications of skeletal muscle that occur secondary to a spinal or supraspinal lesion. This might help in the development of novel therapeutic interventions to improve anti-spastic treatments in patients with overshooting spasticity.

**Contributors**
VD prepared the first draft, which VS modified and supplemented.

**Conflicts of interest**
We have no conflicts of interest.

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