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Published in:
Lancet Neurology

DOI (link to publication from Publisher):
10.1016/S1474-4422(07)70193-X

Publication date:
2007

Document Version
Accepted author manuscript, peer reviewed version

The Lancet Neurology - Volume 6, Issue 8, August 2007, Pages 725-733 - DOI: 10.1016/S1474-4422(07)70193-X

Spastic movement disorder: impaired reflex function and altered muscle mechanics

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Abstract

In clinical practice the dominant view is that the signs of exaggerated tendon tap reflexes associated with muscle hypertonia are responsible for the spastic movement disorder. Consequently, most anti-spastic treatments are directed at reducing reflex activity. During the last years an increasing body of evidence suggests a discrepancy between clinical spasticity and spastic movement disorder.This is primarily due to the different role reflexes play in the passive and active condition, respectively. Today we know that a central motor lesion is associated with a loss of supraspinal drive and a defective utilization of afferent input with an impaired behaviour of short- and long-latency reflexes. This leads to a paresis and a mal-adaptation of the movement pattern. Secondary changes in mechanical muscle fibre, collagen tissue and tendon properties (e.g. loss of sarcomers; sub-clinical contractures) result in spastic muscle tone, which at part compensates for paresis. This allows functional movements on a simpler level of organisation. Anti-spastic drugs can accentuate paresis and therefore should be applied with caution in mobile subjects.

Introduction

Spasticity is a consequence of a central nervous system lesion. It is a well known syndrome, most frequently seen after stroke, multiple sclerosis, spinal cord injury, and in some traumatic brain injuries. Patients with a spinal or cerebral lesion suffer from a spastic movement disorder, with a slowing of stepping and of voluntary limb movements. The clinical diagnosis of spasticity is based on the combination of physical signs in the relaxed patient, i.e. exaggerated tendon reflexes, and muscle hypertonia (defined as a velocity-dependent resistance of a muscle to stretch, cf. [1]) In this review the above definition of spasticity will be related to the actual knowledge about the mechanisms underlying the associated movement disorder.

In some studies it is believed [2-6], that descending overactivity causing exaggerated reflexes are responsible for muscle hypertonia, which then leads to spastic movement disorder[2-6]. This view seems to be supported by experiments on the decerebrate cat [7]: the increased muscle tone to stretch becomes considerably reduced after severing the nerves involved in the stretch reflex loop of this muscle. Therefore, the intention of most treatment approaches is to attenuate / abolish reflex activity and thereby to reduce muscle tone (for review [2, 8]). However, this dominant view does not take into account 1. that exaggerated tendon reflexes represent only a small part of the reflex mechanisms involved in the control of functional movement, such as walking; 2. that most studies on the effect of anti-spastic drugs are focused on isolated clinical signs, such as reflex activity and not on the spastic movement disorder that hampers the patient, 3. that without the development of spastic muscle tone, e.g. after stroke, some patients would be unable to walk due to the paresis and, 4. that a rigid muscle tone immediately occurs after decerebration of the cat, while human spasticity develops over weeks after an acute lesion. Up to now, no adequate animal model exists for human spasticity. One reason

for this might be that the pathophysiology of spasticity is multifactorial. Any changes in the neuronal or biomechanical systems, due, for example, to the site and duration of a central lesion, are of importance in determining which neural control mechanisms are deficient and constribute to the movement disorder[9]. Furthermore, one has to be aware that such deviations may 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 their origin, e.g. inflammatory or traumatic. However, these factors have only a limited influence on the impairment of function..

Research on functional movements during recent years has indicated that the clinical signs of spasticity are little related to the spastic movement disorder, which hampers the patient and should be the focus of any treatment. For example, exaggerated reflexes, a dominant sign in the clinical examination, have little impact on the movement disorder.The aim of this review is to establish the actual state about reflex behaviour and muscle mechanics in the spastic patient, as well as the resulting muscle tone during three different conditions: the passive (clinical), the active non-functional (laboratory setting) and the functional (walking).

Clinical signs: passive condition

In the clinical setting, muscle tone and tendon tap reflexes are routinely examined in the relaxed patient. Exaggerated tendon tap reflexes and an increased resistance of a muscle to stretch indicate the presence of spasticity as a sequel of a central motor lesion.

Short-latency stretch reflex

The nature and mechanisms underlying exaggerated tendon reflex activity (mono- / or oligosynaptic segmental reflexes) has been the focus of many studies in spastic subjects. 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 a hyperreflexia due to a neuronal reorganisation in both cat [10] and humans [11]. Novel connections may cause changes in the strength of reflex excitability. In addition, hypersensitivity caused by the denervation may occur [10].

Exaggerated reflexes were thought to be due to a hyperactivity of fusimotoneurons [12, 13] controlling the sensitivity of the muscle spindles. Although, only indirect approaches were applied, this could never convincingly be shown (cf. [14-16]). In addition after a central lesion it is unlikely that recurrent inhibition of motoneurons via Renshaw cell activity is reduced ([17]; but see [18]) or intraspinal nerve sprouting occurs [18] as possible mechanisms of enhanced muscle electromyographic (EMG) activity.

However, in the lower limb there is evidence for a reduced pre-synaptic inhibition of Ia afferent fibres (which mediate short-latency reflexes) in paraplegic but not in hemiplegic subjects [20, 21]. In the upper limb reduced Ia inhibition seems to be present on the hemiplegic side [22]. No correlation exists between decreased presynaptic inhibition of

Ia afferents and the degree of muscle hypertonia as assessed by the clinical Ashworth's scale[21].

In addition, deficient disynaptic reciprocal inhibition [23], increased excitability of reciprocal Ia inhibitory pathways [24-26], changed post-activation depression [27], and a disinhibition of group II pathway [28-30] might lead to hyperreflexia in spasticity of spinal and supraspinal origin. Probably other mechanisms are involved as well. [21].

A severe central motor lesion is followed by a flaccid paresis with a loss of tendon tap reflexes. After 1-2 weeks, tendon reflexes and muscle tone reappear. At later stages (4-6 weeks) clinical signs of spasticity (i.e. exaggerated reflexes and increased muscle tone) become established. During the course of a complete spinal cord injury, the H-reflex (electrically elicited short-latency reflex excluding muscle spindles) is already present during spinal shock when tendon reflexes cannot yet be elicited [31]. The loss of reflexes is attributed to a reduced excitability of alpha- and gamma (innervating muscle spindles) -motoneurons due to the sudden loss of input from supraspinal centres. When spasticity has developed, the threshold of soleus stretch-reflex is decreased in spastic hemiparetic subjects [32, 33], possibly due to an increase in motoneurone excitability [34]. However, repetitive clonic muscle contractions are assumed to be more likely associated with an 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 which is suggested to be connected with spinal locomotor centers [36]. The dominant view is that flexor reflexes are exaggerated after a central nervous lesion and to be responsible for muscle spasms occurring after a severe spinal cord injury (cf. [37]). Also a spontaneous firing of motoneurons during rest

is suggested to lead to the occurrence of muscle spasms [38], initially due to a receptor up-regulation and later due to sprouting neurons [39, 40].

In fact a windup of flexor reflexes occurs in chronic SCI subjects and 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 SCI subjects [44].

After an acute complete SCI, flexor reflex excitability and spastic muscle tone develop in parallel after spinal shock [31]. However, after a few months, there is a divergent course in so far that the severity and occurrence of muscle spasms further increases, while flexor reflex amplitude decreases [31]. In line with this, complete chronic SCI individuals have a low incidence of the early component of flexor reflex [44, 45] and flexion reflexes produce smaller leg joint torques compared to healthy subjects [46]. Recent observations therefore suggest that the activity of flexor reflexes is little related the occurrence of muscle spasms in spasticity of spinal origin.

Muscle tone

Muscle hypertonia is clinically assessed by the Ashworth scale, and is defined as a velocity-dependent resistance to stretch (see Introduction). This is particularly true for the leg extensor [47, 48] and arm flexor muscles [34,49] , i.e. the anti-gravity muscles. In chronic stroke patients, spastic muscle hypertonia (clinically experienced as an increased resistance of a muscle to stretch) is associated with muscle EMG-activity, which largely exceeds that seen in healthy subjects [50, 51]. Thus muscle hypertonia in the clinical testing condition reflects a combination of intrinsic and reflex mediated muscle

stiffness. Also muscles of the non-affected side of stroke patients show some increase in muscle tone compared to healthy controls [52].

However, despite the "extra-activity", the passive stiffness (e.g. muscle contracture) at the ankle joint is also increased and contributes to the clinically defined spastic muscle hypertonia after stroke [53-55]. In studies that have used a more complete analysis looking at all of the contributing factors, it becomes evident that the abnormal stretch reflex activity is insufficient to explain increased muscle tone in subjects suffering from stroke or multiple sclerosis. [51, 56-58]. Reflex mediated stiffness in the ankle plantar flexors [58] and elbow flexor muscles [34, 50, 59] in spastic stroke subjects is within the range of healthy controls and seems to be only slightly increased in SCI subjects [60].

More recent studies indicate an increase of passive stiffness of a muscle to stretch in spastic stroke subjects due to changes in collagen tissue and tendons [51, 54, 58], an enhancement of intrinsic stiffness of muscle fibres [61] and a loss of sarcomers [62], leading to (sub-) clinical contractures. In addition, morphometric and histochemical investigations show alterations of mechanical muscle fibre properties [63-65] that might contribute to spastic muscle tone. Consequently, clinical muscle hypertonia in stroke subjects appears to be rather associated with subclinical muscle contracture than with reflex hyperexcitability [57, 62, 66]. Alterations of biomechanical parameters of a muscle might also have an important effect on the stretch reflex behaviour (possibly via group III/IV muscle afferents) in stroke subjects [67, 68].

In conclusion, exaggerated stretch or flexor reflexes elicited in the passive muscles, as in the clinical bedside examination, are not solely responsible for the increased resistance of a spastic muscle to stretch. Secondary changes of intrinsic and extrinsic muscle properties contribute to spastic muscle tone. This interpretation is based mainly on observations made in stroke patients. Corresponding results are, however, also

reported for central motor lesions of different origin (e.g. traumatic SCI and multiple sclerosis).

Active muscle in non-functional conditions

Active muscle function in normal and impaired motor control is frequently investigated in a laboratory setting, where subjects can exert a controlled level of voluntary contraction. This is believed to allow for a better insight into the neuronal mechanisms underlying muscle tone regulation compared to the passive condition.

Voluntary elbow movements in stroke subjects are rather disturbed by paresis than by antagonistic muscle hypertonia, even in subjects with marked spasticity, i.e. increased muscle tone [50, 69]. When background contractions are matched to normal levels in spastic subjects, little evidence exists for exaggerated reflex activity [58, 61, 70] (for exception see [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 basically different results to those obtained in the passive muscle. Most of these studies are performed during isometric muscle contractions or isotonic movements of upper [50, 59] and lower [60, 75-77] limbs with matched background EMG-activity of corresponding muscles of the spastic and non-affected side of hemiplegic stroke subjects. The studies show a uniform pattern of compensatory EMG-responses to the displacements. In the unaffected muscles, the short-latency reflex is followed by a long-latency reflex EMG-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 (e.g. during locomotion) and group I fibres on a supraspinal (e.g. hand movements) level. Compared to 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 [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 side.

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

In conclusion, in the voluntarily contracted (non-functional) muscle of healthy subjects, the reflex behaviour differs basically from that in the passive (clinical) condition. In contrast, in spastic subjects the excitability state remains roughly unchanged in the passive and voluntarily activated muscles. In a non-functional perturbation task the overall EMG- 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.

Functional movement – walking

After a central motor lesion subjects suffer from a movement disorder. For an adequate treatment, it is therefore of crucial importance to address the mechanisms underlying the impaired function. During the last years a number of studies indicated, that the clinical signs of spasticity can hardly be related to the movement disorder. Some of the mechanisms underlying the impaired movement, such as altered muscle mechanics, will be established and discussed in this section.

Pattern of leg muscle activation

During a functional movement such as locomotion, a typical EMG-pattern of leg muscle activation is recorded in subjects with spastic hemi- or paraparesis. Spastic gait is associated with a reduced leg muscle activity, compared to the unaffected side of hemiparetic patients or to healthy subjects [75, 76, 79]. The reduction depends on the severity of paresis. Furthermore, after stroke, gait recovery during rehabilitation is not associated with changes in the walking pattern [81]. The timing of the pattern, i.e. the reciprocal activation of antagonistic leg muscles remains basically preserved in spasticity of spinal and supraspinal origin [79, 82, 83]. Only rarely does some co-activation of antagonistic leg muscles occur during the stance phase [84, 85, 86]. A premature leg extensor activation during the stance phase of gait, as described elsewhere [85], depends on the plantar-flexed position of the spastic-paretic foot. In this context one should note that premature leg extensor activation in the early stance phase, or even before impact also occurs when healthy subjects walk by voluntarily tip-toeing, i.e. the extensor activation depends on the foot position before impact. Furthermore, a co-activation of antagonistic leg muscles can be recorded in healthy subjects when they are walking with slightly flexed knees (unpublished observations of the author VD).

Occasionally, in the spastic subject, the impact of the fore-foot is associated with the appearance of stretch reflex potentials [84]. The leg extensor EMG amplitude modulation, which normally is seen in healthy subjects 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 depressed in spastic subjects [88].

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

Reflex behaviour

In healthy subjects, group Ia afferent input to the spinal cord becomes suppressed during the stance phase of gait (for reviews see [78, 87]). Due to a reduced Ia suppression in spasticity, short-latency stretch reflexes often appear in the leg extensor muscles during the transition from swing to stance phase of gait, which is rarely the case in healthy subjects or 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 subjects, H- and short-latency stretch reflexes (both mediated by group Ia afferents) in the leg muscles become modulated in a quite specific way (cf. [91, 92]). In subjects 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 motoneurone discharge, which characterizes functional muscle activation, is absent in spasticity [63, 97]. The quadriceps tendon jerk reflex depression, which is present in healthy subjects, is removed in spinal lesion subjects and is

associated with a loss of modulation during the step cycle. These changes are less pronounced in subjects with cerebral lesions [92]. Besides this, no other qualitative difference in reflex behaviour is known between spasticity of cerebral and spinal origin[92], although direct comparisons were only rarely performed.

During perturbations of gait (e.g. 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) EMG reflexes in the leg extensor [80, 87, 98] and dorsiflexor muscles [99]. In contrast, in the spastic leg, short-latency reflexes appear isolated without a significant long-latency EMG component [75, 100]. Following stance displacements associated with a stretch of the leg flexor muscles, the amplitude of the compensatory tibialis anterior EMG response is smaller on the spastic side compared to the unaffected one without a preceding short-latency reflex potential [99, 101]. Thus, a similar reflex behaviour is seen during displacements applied to activated limb muscles during non-functional and functional conditions.

These findings are interpreted as an impaired utilization of afferent input by spinal neuronal circuits after a central lesion. The consequence is a reduced adaptation of muscle activity to the actual ground conditions [88]. Together with the reduced capacity to modulate reflex activity over the normal range, this might contribute to the spastic movement disorder [70, 87].

Tension development

Muscle tone, as defined clinically (see Introduction), cannot be examined during movement. However, tension development at the Achilles tendon, resulting from a combination of muscle stiffness and EMG-activity, can be recorded. A basically different tension development in the affected and unaffected leg occurs in stroke

subjects with spastic hemiparesis [75]. On the unaffected side, changes in tension at the Achilles tendon parallel the amplitude of triceps surae EMG activity. In contrast, 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 EMG amplitude [75]. This is interpreted as a tension development on a simpler level of organisation on the spastic side due to changes in mechanical properties of the leg extensor muscles. The potential mechanisms underlying these changes are outlined above. Thus, secondary to a cerebral or spinal lesion a major alteration of the normal muscle joint anatomical relationship takes place [62, 102, 103]. This allows for support of the body during stepping movements.

In conclusion, 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 hyperreflexia is little involved in spastic movement disorder. Impaired function and attenuation of long-latency (polysynaptic) reflexes hamper walking performance. Secondary to a central lesion, changes in muscle intrinsic, 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 optimal for a given state of the system of movement production [104]. The knowledge about the nature of these alterations in muscle mechanics is still rudimentary.

Cerebral palsy

Children with a perinatal lesion of the central motor system share some characteristics of spasticity with that observed in adults. However, due to the early onset of the damage, impaired motor system development influences the mechanisms contributing to spasticity.

Although neurophysiological studies indicate an inhomogeneous condition of muscle tone in children with CP [105], typical features exist during walking. The leg muscle activity underlying walking of children with congenital cerebral palsy (CP) has characteristic signs of impaired maturation of the normal gait pattern,' i.e. it closely resembles that of stepping in newborn infants [106, 107]. The EMG pattern recorded in young adults with CP consists of a co-activation of antagonistic leg muscles with a reduced and tonic mode of EMG-activity and the appearance of isolated EMG potentials mainly in the leg extensor muscles after ground contact [108, 109]. Also a short-latency reflex irradiation, usually observed in healthy infants under 2 years of age [110-112] is present in children with CP. This suggests that the early infant stepping pattern persists in CP children [113].

Only when the cerebral lesion is acquired at a later stage and the reciprocal mode of leg-muscle activity is already established (i.e. at around 4 years), reciprocal activation of antagonistic leg muscles remains preserved during spastic gait, similar to what is observed in stroke patients [75]. As in adult spastic subjects, there exists no correlation between the clinical signs of exaggerated stretch reflexes and spastic muscle tone [114]. Studies indicate abnormalities of muscle visco-elastic properties with intramuscular contractures at an early stage [105, 108], similar as in adult spastic patients. These alterations are suggested to result in a gait equinus, as they can hardly be explained in terms of a central paralytic foot drop [115].

In conclusion, CP children share some clinical signs and mechanisms underlying movement disorder with spastic adults. Apart from this, an impaired corticospinal input

during development, associated with a deficient modulation of spinal interneuronal circuits, might lead to the abnormal reciprocal inhibition in CP children during walking. Such a mechanism may contribute to the co-activation pattern.

Therapeutical consequences

Any treatment of spasticity should focus on the movement disorder which impairs the patient. On basis of actual studies, as established above, in most cases the physical signs obtained during the clinical examination are an epiphenomenon rather than the cause of the functional condition which impairs the patient. Recent studies have shown that during functional movements essential reflex mechanisms are involved which are not assessed by clinical testing (Figure 3). Nevertheless, site, origin and severity of a central motor lesion have an influence on the clinical appearance of spasticity and have to be taken into account for the appropriate treatment of an individual subject.

The dominant view of treatment of spasticity is directed towards a reduction of stretch reflex activity. As established in this review, this treatment approach is primarily based on studies on muscle tone and reflex activity under passive conditions (although the treatment with Botulinum toxin is frequently programmed on the basis of EMG-activity during active movements).

Actual investigations on functional leg and arm movements show no causal relationship between exaggerated reflexes and movement disorder following a spinal or supraspinal lesion. Impaired walking is thought to be mainly due to a disabling paresis and an impaired utilization of afferent input by spinal neuronal circuits. Consequently, anti-spastic 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]. It

even may increase weakness, e.g. [118, 121, 122], which might interfere with the ability to perform functional movements such as walking. In contrast, cannabinoids improve mobility in patients with multiple sclerosis but have no effect on spastic muscle tone [123]. Consequently, it is also of no surprise that in children with spastic diplegia, selective dorsal rhizotomy (SDR; reduces afferent input to the spinal cord) combined with physiotherapy results in a similar improvement in mobility as is observed in children without SDR [124, 125]. However, some alterations in gait mechanics were reported after SDR [126]. Similarly, the application of Botulinum toxin is assumed to result in a rather cosmetic effect on spastic signs often without functional improvement (cf. [121, 127]). An influence on intrafusal fibre function of this toxin was discussed [128, 129]. Nevertheless, there are also reports indicating, that intrathecal baclofen can 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, re-learning and activation of residual motor function[133,134], and the prevention of secondary complications such as muscle contractures. With regard to cerebral palsy, there have been a few controlled studies documenting the positive effect of a functional training programme to date [125, 136].

Anti-spastic drug therapy is thought to be predominantly of benefit for immobilised patients by reducing muscle tone and relieving muscle spasms [137], which may in turn improve nursing care for these patients.

Conclusions

This review describes the differential roles of background and reflex activity as well as muscle fibre function in passive, active and functional movement conditions after a

central motor lesion. According to the actual research, exaggerated reflexes play a minor role and secondary alterations of mechanical muscle fibre properties a major role in their contribution to spastic movement disorder as it might be suggested on the basis of the clinical examination. In functional movements, such as walking, changes in muscle fibre properties leading to spastic muscle tone are required to compensate for the loss of neuronal drive. Further studies are required (i) to understand the regulation and importance of spinal and descending control mechanisms during movement in healthy and spastic subjects and, (ii) to detail the intra- and extra-cellular 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.

Search strategy and selection criteria

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

Contributors

A first draft was prepared by VD. This draft was modified and supplemented by TS.

Conflicts of interest

We have no conflicts of interest.

Acknowledgements

This work was supported by the Swiss National Foundation (NCCR Neuro and Grant No. 3200BO-105324) and The Danish National Research Foundation. We thank R. Jurd for editorial assistance.

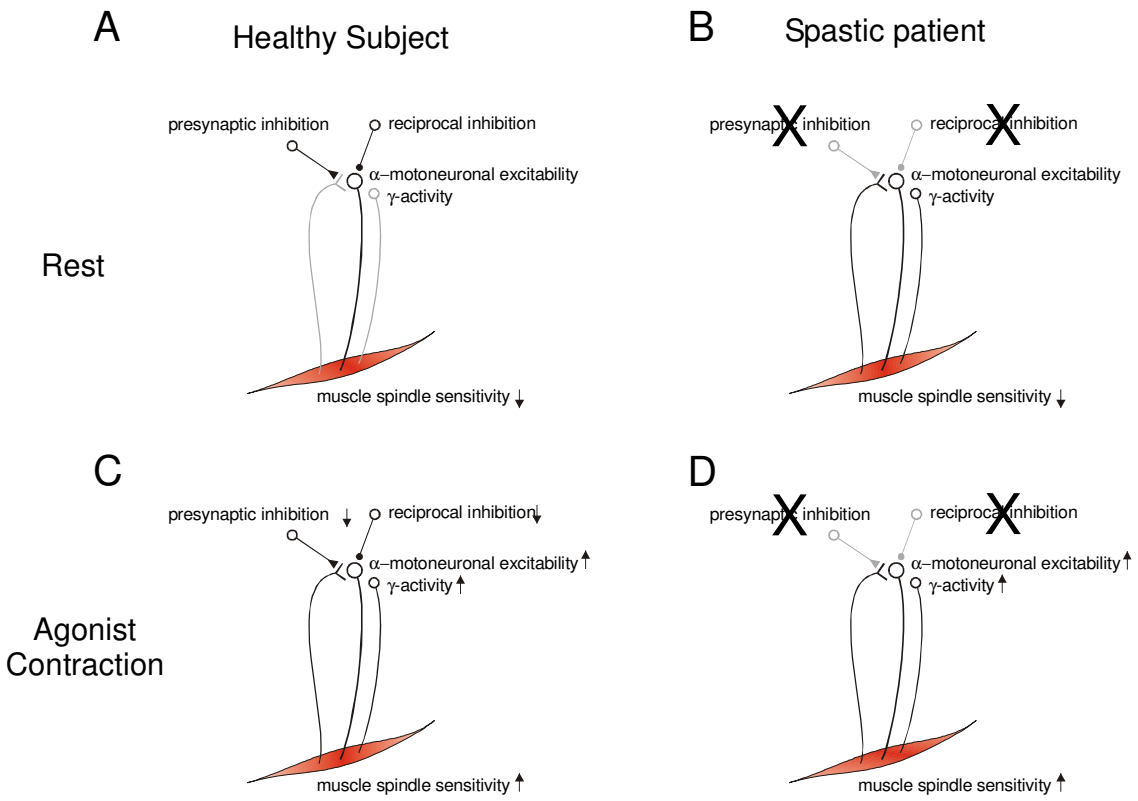

Fig. 1. *Short-latency reflex behavior in the passive and active muscle of healthy and spastic subjects*

In healthy subjects, the stretch reflex activity is low at rest (shown in A). This is explained by low excitability of spinal motoneurons, 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 (shown in C) motoneuron excitability, spindle sensitivity and Ia afferent discharge increases, whereas presynaptic inhibition, Ib inhibition and Ia inhibition decreases. Stretch reflex activity is consequently high. In spastic subjects, presynaptic Ib and Ia inhibitions are already decreased at rest (shown in B) and stretch reflex activity is consequently high already at rest. During voluntary contraction (shown in D) there is only limited change in these parameters and the stretch reflex activity is consequently not much different from rest. The arrows designate, whether the mechanism is decreased or increased during contraction compared to rest. The crosses designate that the mechanism is affected in spasticity (modified from [9]).

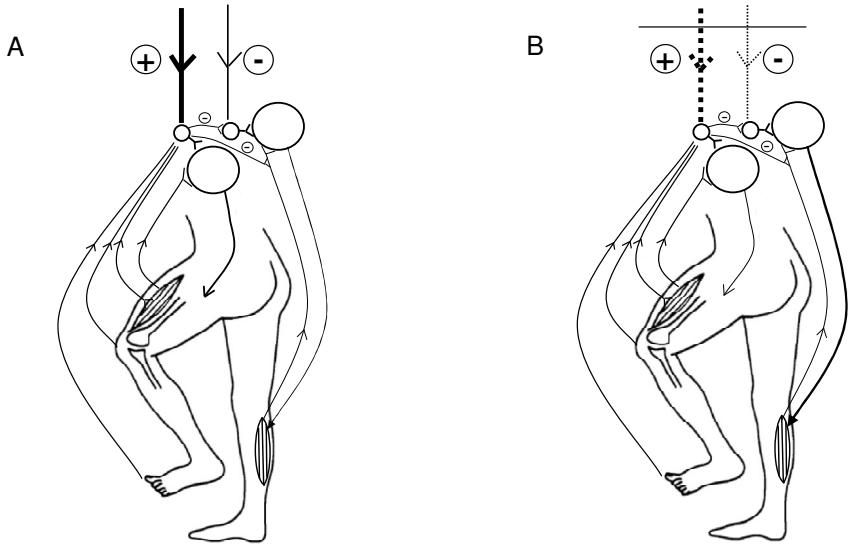

Fig. 2 *Schematic drawing of reflex behaviour during human gait*

A Physiological condition. Long-latency reflex activity becomes facilitated by supraspinal drive. It becomes significantly involved in leg muscle activation to adapt the locomotor pattern on the actual ground conditions. Ia afferent mediated inputs are inhibited.

B Proposed situation after spinal/supraspinal lesion. The functionally essential activity of long-latency reflexes is impaired due to the loss of supraspinal input (modified from [87]).

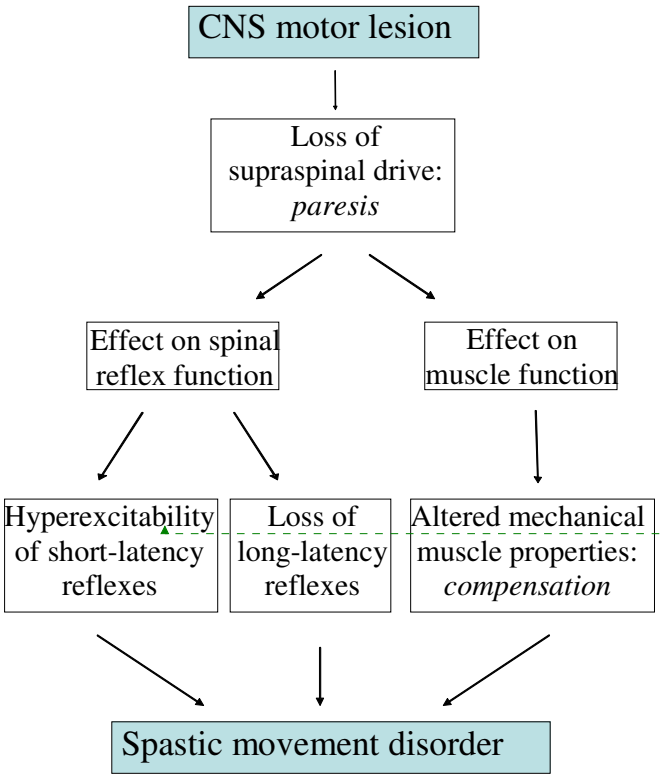

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Fig. 3 *Schematic drawing of the mechanisms involved in spastic movement disorder*

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 sequel of the primary lesion leads to the spastic movement disorder (modified from Dietz [87]).

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