Scientific Review

Spastic movement disorder

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This review deals with the neuronal mechanisms underlying spastic movement disorder, assessed by electrophysiological means with the aim of first, a better understanding of the underlying pathophysiology and second, the selection of an adequate treatment. For the patient usually one of the first symptoms of a lesion within the central motor system represents the movement disorder, which is most characteristic during locomotion in patients with spasticity. The clinical examination reveals exaggerated tendon tap reflexes and increased muscle tone typical of the spastic movement disorder. However, today we know that there exists only a weak relationship between the physical signs obtained during the clinical examination in a passive motor condition and the impaired neuronal mechanisms being in operation during an active movement. By the recording and analysis of electrophysiological and biomechanical parameters during a functional movement such as locomotion, the significance of, for example, impaired reflex behaviour or pathophysiology of muscle tone and its contribution to the movement disorder can reliably be assessed. Consequently, an adequate treatment should not be restricted to the cosmetic therapy and correction of an isolated clinical parameter but should be based on the pathophysiology and significance of the mechanisms underlying the disorder of functional movement which impairs the patient.

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Introduction

Movement disorders are prominent features of impaired function of the motor systems and are frequently best reflected during gait. For the patient, the movement disorder usually represents the first symptom for a lesion within the central motor system. The neurological examination is characterised by changes in reflex excitability and muscle tone and leads to an appropriate diagnosis underlying the gait disorder. The physical signs obtained during the clinical examination can, however, give little information about the pathophysiology underlying the movement disorder: stretch reflex excitability and muscle tone are basically different in the passive (clinical examination) compared to an active motor condition (movement). In addition, during a movement such as gait, several reflex systems are involved in its execution and control. Therefore, for adequate treatment of a movement disorder, we have to know about the function of reflexes and supraspinal motor centres involved in the respective motor task. A movement such as locomotion is determined by the

strength of electromyographic (EMG) activation of antagonistic leg muscles as well as intrinsic and passive muscle properties. The EMG activity recorded from the leg muscles reflects the action and interaction between central programs and afferent inputs from various sources which can only be separated to a limited degree. Therefore, for an assessment of the neuronal control of locomotor movements we have to record the EMG activity from several antagonistic leg muscles and the resulting biomechanical parameters such as joint movements and, eventually, of muscle tension. By such an approach it is possible to evaluate the behaviour of neuronal and biomechanical parameters during the gait disorder. Any changes in the neuronal or biomechanical systems may lead to the pathophysiological basis of movement disorder. One has, however, to be aware that such deviations may already be secondary and compensatory to the primary dysfunction of the motor systems involved in the respective movement. This review is focused on the spastic movement disorder, the development of spasticity after an acute central motor lesion, the clinical signs and their relationship to the spastic movement disorder.

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From spinal shock to spasticity

More than 100 years ago the term 'spinal shock' was introduced to describe the clinical state in patients with acute spinal cord injury (SCI) presenting muscle paralysis with flaccid muscle tone and loss of tendon reflexes below the level of lesion.¹ Although complete loss of sensorimotor function persists, spinal shock ends after some weeks and during the following months a 'spastic syndrome' develops with exaggerated tendon reflexes, increased muscle tone and involuntary muscle spasms. The development of a spastic syndrome is a common finding in surgically or pathologically confirmed complete spinal cord transection,² ie, changes in the excitability of spinal cord neuronal circuits occur independently of supraspinal influences. In a recent study³ clinical and electrophysiological parameters were systematically assessed in patients after an acute SCI.

The period of spinal shock is electrophysiologically associated with a reduced excitability of alpha-motoneurons (MN) reflected by a low persistence of F-waves (the F-wave is due to the antidromic excitation of MN following supramaximal electrical stimulation of a peripheral nerve), which is in line with previous studies.^{4,5} This observation may be related to the clinical signs found at this stage: the loss of tendon reflexes and muscle hypotonia. The neurophysiological basis of the reduced alpha-MN excitability may be due to the sudden loss of tonic input and/or trophic support from supraspinal to spinal neuronal centres.

In contrast to the neuronal depression reported above, H-reflexes can be elicited already at an early stage after SCI,³ which is in accordance with earlier reports.⁶⁻⁸ The discrepancy between preserved H-reflex and the loss of tendon reflexes as a typical clinical sign might at least partially be due to a reduced activity of gamma-motoneurons (gamma-MN).

While H-reflex was present early and F-wave persistence was low (but usually F-waves could be recorded), there is a loss of flexor reflex activity in the tibialis anterior (TA) early after SCI.³ The latter observation indicates a suppression of interneuronal (IN) activity mediating this polysynaptic reflex (for review see⁹).

The recovery from spinal shock is clinically reflected by an increase in the excitability of tendon tap reflexes and in muscle tone (assessed by the Ashworth scale) as well as more frequently occurring muscle spasms (assessed by the Penn spasm frequency scale). These clinical changes are associated in the electrophysiological recordings by an increase of F-wave persistence and flexor reflex activity.³ There is only a minor change in H-reflex excitability during this time period. It may be suggested that the increase in tendon tap reflex excitability is at least partially due to a recovery of alpha-MN and probably also of gamma-MN function mediating this reflex. There is also an increase in flexor reflex activity which should not

only be due to a recovery of alpha-MN excitability, but also to the resolving depression of spinal interneuronal function. The pathway underlying the flexor reflex is a polysynaptic spinal one and allows the integration of inputs from muscles, joints and cutaneous afferents on common interneurons.⁹

The changes in flexor reflex excitability may at least partly be reflected in the appearance of muscle spasms. The increase in muscle tone may be due to a more general recovery of spinal neuronal (alpha-MN and IN) activity. Nevertheless, for both clinical parameters reflecting recovery from spinal shock (Penn spasm frequency and Ashworth scale) it cannot be ruled out that other factors than the neuronal activity recorded here, as well as changes of muscle biomechanics, contribute to the development of spasticity.

Clinically, the development of the spastic syndrome is characterised by exaggerated muscle tendon tap reflexes, increased muscle tone and involuntary muscle contractions. The onset of spastic signs is difficult to determine because there is a smooth transition to a clearly established spasticity.

During this stage, both M-wave and flexor reflex amplitudes remain about stable in tetraplegic or decrease in amplitude in paraplegic patients, ie, develop even opposite to the clinical signs.³ A slight increase of H/M-ratio, which is in line with an earlier study,¹⁰ might contribute to exaggerated tendon tap reflexes. However, a correlation of this increase with the spastic state must be considered cautiously: (1) The high H/M-ratio can express rather a decrease of M-wave than an increase in reflex excitability; and (2) Short latency reflex hyperexcitability was shown to be little related to spastic muscle tone.¹¹

The fact that the decrease of M-wave and flexor reflex amplitude is more pronounced in paraplegic than in tetraplegic patients indicates that several weeks after a SCI secondary, degenerations of spinal tracts occur including pre-motoneuronal circuits and alpha-MN. A direct damage of alpha-MN as an underlying cause is rather unlikely because M-wave amplitude increased up to the fourth week after SCI. Therefore, secondary degenerations are likely to depend on the level of lesion, ie, are less pronounced with higher level of lesion. The phenomenon of trans-synaptic degeneration of alpha-MN due to a degeneration of terminal synapses on a cell after SCI was already claimed earlier.¹²

On the basis of the observations made in the study of Hiersemenzel *et al*³ clinical signs of increasing spasticity, such as muscle tone and spasms, can hardly be related to the electrophysiological recordings. Secondary changes of motor units might contribute to the syndrome of spasticity, especially in respect to muscle tone and spasms.

Several studies claim that 'peripheral changes' (eg, chronic transformation of muscle in spasticity) contribute to the increased muscle tone.^{13,14} A decrease of M-wave in patients with a SCI¹⁵ or those with a cerebral lesion¹⁶ indicates an affection of spinal

MN in the case of a disconnection from supraspinal input. Furthermore, signs of denervation are described to occur during the first 2 months after SCI.¹⁷ It became obvious during recent years that following a central motor lesion changes in mechanical muscle fibre properties occur early after a lesion with the consequences of a significant contribution to muscle tone in the active (for review see¹⁸) and passive muscles¹⁹ (for review see¹¹ and next sections).

Clinical spasticity

Spasticity produces numerous physical signs such as exaggerated reflexes, clonus and muscle hypertonia. Clinically, spastic hypertonia has been defined as a resistance of passive muscle to stretch in a velocity-dependent manner following activation of tonic stretch reflexes.²⁰ On the basis of clinical observations a widely accepted conclusion was drawn for the pathophysiology of spasticity such that exaggerated reflexes are responsible for the observed muscle hypertonia, and therefore the movement disorder.

Hyperflexia is due to the neuronal reorganisation which occurs following central lesions in both cat²¹ and man.²² Novel connections may cause changes in the strength of reflex excitability. In addition, supersensitivity caused by the denervation may occur.²¹ Although recent observations have indicated that central nervous lesions do not cause sprouting of primary afferents in either cat²³ or man,²⁴ changes in the reduction of pre-synaptic inhibition of group Ia fibres occur²⁵ which correlate with the enhanced excitability of tendon tap reflexes. In addition, reduction of pre-synaptic inhibition of group Ia afferents is stronger in patients with paraplegia compared to those with hemiplegia.¹⁰ However, no correlation is seen between decreased presynaptic inhibition of Ia terminals and the degree of spastic muscle tone measured by Ashworth's scale.¹⁰

Studies on muscle tone and reflex activity in patients with spastic hemiparesis following stroke have usually been performed under passive motor conditions. In such a condition a displacement of the elbow joint is associated with an increased EMG activity in the stretched muscles of the spastic side compared to the unaffected side in patients with spastic hemiparesis.^{19,26} Nevertheless, subsequent studies analyzing the different components which contribute to muscle tone clearly revealed that muscle hypertonia is more associated with muscle fibre contracture than with reflex hyperexcitability.^{11,27}

Studies on muscle tone and reflex activity have usually been performed under passive motor conditions.^{26,28} In such a condition, increased elbow torque following a displacement was associated with an increased EMG activity in the flexor muscles of the spastic side compared to the unaffected side in patients with spastic hemiparesis.¹⁹ Nevertheless, spastic muscle hypertonia was found to be closely associated with muscle fibre contracture but not with reflex hyperexcitability.²⁷

Spastic gait disorder

Extensive investigations on functional movements of leg^{13,29,30} and arm^{19,31,32} muscles have not revealed any causal relationship between exaggerated reflexes and spastic movement disorder. In patients with cerebral or spinal lesions the reciprocal mode of leg muscle activation during gait is preserved in spasticity. Exaggerated stretch reflexes in spasticity are associated with an absence or reduction of functionally essential polysynaptic (long-latency) reflexes. Both cutaneous³³ and stretch³⁴ reflex modulation are impaired in patients with spinal cord lesion during walking. In the latter experiments ankle joint rotations were induced in any phase of the step cycle. It was proposed that impaired modulation of the stretch reflex along with increased ankle joint stiffness contribute to the impaired walking ability in spastic patients.

In spastic patients the EMG activity in the calf muscles during gait is smaller in amplitude and less well modulated compared to healthy subjects.¹³ This observation is most probably due to the impaired function of polysynaptic reflexes. Fast regulation of motoneuron discharge, which characterises the normal muscle, is absent in spasticity.^{35,36} This corresponds to a loss of EMG modulation during gait. In patients with spastic hemiparesis due to cerebral lesion, the strength of EMG activity in the affected leg is reduced compared to the unaffected leg. This corresponds to the degree of paresis observed during both gait²⁹ and elbow movements.³¹ Thus it becomes clear that inhibition and facilitation of spinal reflexes depends upon supraspinal control which is impaired in spasticity.

In spastic paresis (acquired at an early or later stage), a fundamentally different development of tension of the triceps surae takes place during the stance phase of the stride cycle.^{29,30} In the unaffected leg, the tension development correlates with the modulation of EMG activity (the same is true in healthy subjects) while in the spastic leg tension development is connected to the stretching period of the tonically activated (with small EMG amplitude) muscle. During gait there is no visible influence of monosynaptic reflex potentials on the tension developed by the triceps surae. A similar discrepancy between the resistance to stretch and the level of EMG activity has been described for flexor muscles of the upper limb in spastic patients.^{37,38} Muscle hypertonia in spastic patients is primarily associated with muscle contracture but not with exaggerated reflexes.^{11,27}

Muscle tone during functional movements in patients with spastic paresis cannot be explained by an increased activity of motoneurons assessed by EMG recordings. Instead, a transformation of motor units takes place such that a higher tension to EMG activity relationship occurs during the stretching phase of the triceps surae. This has the consequence that regulation of muscle tension takes place at a

lower level of neuronal organisation. Therefore, the changed regulation of spastic gait should be considered as optimal for the given state of the motor system (*cf.*³⁹).

Motor unit transformation and spastic muscle tone

There are several findings which support the suggestion that changes in mechanical muscle fibre properties occur in spasticity. Contraction times in hand muscles⁴⁰ as well as in the gastrocnemius⁴¹ in patients with spastic hemiparesis are prolonged. Torque motor experiments applied to upper and lower limb muscles indicate a major, non-reflex contribution to the spastic muscle tone in the antigravity muscles, ie, in the leg extensors and elbow flexors.^{42,43} Histochemistry and morphometry studies of spastic muscle have revealed neurogenic changes of the muscle fibres.^{14,36} These include: (a) increased levels of muscle fibre atrophy (especially of type II fibres); (b) a predominance of type I fibres in the gastrocnemius during later stages, when spasticity of cerebral origin has been established; (c) structural changes, such as the appearance of target fibres, mainly in type I fibres and (d) a significant part of changes of mechanical muscle fibre properties may be attributed to a shortening of muscle length as a result of a decrease in the number of sarcomeres in series along the myofibrils accompanied by an increase in resistance to stretch.²⁷ Such muscle contracture can be produced in experimental animal by plaster cast immobilisation of muscles in shortened positions.¹¹

The alteration to a simpler regulation of muscle tension following paresis due to spinal or supraspinal lesions is basically advantageous for the patient as it enables him to support the body during gait and, consequently, to achieve mobility.²⁹ However, rapid movements are no longer possible due to the absence of modulation of muscle activity. Following a severe spinal or supraspinal lesion, these transformative processes can overshoot with unwelcome consequences, ie, painful spasms and involuntary movements.

Therapeutic consequences

The treatment of spasticity is usually directed towards reducing stretch reflex activity as it was thought that exaggerated reflexes were responsible for increased muscle tone and therefore for spastic movement disorder.

However, therapeutic outcomes in spasticity should be considered in terms of physiotherapeutic approaches. These should be directed to train and activate residual motor functions (eg,⁴⁴) and to prevent secondary complications such as muscle contractures and spasms. More severely affected patients are usually less able to perform stepping movements as the spinal locomotor centres do not provide sufficient basic activation of leg muscles. In recent years it has been shown that specific locomotor

training could enhance locomotor activity and thereby improve mobility.^{44,45}

Antispastic drug therapy is the second tool. It reduces muscle tone and spasms by the induction of a paresis which may interfere with the performance of functional movements.⁴⁶ Antispastic drug therapy is, therefore, predominantly of benefit for immobilised patients in whom it reduces muscle tone and relieves muscle spasms, both of which may also improve nursing care for these patients.

Conclusions

In patients with spastic paresis, the central programming, with the timing and reciprocal mode of leg muscle activation during gait, is basically intact. Exaggerated monosynaptic reflexes are associated with a loss of the functionally essential polysynaptic reflex mechanisms, both being dependent on supraspinal control. When this control is impaired, inhibition of monosynaptic stretch reflexes is missing in combination with a reduced facilitation of polysynaptic reflexes.

A spinal or cerebral lesion, associated with paresis, is followed by a transformation of motor units and shortening of muscle fibres, leading to a (subclinical) muscle contracture, such that tension development in the antigravity muscle occurs in a simpler fashion. Development of tension by the calf muscles during gait is normally determined by modulated gastrocnemius/soleus electromyographic activity. In the spastic leg, calf muscle tension is associated with the stretching of the tonically activated muscle. This regulation of muscle tension at a lower level is efficient in so far as it enables the patient to support body weight during gait.

Consequently, in mobile patients primarily physiotherapeutic approaches should be applied, while antispastic during therapy represents a second tool. Only in immobilised patients could antispastic drugs be of benefit to relieve muscle spasms and improvement of nursing care.

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