Neurophysiological and kinesiological aspects of spastic gait: the need for a functional approach

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Summary

Many instruments have been employed in recent years in order to quantify the posture and motion of the head in normal and pathological subjects. Evaluations of this type present many difficulties related to the influence of individual and external factors and to the accuracy of the system used. In patients with cervical dystonia (CD) the only rating scales currently used are semi-quantitative and subjective. More precise information on disease severity and response to the treatment is needed.

Posture and motion of the head were evaluated by means of ELITE motion analyser (BTS, Milan, Italy) in 6 patients with the left laterocollis form of CD undergoing treatment with botulinum toxin (BTX). The method emerged as very useful for the quantification of the therapeutic response (which was more

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Introduction

The last two decades of the twentieth century brought significant advances in our understanding of the mechanisms of the central nervous system control of human locomotion. More widespread use of advanced gait analysis techniques (1,2) has allowed interesting and often surprising insights into gait neurophysiology and kinesiology. These revolutionary developments have given rise to new hopes and new approaches for the restoration of walking in human neurological disorders. Nevertheless, the application of this knowledge in clinical practice, especially in spastic patients, is more complex than might be expected. In particular, the neurophysiology, kinesiology and therapy of spasticity, and their implications on human locomotion, represent a multifaceted and rather controversial topic. Recent research has also shown that basic neurophysiological and kinesiological terms, such as muscle hypertonus and hypotonus, muscle weakness and hyperactivity, reflex and non reflex, and even agonist and antagonist, need to be thoroughly revised.

What is spasticity?

It is much easier to recognise spasticity than to quantify, characterise and define it (3). Spasticity is classically defined as a "motor disorder characterised by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome" (spasticity in the narrow sense) (4). The phenomenon of spasticity cannot be distinguished clearly, especially from the clinical point of view, from other consequences of central nervous system lesions (3,5-7), consequences such as:

- velocity independent neurogenic increase of muscle tone (increased plasticity);
- altered viscoelastic properties of the affected muscles and connective tissue;
- contractures, trophic changes, ectopic calcifications;
- weakness, incoordination, clumsiness (due to paresis);
- associated movements, mass reflexes, abnormal reflex reactions and tonic postural response;
- loss of precise autonomic control, autonomic dysreflexia.

In most cases, the decline of motor performance in spasticity is exacerbated by further factors resulting from central nervous system lesions – hemineglect, anosognosia, apraxia, aphasia, sensory impairment and altered sensory, especially proprioceptive and visuomotor, processing (8).

Remarks on the neurophysiology of spasticity

Lesions of the pyramidal tract – or of the upper motor neuron – constitute, according to traditional concepts, the main pathogenetic factor in spasticity. However, further research has revealed that other structures may be even more involved. These include, in particular, extrapyramidal (or non pyramidal) projections, such as the rubrospinal, tectospinal, reticulospinal and vestibulospinal systems. These structures are linked not only functionally but also anatomically with the corticospinal tract. Selective destruction of the corticospinal tract is thus rare. When it does occur, it results not in spastic hypertonia, but rather in hypotonia (5,9,10). In spinal cord injuries, direct lesions of the spinal interneuronal
pool may substantially contribute to the development of spasticity (3,5,10,11). The above-mentioned structural lesions lead to the development a number of more or less well understood secondary neurophysiological consequences occurring particularly (but not exclusively) at spinal and reticulospinal level (5,10,12). A principal current view is that spasticity is caused by long-term reductions in inhibition rather than by a permanent increase in excitation per se. There is also involvement of altered segmental afferents and activated regional excitatory interneurons. Synaptic input is reduced (due to recurrent Renshaw cell inhibition, presynaptic inhibition of la fibres, reciprocal Ia inhibitory interneurons, non reciprocal inhibition by Ib afferent fibres). Furthermore, gamma efferents are described as hyperactive, and denervation hypersensitivity and sprouting can play a role. A decrease in presynaptic inhibition has been described by some authors. Motor neurons may have, for a number of reasons, increased intrinsic excitability. Polysynaptic connections mediating the flexor reflex afferents and nocioceptive reflexes are disturbed and upregulated (3,5,10,13).

The background to the general velocity-dependent increase in tonic stretch reflexes may be different to that previously hypothesised. Under experimental conditions, healthy and spastic muscles can be partially “pre-loaded” with comparable background forces. This makes it possible to dissociate the influence of stretch reflex gain from that of the stretch reflex threshold. Surprisingly, the reflex stiffness and velocity sensitivity of reflex torque are normal under these experimental conditions. The afferent inflow induced by stretch is comparable in normal and spastic muscles. There is also, in contrast to classical concepts, no preference for dynamic or static reactivity (3). Similar results, indicating a reduced contribution of stretch reflexes to increased tone in spasticity, have been reported by Sinkerjaer et al. (14) in multiple sclerosis patients and by Ada et al. (15) in simulated walking in ambulatory stroke patients and controls.

In other experiments, in which the stretch velocity remains constant, a length-dependence of the stretch reflex response has been observed. Burke et al. (16) demonstrated in the quadriceps muscle of spastic subjects that the magnitude of the stretch reflex, measured by both linear and sinusoidal stretching, is inversely proportional to the initial length of the muscle if the stretch velocity remains constant. This length-dependence inhibition of the stretch reflex in spasticity is probably mediated by secondary spindle endings. This phenomenon probably underlies the clasp-knife phenomenon (16).

Disordered motor control in spasticity

The main and crucial problem in spasticity is, however, the abnormal execution of a more or less impaired motor programme, which results in a loss of orderly recruitment and rate modulation of motor neurons (17) and in altered modulation and timing of muscle activation and relaxation during voluntary movement. The phase-dependent reflex modulation of the gait cycle is severely impaired (18). The pattern of muscle activation and the development of increased muscle tone in patients with spasticity may, in the active movement, be dramatically different from that observed in clinical testing of the passive muscles (1,6,19). Moreover, the same muscle groups may be both hypertonic and hypotonic depending on the movement pattern. In this regard, the term spastic dystonia, instead of spastic hypertonia, would seem to be more appropriate (5). Poly-EMG evaluation of spastic patients reveals a lack of the fast and precise modulation of motor neuron discharge typical in normal movement. In particular, rapid and concentric contractions are affected. In locomotion in spastic patients, a stereotyped coactivation, resulting in contraction instead of reciprocal activity, develops (3). Abnormal coactivation includes both the co-contraction of agonists and antagonists during phasic movements and the coactivation of limb proximal and distal muscles (20). The ability to perform isolated movements and control the correct timing of muscle activation is impaired (21,22).

Maximum EMG activity of normal movement (e.g., in elbow flexion) is located at the angle of maximal mechanical advantage. In spasticity there is a substantial shift in the angle of peak EMG, and the muscle is maximally activated away from the optimal angle (3). There is also poor correlation between the muscle activation reflected by EMG activity and the development of tension in the spastic muscle. This tension increases in the absence of sufficient muscle activation and a state of disconnection and dyscoordination between muscle activation, tension development and motor performance ensues (1,6,23). EMG activity per unit force is thus augmented (24). In other words, comparable motor performance requires much greater muscle activation and is accompanied by an inappropriate increase in muscle tone. And the increased muscle tone further worsens the motor performance.

“Stiff-leg” gait

The most notable gait feature in the majority of patients with spastic paresis is “stiff-leg” walking. This term denotes a lack of, or reduction in, normal knee flexion during the swing phase of walking (25-28). This reduction of normal knee flexion is linked to toe drag and creates a large moment of inertia during swing. This situation, in turn, increases the energy required to initiate the swing phase. This is compensated by circumduction, vaulting, upward pelvic tilt or pelvic lag (27-29). Abnormal EMG activity during pre-swing, initial swing or during the whole gait cycle in one or more heads of the quadriceps muscle can indeed be observed. However, gait analysis data show that the traditional view of the spastic quadriceps as the main culprit in spastic stiff-leg gait must be thoroughly revised. More important factors contributing to the stiff-leg gait pattern seem to be poor ankle motion and a reduced ankle moment corresponding to dynamically weak ankle plantarflexors and limited hip flexion and hip power generation during the pre-swing and early swing phases (27-29).

In healthy controls, during the end of stance (pre-swing phase), the knee swiftly flexes to an angle of about 40° as the ground reaction force progresses rapidly posterior or to the knee. Probably, during this phase, ankle me-
Mechanics play a role in allowing the knee to flex. Furthermore, during the initial swing phase, the knee continues to flex to its peak of approximately 65°. This is combined with hip flexion and the momentum generated in pre-swing (30). Hence, the insufficient knee flexion in pre-swing and initial swing may be due to dynamically insufficient hip flexors and impaired active ankle mechanics. The generation of hip power during concentric contraction in the pre-swing phase is markedly reduced in spastic patients (27-29).

Interestingly, the swing phase as a critical gait event requiring fine control is much more dependent on cortical control and an intact corticospinal pathway than the stance phase (31,32). Corticospinal lesions are thought to impair the swing phase and the dynamics of the tibialis anterior is especially affected. The role of corticospinal control increases and becomes essential and decisive for all muscle groups involved in corrective actions and rapid modifications of the limb trajectory and centre of mass transfers, such as when an obstacle suddenly appears or the gait cycle is voluntarily modified (32).

Common complications of lower limb spasticity include shortening of the iliopsoas muscle and development of hip flexion contractures. The latter are believed to result in anterior pelvic tilting, increased knee flexion and decreased contralateral step length. When investigating the gait of patients with hip flexion contractures, the anterior pelvic tilt was found to correlate mainly with reduced hip extension during gait and less with the severity of hip flexion contractures found on static testing (33). A lack of hip extension during the terminal stance may result in a shorter stride length and decreased gait velocity in hemiparetic subjects (34). Interestingly, sufficient hip extension is an important factor contributing to activation of antigravity muscles at lower limb level, at least in animal experiments (35,36).

Spastic and medial hamstrings or adductors are often considered to be among the main factors contributing to excessive internal hip rotation during gait in cerebral palsy and surgical lengthening of these muscles is expected to improve the alignment of the limb during the gait cycle. This concept is based on clinical observations supported by EMG studies (25,37-40).

Arnold et al. (41) have developed a computer graphic model based on MRI images in correlation with joint kinematics. Their research in three individuals with crouched, internally rotated gait showed that the medial hamstrings, adductor brevis and gracilis had negligible internal rotation moment arms or even had external rotation moment arms throughout the gait cycle. Changes in the rotational moment arms of the hamstrings and adductors after derotational osteotomy are minimal (42). The authors concluded that other factors are more likely to cause internally rotated gait in cerebral palsy patients. Unexpectedly, the gluteus medius, especially its anterior compartment, and the gluteus minimus might be contributors to excessive internal rotation in cerebral palsy. Internal rotation moment arms of the gluteus medius and minimus increase dramatically with flexion of the hip (43) and excessive hip flexion usually accompanies internal rotation gait in cerebral palsy (41). On the other hand, dynamic weakness of the gluteus medius during the stance phase may be related to hip deformities, especially hip subluxation in patients with cerebral palsy (44).

Another mechanism contributing to stiff-leg gait may be the abnormal activity of the long hamstrings muscles (long head of the biceps femoris muscle and the semimembranosus muscle). In quadrupeds the hamstrings are mostly knee flexors, in humans their primary function is hip extension. As hip extensors they act especially when they are active in the pre-swing and early-swing phases. The hamstrings inhibit hip flexion and paradoxically reduce knee flexion (27,28). At the end of the swing, they are rapidly stretched and there is a large burst of EMG activity in this phase. Coordination of the biceps femoris, semitendinosus, vastus lateralis and medialis muscles is important for dynamic stability of the knee joint, especially in stance and postural adjustment of the lower limb (35). Preserved function of the hamstrings is naturally important for knee flexion. Careful analysis should be thus performed before blocking, surgically lengthening (or in some cases strengthening) etc. these muscles.

Foot and ankle dynamics in spasticity

Ankle dynamics can probably be considered the most critical component of the gait pattern and the most sensitive to disordered motor control. Benedetti et al. (45) performed gait analysis in minimally impaired patients with multiple sclerosis. The most consistent findings were, apart from limited ankle dynamics, atypical activation of the tibialis anterior during the first part of the stance phase and earlier activation of the gastrocnemius medialis during the stance phase. Furthermore, the tibialis anterior was atypically active during the late double support phase. These findings may reflect both impaired motor control and compensatory mechanisms for improving stability during the gait cycle.

In patients with hemiparesis, the maximum ankle power during walking correlated with self-selected speed (46). An apparently analogous finding, using multichannel EMG, was described by Dimitrijevic (47), who found a correlation between good phase-dependent modulation of the tibialis anterior and triceps surae and walking speed. According to the traditional view, an overactive triceps surae group impedes dorsiflexion and causes toe-walking (48). It is believed that the triceps surae (and spastic muscles in general) should be suppressed to improve the reciprocal pattern.

In healthy adults, the triceps surae is much stronger than the tibialis anterior muscle (49) and the plantarflexors contribute nearly two thirds of the forward power needed for walking (48). The ankle plantarflexor work is primarily used to accelerate the leg into the swing, most of the energy is recovered by transfer into the trunk at the end of the swing. Ankle plantarflexors generate significant positive work only during “push-off”, the phase of concentric contraction beginning during the end of single limb support and extending throughout the double limb support phase. This important aspect of plantigrade gait matures definitively between the ages of seven and ten years (50). Apart from the effective push-off mechanisms, the calf muscle group is important for posture, foot alignment, balance and gait. Spastic children with cerebral palsy are able to generate only a third of the power required for normal gait (51). Weak-
ness of the plantarflexors should be considered as one of the factors limiting gait speed in hemiparetic subjects. Hip flexors can, in some patients, compensate for this weakness in the push-off phase (52). Surprisingly, strengthening and electrostimulation programmes for spastic plantarflexors do not increase spasticity and this therapy in fact improves the gait pattern, promoting a plantigrade pattern (21,33,53). Ada et al. (54) have also confirmed, both in a control group and in stroke patients, that the muscles are weaker in the position in which they are shorter. Accordingly, the authors recommend specific training programmes to strengthen the weakened muscles, especially in these ranges.

Kinesiological studies have shown that the tibialis anterior is stronger in plantarflexion while the triceps surae, on the contrary, becomes weaker with increasing plantarflexion (55,56). As mentioned above, the tibialis anterior is stronger in hemiparetic patients and shows higher EMG activity than the triceps surae in the more involved leg (57,58). Improvement of the gait patterns after application of ankle-foot orthosis or splint can be explained, also, by the loading of the weakened triceps surae (21). From this point of view, surgical lengthening of the spastic muscles may in some cases further unload and weaken them.

Decreased activation of antigravity muscles, namely the gastrocnemius and quadriceps, could be a limiting factor in the treadmill/partial body support treatment. Weight acceptance is one of the key factors contributing to activation of the antigravity muscles and there is a correlation between weakness of the quadriceps muscle and the speed of hemiparetic gait (59). Table I summarises some of the paradoxes presented by the neurokinesiology of spastic gait.

### Contribution of viscoelastic factors

Dysfunction of spastic muscles and especially spastic plantarflexors is apparently related to alterations of mechanical properties of the muscles and especially of the supporting soft tissue structures. Given et al. (7) reported significant differences in the torque-angle hysteresis loop during passive movement between paretic and contralateral ankles, especially in ankle dorsiflexion, in hemiparetic patients. This was not observed during similar testing of the elbow. These authors confirm, in their system, the velocity insensitivity of passive muscle stiffness. They speculate that these findings are due to the higher amount of intramuscular connective tissue in plantarflexors in comparison with flexors and extensors of the elbow. The connective tissue surrounding the slow twitch muscle fibres is more sensitive to immobilisation than the connective tissue of fast twitch muscles (60). The effect of abnormal muscle activation upon viscoelastic properties of the muscle in spasticity is also supported by other studies (62,63).

Lehman et al. (63) measured sinusoidal displacement of 5° to measure elastic and viscous stiffness of ankle excursion at frequencies from 3 to 12 Hz. Both the

<table>
<thead>
<tr>
<th>Problem</th>
<th>Traditional view (empirical)</th>
<th>Evidence-based view (supported by gait analysis techniques)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triceps surae spasticity</td>
<td>The triceps surae is hyperactive and must be suppressed, its weakened antagonists must be strengthened.</td>
<td>The triceps surae is both absolutely and relatively (compared to its antagonists) weakened, and must be activated.</td>
</tr>
<tr>
<td>Stiff-leg gait</td>
<td>Hyperactive quadriceps is the culprit.</td>
<td>1. Phase-dependent dynamic weakness of hip flexors (iliopsoas and rectus femoris) during initiation of the swing phase contributes significantly. 2. Abnormal activation of the hamstrings in pre-swing and early swing phases interferes paradoxically with knee flexion in the swing phase.</td>
</tr>
<tr>
<td>Increased pelvic anteversion during terminal stance phase</td>
<td>Shortened iliopsoas is the culprit.</td>
<td>Phase-dependent dynamic weakness of hip extensors in terminal stance is the main factor.</td>
</tr>
<tr>
<td>Hip internal rotation deviation/deformity</td>
<td>Medial hamstrings and adductors are the culprits; elongation or tenotomy should be performed on them.</td>
<td>Internal-rotation moment of “weakened” gluteus medius dramatically increases during pathological hip flexion and may contribute to excessive hip internal rotation.</td>
</tr>
</tbody>
</table>

Detailed explanation in the text

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Table I - Paradoxes in the neurokinesiology of spastic gait.
Neurophysiology and kinesiology of spastic gait

elastic and viscous properties of the spastic muscles decreased around the mid-range frequencies and this phenomenon was probably related to a reflex response. To eliminate this reflex response, the authors also performed the study with persons after peripheral nerve blocks of the tibialis anterior and triceps surae. Significant differences emerged between the elastic properties of the passive tissues in the spastic group compared to healthy controls. The passive viscous stiffness was very similar in the spastic group with nerve blocks and in control groups. A total stiffness vector containing both elastic and viscous spastic response (expressed by Nyquist diagram) displayed a shift in passive properties during spastic responses in relation to the responses measured in patients with nerve blocks. These findings may, among other things, indicate early changes preceding contracture development (63).

Concluding remarks: implications for therapy

Spasticity is a complex problem that requires a multidisciplinary approach. Modern gait analysis tools have significantly improved our understanding of physiological and pathological gait. They also allow motor performance to be evaluated on a case-by-case basis, and are thus crucial in the planning of optimal treatment strategies. One of the main messages emerging from modern neurophysiological and kinesiological research is that spastic muscles should be activated and facilitated, as far as possible in accordance with physiological phasic and postural patterns. Therapeutic strategies used in the functional treatment of spasticity should be aimed at training and activating residual motor functions, suppressing pathological and unfavourable movement and postural patterns and preventing secondary complications. It should be emphasised that the effectiveness of antispastic therapy should be evaluated not in terms of reduced muscle tone per se, or of improved muscular strength in static situations, but rather in terms of improvements in gait performance. Functional goals should be clearly established not only from the kinesiological (impairment) perspective, but also considering the impact on disability and handicap.

In conclusion, the neurophysiology and kinesiology of spasticity and gait disturbances in central nervous system disorders constitute a complex and multifaceted issue. The wider application of the results of extensive basic and clinical research to clinical practice is a great and ongoing challenge.

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