REVIEW

Clinical neurokinesiology of spastic gait

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Abstract

Locomotor control requires a spatiotemporal coordination of passive and active forces across the movement system. Both anticipatory and reactive strategies operate in locomotor control. Mammalian locomotion is based on a rhythmic, „pacemaker" activity of spinal stepping generators. Reflex modification of the gait cycle is task-, context- and especially phase-dependent. In spasticity, together with disturbed supraspinal control, the phase-dependent reflex modulation of the gait cycle is severely impaired and there is altered modulation and timing of muscle activation and relaxation during voluntary movement. There is also a poor correlation between EMG activity and tension development in the spastic muscle. The tension increases without sufficient muscle activation and disconnection and dyscoordination between muscle activation, tension development and motor performance develops. The pattern of muscle activation and the development of increased muscle tone in patients with spasticity may be dramatically different in active movement from that observed in clinical testing of the passive muscles. Strategies used in the functional treatment of spasticity should be aimed at training and activating residual motor function, suppression of pathological and unfavourable movement and postural patterns and preventing secondary complications. In the 1990s a number of new specific instrumental methods and technical equipment supporting gait rehabilitation in patients with CNS lesions were developed: rhythmic auditory stimulation and other types of rhythmic stimulation, partial body support, usually with treadmill walking, complex orthotic support of bipedal locomotion, multichannel functional electrical stimulation, usually with programmable computer control, and advanced gait trainers. In therapy of spastic gait, the functional goals should be clearly determined from the kinesiological point of view of the impairment, and the impact on disability and handicap should be considered and a multidisciplinary approach is essential. ***(Ref. 139.)***

Key words: gait, kinesiology, neurophysiology, spasticity, therapy.

The human gait can be described as bipedal, orthograde, and plantigrade. It is unique among the mammals and only humanoid primates and partially beards can use similar patterns (although the quadruped gait is energetically much effective for these species). The fossil remnants and fossil tracks indicate that this type of humanoid gait developed about 1.5 million years ago. It is interesting, that in the phylogenesis the development of orthograde gait preceded the growth of the human brain. This fact may be related among other things (increased inflow to the telereceptors located on the head) to releasing the wrist-lock, which is essential for effective quadruped locomotion in primates. This wrist locking mechanism interferes with effective radial opposition of the thumb and with differentiated hand movements.

The term plantigradity of human gait reflects the fact that the main propulsive power of the normal gait in healthy adult humans is generated by the foot plantar flexors. Beside the shift of ground reaction forces from the heel to the toe (heel strike/toe off) the shift of ground reaction forces from lateral (fibular) part of the sole to the anteromedial part of the sole occurs during the stance phase. This mechanism is phylogenetically and ontogenetically "young", and is extremely demanding on perfect reflex and supraspinal control. And it is also the most critical and fragile part of the gait cycle both from the biome-

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chanical and the neurophysiological points of view (Sutherland et al, 1998; McFayden and Bélanger, 1997; Forssberg 1985).

Basic principles of the neuronal control of human gait

In neuromechanical terms, locomotor control requires a spatiotemporal coordination of passive and active forces across the movement system. These forces must dynamically maintain the integrity of the intended displacement within the plane of progression together with maintenance of equilibrium in all directions (McFayden and Bélanger, 1997; Massion and Viallet, 1990). Both anticipatory and reactive strategies operate in locomotor control. Mammalian locomotion is based on a rhythmic, "pacemaker" activity of spinal stepping generators.

There are at least four groups of spinal locomotor generators, one for each limb. The generator for nondominant lower limb appears to be the dominant pacemaker. There is some evidence that within each generator a distinctive half-centre of flexors and extensors, respectively, exists (Leblond, 2001). Human walking is a complex activity involving a large number of cyclical moving, not only of the legs, but also of the pelvis, trunk, arms and head. The "normal" 1:1 coupling between arms and legs occurs typically at normal velocities. The arm swing begins with paradoxical activation of posterior deltoideus and it is synchronised with ground contact of the foot. At lower velocities (below 2.5 km/h) in some persons a less stabile and less consistent 2:1 frequency coordination appears. The arm swing is synchronised with knee flexion and no posterior deltoideus can be observed (Donker et al, 2001; Wagenaar and Van Emmerik, 2000).

Functioning of the locomotor generators appears to be to a great extent autonomous. The motor program controlling complex movement including gait is, however, organised on multilevel, parallel, "holographic" principles (Sutherland et al, 1988; Morris et al, 1994; Johansson, 2000). Thus, for effective bipedal gait in humans, complex interactions of the rhythmic spinal activity, supraspinal influences, polysynaptic processing within the spinal cord and reflex spinal mechanisms are required.

Reflex modification of the gait cycle is task-, context- and especially phase-dependent. The same stimuli elicit different and even oposite (phase-reversed) response depending on the phase of the gait cycle. Supraspinal structures involved in human gait control are represented in particular by the brain stem reticular formation, basal ganglia, motor, premotor and supplementary motor area of the motor cortex, cerebellum (Zehr et al, 1998; Zehr and Stein, 1999; Barbeau and Rossignol, 1994; Dietz, 1997; Dietz and Wirt, 1997; Forssberg et al, 1975; Duysens and Van de Crommert, 1998).

It is worth emphasising that the spinal cord is not a rigid "hardware" structure but that it posseses great potential of neuroplasticity and motor learning (Barbeau and Rossignol, 1994; Dietz, 1977; Dietz and Wirt, 1997). Further, research into the neurophysiological background of locomotion has questioned the traditional principles of motor control. Namely, the basic terms „reflex", „automatic" or „voluntary" need to be revised (Prochazka et al, 2000; Zehr and Stein, 1999).

Pathophysiology of spasticity

The key structures involved in the pathogenesis of spasticity are extrapyramidal (or nonpyramidal) projections, such as rubrospinal, tectospinal, reticulospinal and vestibulospinal tracts. These structures have not only functional but also anatomical relations to the corticospinal tract. Selective destruction of the corticospinal tract is thus rare and when it occurs results in hypotonia (Bucy and Keplinger, 1994; Braun, 1994; Young, 1994). In spinal cord lesions, a direct lesion of spinal interneuronal pool may further substantially contribute to the development of spasticity (Braun, 1994; Katz and Rymer, 1989; Young, 1994; Dietz and Wirt, 1997; Chapman and Wiesendanger, 1982; Braun, 1994; Gilman et al, 1974). An important current view states that spasticity is caused by long-term reductions in inhibition. Synaptic input is reduced (recurrent Renshaw cell inhibition, presynaptic inhibition on Ia fibres, reciprocal Ia inhibitory interneurons, nonreciprocal inhibition by Ib afferent fibres, especially Ib nonreciprocal inhibition). Further, gamma efferents are described as being hyperactive, and denervation hypersensitivity and sprouting can contribute. A decrease in presynaptic inhibition has been described by some authors. Motoneurons may have, for a number of reasons, increased intrinsic excitability. Polysynaptic connections mediating the flexor reflex afferents and nociceptive reflexes are disturbed and up-regulated (Delwaide and Pennisi, 1994; Young, 1994; Braun, 1994; Katz and Rymer, 1989; Cheney, 1997). The loss of control over presynaptic inhibition of the motor neuron pools is one of the most obvious neurophysiological mechanisms underlying spasticity (Stein, 1995). This corresponds well to the lambda model of motor control suggesting that the motor neuron threshold properties play an important role in the regulation of active movement (Feldman and Levin, 1995).

Some studies indicate, in contrast to classical concepts (Lance, 1980), no preference for dynamic or static reactivity (Katz and Rymer, 1989; Sinkjaer et al, 1993; Ada et al, 1998).

Moreover, the phenomenon of phasic, velocity-dependent spasticity (Lance, 1980), cannot be sharply delineated and separated, especially from the clinical point of view, from other consequences of central nervous system lesion (Young, 1994; Katz and Rymer, 1989; Dietz et al, 1981; Given et al, 1995; Barnes, 1998). In most cases, the motor performance in spasticity is usually further worsened by factors resulting from central nervous lesions — hemineglect, anosognosia, apraxia, aphasia, senzoric impairment and altered senzoric, especially proprioceptive and visuomotor processing (Piek and Coleman-Carman, 1995).

From the neurobiomechanical point of view, there is a poor correlation between EMG activity and tension development in spastic muscle. The tension increases without sufficient muscle activation and the disconnection and dyscoordination between muscle activation, tension development and motor performance develops (Berger et al, 1984; Dietz, 1997; Tang and Rymer, 1983). Moreover, the muscle is maximally activated away from the optimal angle of the segment (Katz and Rymer, 1989).

Abnormal muscle coactivation instead of fluent inhibition-activation interplay includes cocontractions of agonists and an-

tagonists, coactivation of limb proximal and distal muscles) the inability to perform isolated movements and to control the correct timing of muscle (Woollacot and Burtner, 1996; Katz and Rymer, 1989; Carmick, 1995; Yokochi et al, 1991). It is worth mentioning that a pathologic increase of cocontraction patterns is not typical only for spasticity. In low back pain patients an increase in the trunk flexor/extensor coactivation pattern has been reported. It is questionable whether this increased coactivation represents a desirable stabilisation phenomenon or it reflects simply impaired motor control in these patients (Radebold et al, 2000).

The phase-dependent reflex modulation of the gait cycle is severely impaired and altered modulation and timing of muscle activation and relaxation during voluntary movement occurs (Zehr and Stein, 1999; Zehr et al, 1998; Carmick 1993). The pattern of muscle activation and the development of increased muscle tone in patients with spasticity may be dramatically different in active movement from that observed in clinical testing of the passive muscles (Dietz and Wirt 1997; Thilmann et al, 1991). 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 seems to be here more appropriate (Young, 1994).

Disturbances of the reciprocal control of eccentric and concentric movements of antagonists is another important factor in spastic neurophysiology. In healthy subjects, the level of activation of the quadriceps muscle in concentric knee extension increases with velocity, and this may be due to smaller afferent inflow from tendon organs at slower velocities (Komi et al, 1987; Westing et al, 1991). In spastic patients, there is no increase in the quadriceps EMG activity in concentric knee extension at fast speeds. Instead, the agonist activity is decreased with increasing velocity. This fall in agonist EMG activity may be due to inhibitory interneurons activated by Ia afferents from spastic antagonists (Ashby and Wins, 1989; Knutsson et al, 1997; Berbayer and Ashby, 1990).

On the other hand, the physiological reciprocal inhibition from agonist to antagonist motorneurons, which is controlled in parallel by Ia afferents and supraspinal commands, is reduced in spastic patients especially during voluntary dynamic action of high effort. In contrast, reciprocal inhibition in the opposite direction (i.e. from spastic antagonist to voluntary activated agonist motor neuron), seems to be preserved in spastic patients. This is one cause of the suppression of voluntary activation, at least in spinal spasticity. The above mentioned mechanism probably underlies the asymmetry of muscle activation during concentric and eccentric movement in these patients. The dynamic motor capacity is severely compromised in concentric movements, especially at high velocities. On the other hand, voluntary strength may be relatively well preserved in eccentric movements (Knutsson et al, 1997). The asymmetry of the reciprocal inhibition may be related to the finding that co-contraction of antagonists in spasticity in stroke is greater during knee extension than flexion (Newham and Hsiao, 2001).

Spatiotemporal (time-distance), kinematic and kinetic characteristics of spastic gait

The detailed kinesiological characteristics depends naturally on the type and localisation of the CNS lesion(s), on the course and severity of underlying disease or trauma and on many other factors (intensity and quality of rehabilitation, complicating conditions, psychosocial background etc). There are great walking differences between hemiparetic, paraparetic or quadruparetic subjects. The gait disturbances are different in patients with focal, multifocal, and diffuse CNS lesions. Even in patients with the same diagnosis, such as in cerebral palsy, several distinctive and kinesiologicaly different types of gait patterns can be distinguished (Sutherland et al, 1969; Sutherland and Davids, 1993; Lin et al, 2000). Despite great variability of spastic gait patterns some common features and problems can be observed and pointed out. Attributes of normal locomotion are characterised by smooth forward progression of the centre of gravity and by coordinated and controlled intralimb and interlimb movement and phasing (Giuliani, 1990). In spastic patients, this is replaced by mass limb movement patterns and by altered phasing between limbs. Particular components of the gait cycle are dissociated.

Hemiparetic gait

Hemiparetic gait in stroke survivors is among the most investigated neurological gait disorders.

A pronounced asymmetrical deficit is typical for hemiparetic gait. Perry et al (1978) gave a description of hemiparetic gait and the author’s conclusions have been confirmed by later studies. One of the basic problems here is poor single-limb balance and difficulties of controlling forward movement. The gait asymmetry includes decreased stance time and prolonged swing period of the involved lower limb. Stance phase does not prepare the body for forward progression. Weight distribution biases toward the noinvolved lower limb.

Subjects with hemiparesis have a slower walking speed, shorter stride length and cycle duration. Stance phase is shortened and the swing phase lengthened in the paretic limb compared to healthy individuals. To compensate these changes, the uninvolved limb has an increased stance and decreased swing phase. Periods of double-limb support are longer in the hemiplegic than in normal subjects. The paretic limb has a shorter stance time and step length than the non-involved leg (Wall and Turnbull, 1986; Dettmann et al, 1987; Roth et al, 1997; Mizrahi et al, 1982; Branstater et al, 1983; Knutsson, 1981; Guiliani, 1990; Olney et al, 1990, 1994).

When investigating the spatiotemporal dynamics of ground reacting forces, transfer of the initial foot contact from hindfoot (heel strike) to forefoot, increased lateral plantar support, limited rolling-over, reduced or absent push-off have been found on the hemiplegic side. The maximal anteromedial force tends to decrease and the anterior force difference tends to increase. This finding reflects abnormalities of propulsion dynamics. The normally occuring medial force shift from the fifth to the first meta-

tarsal head (corresponding to pronation of the forefoot at the end of propulsion) is absent or inverted (Gaviria et al, 1996).

As mentioned above, abnormal movement behaviour can also be found in the non-hemiplegic („non-involved“) side. There are longer intervals of support associated with generally greater ground reacting forces. The flat foot contact interval tends to increase and cover the whole stance phase on both sides, but more on the non-hemiplegic side. The above-mentioned impairment of the medial force shift (forefoot transfer) seen on the hemiplegic side can be observed on the non-hemiplegic side, too. Ground contact is usually shorter on the hemiplegic than on the non-hemiplegic side. On both sides, a reduction of weight acceptance at the end of the stance phase has been described (Gaviria et al, 1996).

The finding of kinesiological abnormalities on the non-involved side are related to a great extent to compensatory and adaptative mechanisms, although the direct effect of impaired motor control can also be considered (Gaviria et al, 1996; Roth et al, 1997).

Gait in other diseases and injuries of CNS

There are great variations in the patterns of basic temporospatial characteristics of gait in subjects with other CNS disease and lesions. In hemiparetic gait in traumatic brain injury significant differences compared to hemiparesis resulting from stroke can be found. Ochi et al (1999) reports faster walking speed and longer step length in traumatic brain injury patients compared to ambulatory stroke survivors. The stance period is prolonged for the unaffected limb, without a longer stance period for the affected limb.

The gait parameters in subjects with spinal cord injury depend on the level of the injury. Subjects with thoracic lesions demonstrate reduced cadence, forward velocity and knee angular velocity while patients injured in the lumbar region have reduced stride length and ankle velocities (Krawetz and Nance, 1996; Kerrigan et al, 1999).

Children with cerebral palsy may demonstrate gate velocities comparable with those of healthy children. However, the inefficient gait biomechanics leads to a significant increase in energy costs and to increased cardiorespiratory workload (McNevim et al, 2000; Unithan et al, 1996). When the lesion is acquired before maturation of normal gait, the basic reciprocal pattern of muscle activation during locomotion does not develop or is severely impaired. The main and most cumbersome feature of an early immature gait pattern consists in coactivation of all leg muscles during the stance phase (Dietz and Berger, 1995; Dietz, 1997; Cheney, 1997). Stepping generators are active in neonates. However, the pattern of newborn stepping is quite different from mature human independent ambulation. Marked hip flexion, digitigrade strike pattern, and coactivation of antagonistic muscles lead to kinesiologically ineffective synchronisation of particular segment movement. External support is needed to initiate the gait cycle and maintain postural control. Independent walking is characterised by a more vertical posture, with decreased flexion of the hip and knee, increased step length, desynchronisation of the hip, knee, and ankle joint. Transformation to this pattern occurrs relatively rapidly prior to age 3 and then more slowly until age of 5 years, when the mature pattern is almost complete (Thelen and Cooke, 1987; Forssberg, 1985; Leonard, 1990; Riach and Hayes, 1987; Rang et al, 1986; Winter et al, 1987; Berger et al, 1984).

Axial motor system

The importance of the axial motor system including abdominal muscles and mechanisms of head stabilisation for execution of effective gait has been increasingly recognised. In patients with spasticity, alterations in axial kinesiology are studied mostly in nonambulatory situations (stance balance, sitting balance, reaching tasks, stand sway, sit-to stand tasks (Burtner et al, 1998; Seelen et al, 1998; Hadders-Algra et al, 1999; Hodges et Richardson, 1997; Woollacot and Burtner, 1996). It is worth mentioning the poly EMG study of Dickenstein et al (1999) of trunk muscles in hemiparetic and hemiplegic patients during dynamic symmetric voluntary activities. Compared to controls, hemiparetic patients displayed lower levels of activation of trunk extensors, while the activity of recti abdominis were comparable between both groups. For both muscles, EMG activation on the paretic side was not lower than the nonparetic side.

Less is known about the behaviour of the axial motor system in spastic gait. Erectores spinae show two crests of activity occurring in the interval of 0—20 % and 50—70 % of the gait cycle. Their function is to diminish the forward and the lateral movement during the initial terminal double support phases. In hemiparetic subjects, the reduction of the first peak on the treadmill with the partial body support can be beneficial by impending the trunk movement in the sagital plane (Hesse et al, 1999; Thorstenson et al, 1984).

The location of several sensory systems in the head implies that maintenance of head stability may be a potentially important part of locomotor activity (Pozzo et al, 1990). Maintaining dynamic stability of the head is an important task in preventing falls during locomotion (Holt et al, 1995). Holt et al (1999) found increase in mean head fluctuations in period of the head during treadmill walking in children with cerebral palsy in comparison to healthy children and healthy adults. This finding reflects decreased dynamic stability of the head trajectory during locomotion.

Implications for therapy

The problem of spasticity requires a multidisciplinary approach and a complex scope. One of the main messages ensuing from the modern neurophysiological and kinesiological research is that the spastic muscles should be activated and facilitated, as much as physiological phasic and postural patterns are. Suppression of spastic muscle groups by nerve blocks, botulotoxin, surgery etc should be done only after careful and cautious interdisciplinary analysis. Modern tools of gait analysis have signifi-

cantly improved our understanding of physiologic and pathologic gait and they allow an evaluation of motor performance on a case-by case basis and provide great support for choosing an optimal treatment strategy.

Therapeutic strategies used in the functional treatment of spasticity should be aimed at training and activating residual motor functions, suppression of pathological and unfavourable movement and postural patterns and preventing secondary complications. Traditionally, a number of complex physiotherapeutic strategies worked out on an empirical basis and based on experimental neurophysiological background are widely used in patients with spasticity (Brunstrom, 1970; Barry, 1996; Hastings-Smith and Sharp, 1994; Knott and Voss, 1968; Bobath, 1978; Vojta, 1984). In general, the intended aim of these techniques is to normalise the postural and reflex backgrounds, provide a „framework", for more normal locomotor activity, and to suppress the pathological movement and undesirable tonic and postural patterns. This should enable the paretic and spastic muscle group to work in a kinesiologicaly more effective manner. Novel concepts of rehabilitation include a *goal oriented and task specific approach* (Carr and Shepperd, 1989; Seif-Naraghi and Herman, 1999; Dean et al, 2000). Emotional wellness, motivation, and appropriate social context all should support therapy. There is an increasing effort to evaluate the effectivity of these approaches in controlled trials (Hesse et al, 1998; Girolami and Campbell, 1994).

In the 1990s, a number of new specific instrumental methods and technical equipment supporting gait rehabilitation in patients with CNS lesions have been introduced into clinical practice and their beneficial effect is partially documented in controlled clinical studies. They are as follows:

— rhythmic auditory stimulation and other types of rhythmic stimulation,
— partial body support, usually with treadmill walking,
— complex orthotic support of bipedal locomotion,
— multichannel functional electric stimulation, usually with programmable computer support,
— advanced gait trainers.

There is an interesting dynamic parallel between the temporal nature of auditory information and movement performance. Rhythmic auditory stimulation has a direct tuning, phase-anticipatory effect on the gait cycle via reticulospinal afferents. Auditory rhythmic patterns exert a strong „magnet" effect on the timing of the motor response. The effect is immediate and can persist several weeks following termination of the therapy. The efficacy of this method in the support of gait rehabilitation has been proven in cases of stroke, Parkinson,s disease, traumatic brain injury, Huntington's disease (Prassas et al, 1997; Thaut et al, 1997; Thaut et al, 1999; McIntosh et al, 1998; Hurt et al, 1998). Other types of rhythmic stimulation have been reported. For example, in less impaired patients, a strong rhythmic effect of running or even jumping can be used (Schallow and Zäch, 2000 a). Important means of rhythmic stimulation of gait cycle is represented by synchronisation with rhythmic activity of the therapist. Interindividual coordination of the rhythmic movement belongs to basic movement phenomena. Typical examples are marching, dancing, horse riding (and hippotherapy). It is phylogenetically very old mechanism, related to defence and migration (herds of fishes, great herbivora, birds). Perfect step synchronisation occurs during prey hunting, especially by feline predators. Interindividual tuning reduces energy costs and allows the predator to optimise the starting point for the final attack. Fossil footprints demonstrate, that similar „tuning" tactics were used by the great carnivore dinosaurs (Thomas and Farlow, 1997; Schalow and Zäch, 2000 b). Using this approach one must be aware that not only a transfer of the therapist's rhythm to the patient but also an undesirable transfer of the patient's rhythm to the therapist may occur (Schalow and Zäch, 2000 b).

Synchronisation in locomotion appears to be more general phenomenon. During running, the locomotor rhythm approximates to the cardiac rhythm (at frequencies around 2.5 Hz) and synchronisation between them has been reported. The question is whether the synchronisation relates to a neurophysiological mechanism or occurs by chance. The results of Nomura et al (2001) indicate, that this phenomenon might represent rhythmic entrainment.

The unweighting — *partial body support* with „parachute" harness activates the gait stepping generators, leads to a more regular gait pattern, reduces spasticity of the plantar flexors, and improves gait symmetry in hemiparetic subjects. External assistance of the gait cycle is usually required during the transition from stance to swing. Knee and hip joints should be fully extend during the stance phase to optimally support body weight. Hyperextension of the knee joint should be avoided. The pelvis should be kept fixed, so that loading of the knee and ankle occurs in a physiological manner. Optimal velocity and rhytmicity must be grasp, „tuned". Increase in the velocity of the spastic gait should be achieved by reducing the duration of the stance phase rather than the swing phase (Gardner et al, 1998, Dietz and Wirt, 1997; Hesse et al, 1999 a; Wernig et al, 1999).

The idea of *functional electric stimulation* is not new (Liberson et al, 1961). Recently, multichannel, computer-aided stimulation, sometimes using biofeedback controlled artificial intelligence, has been used for some patients. However, there are some limitations such as time variance and non-linear characteristics of the response and technical problems with standardisation of the stimulation. Therapists using multichannel stimulation in gait rehabilitation in spastic patients must be aware of the possibility of abnormal and even inverted responses to stimulation (e.g. withdrawal flexor activation during quadriceps stimulation (Stein, 1999; Dimitrijevic, 1994; Tong and Granat, 1999; Gallien et al, 1995; Moynahan et al, 1996; Zehr and Stein, 1999).

A well-designed *gait trainer device* should permit performance of gait-like activity in physiological proportions as much as possible, especially with regard to the optimal ratio of swing and stance phases. Further it should assist weight-shifting, to control the vertical and horizontal displacement of the centre of mass and maintain trunk erection. An important contribution of this approach is to save the energy cost of both patients and the therapist, which allows a focusing of the therapeutic

effort on qualitative improvement of particular gait phases (Hesse et al, 1999 b).

A *combined (hybrid) approach* usually includes various combinations of partial body support, treadmill walking and multichannel functional electrical stimulation, sometimes combined with advanced orthotic support and walking trainers. There is increasing effort to use complex walking support in otherwise nonambulatory patients (Ferguson et al, 1999; Hesse et al, 1995; Ijzerman et al, 1999; Wieler et al, 1999).

The outer tuning of the gait cycle and involuntary or reflex techniques can improve the kinesiological background, but effective motor learning requires active participation (Van der Weel et al, 1991). Electromyographic or goniometric *biofeedback* can contribute to restoring the physiological pattern of gait (Olney et al, 1989; Brown and DeNacher, 1987).

The role of the *pharmacotherapy* (including intrathecal application of the antispastic drugs) in complex rehabilitation of spastic gait disorders is unquestioned. Integral parts of multidisciplinary approach to gait rehabilitation may include pharmacological support focused on spasticity reduction, neuroprotection and neuroplasticity, and activation of spinal stepping generators. There is a real perspective that the pharmacotherapy will be used not only to reduce the spasticity, but also for stimulation of the stepping locomotor generators (Leblond et al, 2001).

It should be emphasised that the effectiveness of antispastic therapy should be evaluated not in terms of reduced muscle tone or in improved muscular strength in the static situation but rather according improvement of gait performance (Barnes, 1998; Orsnes et al, 2000; Delwaide and Pennisi, 1994; Dietz, 1997; Barnes, 1998; Kirschblum, 1999; Nance and Young, 1999; Penn et al, 1995).

Splitting, casting, orthotics, phenol nerve blocks, botulotoxin application other modalities can, after careful and complex analysis, further significantly improve the results of gait therapy (Barnes, 1998; Bentivoglio and Albanese, 1999; Barry, 1996; Young, 1994; Botte et al, 1985; Dietz, 1977; Kirschblum, 1999).

The indications for partially or completely irreversible *surgical procedures* such as neurotomies, root and dorsal root entry zone lesion (DREZ-tomy) lesion, tendotomies and tendon transpositions, muscle lengthening, osteotomies, must be considered after careful, thorough and multidisciplinary analysis respecting the above delineated complexity of gait neurophysiology and biomechanics in spasticity. The functional goals should be clearly determined from the kinesiological point of view reflecting the impairment and the impact on disability and handicap must also be considered (Barnes, 1998; Smyth and Peacock, 2000; Dietz, 1977; Kirschblum, 1999; Hoffer, 1986; Sutherland et al, 1990).

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