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Muscle Eccentric Control in Gait Initiation

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국문요약

보행 시작 시 원심성 근육 수축 조절

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전시냅스 억제(presynaptic inhibition)와 동시냅스 억제(homosynaptic depression)는 보행 시에 분절반사(segmental reflex)를 조절하는 두 가지 독립적인 기전이다. 근방추 피드백(feedback)은 전시냅스 억제(inhibition)를 통해 보행 시 원심성 근육 수축기에서 적절이 조절될 수 있다. 이러한 전시냅스 억제 작용은 H-reflex의 강도로 나타내질 수 있는데, 원심성 근육 수축기 동안 H-reflex의 강도가 약해지는 것으로 보아 전시냅스 억제 작용은 증가되는 것으로 보여진다. 근방추 구심성 피드백(feedback) 역시 동시냅스 억제를 통해서 조절될 수 있다. 따라서 전시냅스와 동시냅스 억제는 보행 시작 중에 반사의 기전을 조절하는 중요한 역할을 한다. 반사의 조절 기전은 알파(alpha) 운동 신경원의 흥분도와 더불어 상위 척수의 기전들을 통해서 영향받고 조절된다. 경직성 마비 환자들은 초기의 입각기, 혹은 유각기 중에 손상된 비정상 비복근 H-reflex 조절기전을 보여준다. 이러한 비정상적인 조절기전은 발바닥의 말초신경을 자극함으로써 부분적으로 회복될 수 있다.

핵심단어 : 동시냅스 억제; 분절 반사; 원심성 근육 수축; 전시냅스 억제; H-reflex.

Introduction

Patients with central nervous system (CNS) disease usually show great difficulty bringing their tibia over the support foot during the stance phase of gait (Perry et al, 1974). Normally, a certain degree of tension in the triceps surae (developed during standing to maintain upright stance for postural regulation) should be overcome during walking, such that the body can progress forward (Fung and Barbeau, 1994). It is thought that this limited ability, which patients with CNS dysfunction experience during walking, is related to an impaired modulation of the spinal reflex system (Yang et al, 1991). Normally, the segmental reflexes provide the CNS with the means to regulate muscle stiffness (Houk, 1979) and produce a proper adaptation in the musculoskeletal system (as a response to the changing environmental conditions). These are known to be highly modulated during movement as a function of different body positions (Capaday and Stein, 1986, 1987). Moreover, the amplitude of the H-reflex is increased in patients with CNS dysfunction (Sinkjaer et al, 1995; Yang et al, 1991) interfering with their ability to gate peripheral afferent feedback during forward

progression (Yang et al, 1991). This may be due to interrupted descending input to spinal interneurons as a result of an injury to the CNS (Ashby and McCrea, 1987).

During voluntary movement the overall neural drive to motor neurons is contributed to by both the supraspinal control and spindle afferent feedback (Matthews, 1986), which increases or decreases the overall neural drive to the motor neurons, according to the load on the muscle (Burke et al, 1978; Matthews, 1986). These changes in reflex excitability are a mechanism with which to match the nervous command to the mechanical properties of the muscle (Romano and Schieppati, 1987). During muscle isometric and shortening muscle activation the efficacy of the excitatory connections between the Ia inputs to the motor neurons is increased (the gain of the stretch reflex pathway) in order to enhance the overall neural drive. This mechanism appears to be appropriate in allowing the muscle to increase its force by recruiting more motor units when the force would otherwise decrease due to the decrease in muscle length (Romano and Schieppati, 1987). This result is in agreement with the fact that during shortening muscle activation additional motor units are necessary to overcome the disadvantage caused by the decrease in the muscle spindle activity (owing to a relative unloading of the slower intrafusal muscle fibers) (Burke et al, 1978; Romano and Schieppati, 1987). During lengthening muscle activation such as seen with the soleus (which undergoes an active lengthening activation during most of the stance phase) the muscle being activated would produce a greater force due to the increase in its length and to the increase in its lengthening velocity (Romano and Schieppati, 1987). The synaptic effectiveness of the spindle afferent feedback to the motor neurons becomes reduced progressively in order to regulate the level of muscle contraction (Romano and Schieppati, 1987).

Modulation of the presynaptic inhibition is reflected in the level of amplitude of the H-reflex at a constant EMG level, so task-dependent changes in the H-reflex amplitude (such as walking, standing and running) at a constant EMG level are due to the presynaptic inhibition. For example, during eccentric muscle activation, the presynaptic inhibition should increase to account for the lower amplitude level of H-reflex at a constant level of EMG (Stein, 1995). Homosynaptic depression is another mechanism responsible for gating primary afferents feedback (Curtis and Eccles, 1960). In pathological conditions the decrease in homosynaptic depressions is responsible for the decreased vibratory inhibition in spastic patients (Nielsen et al, 1993). It may result in the exaggeration of stretch reflexes (may be involved in the pathophysiology of spasticity) (Nielsen et al, 1993).

These two neural mechanisms are functionally important during walking, especially during gait initiation. The disruption in such mechanisms, which is shown in the patients such as spinal cord injury (SCI) and multiple sclerosis (MS), interferes with their forward progression during walking (Yang et al, 1991). Normally, in the early part of the stance phase, as the ankle dorsiflexes and the body progresses forward (soleus muscle

lengthens), the soleus H-reflex is depressed in normal individuals. This may prevent excessive reflex activation from the increased spindle afferent feedback associated with the eccentric lengthening of triceps surae as it is stretched (Yang et al, 1991). However, in many patients, the increased reflexes at this period produce large stretch-induced responses which interfere with the smooth forward advance over the body and tibia (Yang et al, 1991). During the swing phase in normal subjects the low H-reflex amplitude, which represents the increased presynaptic and reciprocal inhibition, allows the ankle to dorsiflex without any interference from the stretch-induced strong reflex of the triceps surae. In contrast, patients whose reflex modulation is impaired such as SCI or MS show abnormally active reflexes, resulting in difficulty clearing the foot over the ground (Sinkjaer et al, 1995; Yang et al, 1991).

The purpose of this paper is to determine and review the factors that contribute to the neural control of the eccentric muscle activation during gait initiation. First, the modulation of segmental reflexes will be studied, followed by the presynaptic inhibition and finally homosynaptic depression. Second, clinical implications will be discussed.

The segmental reflex during movement

Segmental stretch reflexes serve to supply the CNS with both a control of muscle stiffness, and a rapid adaptive role to changing environmental conditions (Houk, 1979). Changes in the gain of stretch reflex can occur independent of the level of muscle activation and loading (Akazawa et al, 1982; Capaday and Stein, 1986; Koceja et al, 1995). This mechanism allows the body to transfer smoothly during gait. The modulation of the stretch reflex gain can be varied when subjects change their positions (Katz et al, 1988; Miyake et al, 1984). At equal stimulus intensities and background EMG activation, the amplitude of the H-reflex is larger during standing as compared to walking (Capaday and Stein, 1986; Crenna and Frigo, 1987). In addition, the amplitude of H-reflex is lower during a standing position as compared to a sitting position (Katz et al, 1988). Koceja et al (1993) reported that the amplitude of the soleus H-reflex is decreased 16% during a quiet standing as compared to the prone position. Capaday and Stein (1987) proposed that the soleus segmental stretch reflex is depressed as a function of an increased postural instability. For example, in humans during beam walking, lower reflex gain was achieved, as compared to normal treadmill walking (Liewellyn et al, 1990). As task complexity increases, fusimotor drive is also increased (Koceja et al, 1995), but at the same time the segmental reflex gain appears likely to be reduced in order to maintain an appropriate level of muscle activation and stiffness. Through this mechanism, both the appropriate level of muscle activation and muscle stiffness are achieved. For example, during standing, which is viewed as a static posture, the depression of the soleus stretch reflex (reduced reflex gain) is produced to prevent a saturation of the primary afferent feedback from occurring or from developing instability (Koceja et al, 1995; Stein and Capaday, 1988).

Therefore, the reduced modulation of the soleus H-reflex becomes greater in proportion to the greater degree of postural instability. This occurs because the increase in the velocity of postural sway, due to the reduced base of support, provides a greater muscle spindle discharge. As a result, the decreased gain of the segmental reflex is needed (Koceja et al, 1995). In pathological conditions, the importance of this mechanism to stability in standing is evident when the inability to modulate the segmental stretch reflex in a condition often seen in the patient population may create a destabilizing oscillation, as well as a difficulty responding and adapting to postural perturbations in standing (Koceja et al, 1995; Mauritz et al, 1979).

An increased presynaptic modulation of the Ia afferents to the alpha motor neuron is closely related to the decreased level of soleus H-reflex (Yang et al, 1991). During the walking cycle, the amplitude of the soleus H-reflex is strongly modulated (Capaday and Stein, 1986). Capaday and Stein (1986) found that the H-reflex was increased rapidly to a maximum level during stance in order to assist in maintaining the upright position of the body against gravity and eventually lifting the body off the ground. In addition, after the toe-off phase of the stance limb it was abruptly decreased to a low value (Capaday and Stein, 1986). During the swing phase the reflexes were smallest or absent while attempting to oppose ankle dorsiflexion (Capaday and Stein, 1986).

Presynaptic inhibition

The changes in presynaptic inhibition of the la afferent on the motor neurons are responsible for gating spindle afferent feedback. The presynatic inhibition of the la afferents to motor neurons of the contracting muscles is decreased (Hulborn et al, 1987). Hulborn et al. (1987) demonstrated that such a decrease in the presynatic inhibition to the contracting muscle allows the la activity to contribute to the excitation of voluntarily activated motor neurons (the increase in the effectiveness of the muscle spindle feedback). In contrast, the presynaptic inhibition of the la afferents to motor neurons of the uninvolved muscles is increased (Hulborn et al, 1987). The increase in the presynaptic inhibition on the Ia afferents to motor neurons of the uninvolved muscles might prevent the motor neurons of those muscles from being activated, permitting the proper role of the antagonistic muscle activation (Hulborn et al, 1987).

At the beginning of the voluntary movement contraction the presynaptic inhibition of the Ia afferents to the motor neurons is decreased, resulting in the increase of the gain of the monosynaptic stretch reflex (Meunier and Pierrot- Deseilligny, 1989). A descending inhibition of presynaptic interneurons, which is already established at the onset of movement, might contribute to a decrease in presynaptic inhibition (Meunier and Pierrot-Deseilligny, 1989). The descending control selectively inhibits those interneurons which are the mediating presynatic inhibition of the la afferents (which project to voluntarily activated motor neurons) (Meunier

and Pierrot-Deseilligny, 1989). Later, as the presynaptic inhibition is increased, the gain of the reflex is decreased (Meunier and Pierrot-Deseilligny, 1989).

Changes in presynaptic inhibition are centrally programmed (Meunier and Pierrot-Deseilligny, 1989). Hulborn et al (1987) described the changes in the presynaptic inhibition of the Ia afferents to motor neurons as being supraspinal control in origin and serving as an aid in achieving a selectivity of the muscle activation in order to increase the motor control. Additionally, at the beginning of a movement, a high gain may be an advantage which allows the monosynaptic reflex to compensate rapidly for the actual load (because the actual load is not yet decided at the initial phase of movement) (Meunier and Pierrot-Deseilligny, 1989). Later, the decrease in the gain may be necessary in order to prevent oscillations from building up (Meunier and Pierrot-Deseilligny, 1989).

In pathological conditions such as seen in SCI patients, and MS patients with spasticity, the reduced presynaptic inhibition of the la primary afferent fibers is responsible for hyperactive tendon and H-reflexes (Nielsen et al, 1993; Sinkjaer et al, 1995; Yang et al, 1991). Therefore, with the impaired or reduced presynaptic inhibition directed at the Ia afferent terminals these patients may have a higher reflex gain contributing to the clonus encountered during walking (Yang et al, 1991). At similar background levels of soleus activity these patients show a greater soleus H-reflex amplitude in walking than during standing (Fung and Barbeau, 1994). These patients even show a greater amplitude of the soleus H-reflex during midswing than in the static unloading position at the same degree of joint angle (Fung and Barbeau, 1994). The manifestation of spasticity that eventually leads to the exaggerated stretch reflexes and clonus shown in the CNS patients is reflected in the reduced spinal presynaptic inhibition (Burke and Ashby, 1972; Delwaide, 1973; Fung and Barbeau, 1994; Pierrot-Deseilligny and Mazieres, 1985).

Homosynaptic depression

The depression of the soleus H-reflex tends to last for several seconds when it is elicited by any of the following: a slow passive stretch of the soleus muscle, short-lasting vibration of the Achilles tendon, and/or a previous voluntary contraction (Crone and Nielsen, 1989; Katz et al, 1977). This post-activation depression, which is evoked exclusively by either the stretched or the activated muscle (not in other muscles), appears to be caused by homosynatic depression which is the long-lasting depression of the spinal monosynaptic transmission (Curtisa and Eccles, 1960; Ballegaard et al, 1991). This depression is due to a decrease in synaptic transmission in the sensory neurons after the activation of reflex pathways (Capek and Esplin, 1977).

Nielsen et al (1993) reported that post-activation depression of the soleus H-reflex, which is evoked by a slow passive stretch of the soleus muscle, is significantly smaller in SCI patients than in healthy subjects. This suggests reduced homosynaptic depression. A decreased post-activation, likely

caused by a mechanism similar to the homosynaptic depression, will result in an exaggeration of the stretch reflex (Nielsen et al, 1993). In other words, the reduced homosynaptic depression may increase the reflex gain. In addition, the increased reflex gain would develop a greater muscle activation (increased level of muscle activation). A good example is the fact that the homosynaptic depression is responsible for controlling the effectiveness of the muscle spindle la afferent feedback.

Clinical implication

Spastic paretic patients show the impaired soleus H-reflex modulation in the initial stance phase or during the swing phase as a result of spinal cord or head injury as compared to normal subjects (Yang et al, 1991). In normal subjects the H-reflex should be low during the early stance and swing phase. A low H-reflex allows the tibialis anterior activity and ankle dorsiflexon movement to be unopposed by the stretch reflex evoked in the triceps surae (Capaday and Stein, 1986). In contrast, spastic SCI patients show abnormal activity in the silent phase of the gait cycle such as triceps surae bursting, and even triggering clonus (Fung and Barbeau, 1994). This is owing to a defective Ia regulatory mechanism resulting from the disruption of supraspinal and/or propriospinal influence (Yang et al, 1991). Yang et al.(1991) reported that an inadequate control of the presynaptic inhibition that terminates at Ia afferent terminals may be responsible for a diminished or absences of H-reflex modulation observed in spastic paretic subjects. In those circumstances the spindle afferents feedback of the triceps surae increases considerably leading to the exaggerated stretch reflexes and clonus and making the forward advance of walking difficult (Fung et al, 1990; Yang et al, 1991).

The abnormal Ia modulatory mechanisms which occur during walking can partially and artificially be restored by the application of a peripheral stimulus to the sole of the foot, provided that the segmental circuitry remains intact (Fung and Barbeau, 1994). The underlying segmental mechanisms, which are responsible for the conditioning inhibition of an otherwise excessive H-reflex in the early stance and swing phases of gait (in spastic SCI patients), are presynaptic and postsynaptic inhibition. In paraplegic patients a significant and regular decrease of the heteronymous Ia facilitation was observed from the quadriceps and soleus when the flexor reflex afferent stimulation evokes presynaptic inhibition to Ia transmission (and then to alpha motor neurons) (Roby-Brami and Bussel, 1990). This result strongly suggests that the presynaptic inhibition can be mediated by intraspinal mechanisms. In addition, the functional organization of the spinal cord in SCI patients is similar to that seen in an acute spinal cat injected with DOPA (Jankswska et al, 1967). It is highly possible that the intraspinal mechanism by which presynaptic inhibition is mediated is similar to a depolarization of both the ipsilateral (Anderson et al, 1986) and contralateral (Jankswska et al., 1967) Ia terminals of the acute spinal cat injected DOPA.

In order to advance the body forward

during walking, an excessive soleus stretch must be removed in order to maintain a compliant interface between the body and the ground (Capaday and Stein, 1986; Stein and Capaday, 1988). Consequently, one of the goals of the rehabilitation for CNS patients is to inhibit the abnormal reflex segmentally in the spastic paretic subjects during gait (Fung and Barbeau, 1994). Under these circumstances cutaneous stimulation can be applied to restore some adaptive control of gait in subjects with spastic muscle activation (Fung and Barbeau, 1994; Yang et al, 1991).

Summary

There are two independent mechanisms to control the segmental reflex gain in humans during gait. They are presynaptic inhibition and homosynaptic depression. Through the mechanism of the presynaptic inhibition, the muscle spindle afferent feedback can be properly gated during eccentric phase of gait. The modulation of the presynaptic inhibition is reflected in the level of H-reflex at a constant EMG level. During the eccentric muscle activation presynaptic inhibition should increase to account for the lower amplitude level of H-reflex at a constant level of EMG. Homosynaptic depression is another mechanism responsible for regulating the effectiveness of the muscle spindle afferent feedback. Both the presynaptic inhibition and the monosynaptic depression are responsible for modulating reflex gain during gait initiation. Reflex modulation is influenced not only as a passive consequence of the alpha motor neuron excitation level, but also through supraspinal mechanisms. Spastic paretic patients show the impaired soleus H-reflex modulation either during the initial stance phase, or during the swing phase. This abnormal modulatory mechanism can partially and artificially be restored by the application of peripheral stimulus to the sole of the foot, provided that the segmental circuitry remains functional.

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