Biomechanical and neurophysiological mechanisms related to postural control and efficiency of movement: a review

Andreia Sousa (MSc)
Escola Superior da Tecnologia de Saúde do Instituto Politécnico do Porto,
Área Científica de Fisioterapia,
Centro de Estudos de Movimento e Actividade Humana,
Rua Valente Perfeito, 322 - 4400-330 Vila Nova de Gaia, PORTUGAL
Faculdade de Engenharia, Universidade do Porto,
Rua Dr. Roberto Frias, s/n, 4200-465 Porto, PORTUGAL
E-mail: asp@estsp.ipp.pt/andreia.asps@gmail.com

Augusta Silva (MSc)
Escola Superior da Tecnologia de Saúde do Instituto Politécnico do Porto,
Área Científica de Fisioterapia,
Centro de Estudos de Movimento e Actividade Humana,
Rua Valente Perfeito, 322 - 4400-330 Vila Nova de Gaia, PORTUGAL
E-mail: smaugusta@gmail.com

João Manuel R. S. Tavares (PhD)
Instituto de Engenharia Mecânica e Gestão Industrial,
Departamento de Engenharia Mecânica,
Faculdade de Engenharia, Universidade do Porto
Rua Dr. Roberto Frias, s/n, 4200-465 Porto, PORTUGAL
E-mail: tavares@fe.up.pt
(corresponding author)

Abstract

Understanding postural control requires considering various mechanisms underlying a person´s ability to stand, to walk and to interact with the environment safely and efficiently. The purpose of this paper is to summarise the functional relation between biomechanical and neurophysiological perspectives related to postural control in both standing and walking based on movement efficiency. Evidence related to the biomechanical and neurophysiological mechanisms is explored as well as the role of proprioceptive input on postural and movement control.

Key words: postural control, upright standing, gait, efficiency

1. Introduction

Postural control has been defined as the control of the body’s position in space for the purposes of balance and orientation (Horak, 2006; Massion, 1998; Shumway-Cook & Woolacott, 2000, 2007). Postural orientation involves the active control of body alignment and tonus in relation to gravity, base of support, environment and internal references (Horak, 2006; Kandel et al., 2000; Lundy-Ekman, 2002; Massion, 1998; Raine et al., 2009; Winter et al., 1990). Postural equilibrium involves the coordination of sensorimotor strategies to stabilise the body’s centre of mass (CoM) during both self-initiated and externally triggered disturbances in postural stability (Horak, 2006). Postural stability has been defined as the ability to control the CoM in relation to the base of support (Shumway-Cook & Woolacott, 2000, 2007). The weight of each component (orientation and stability) varies according to the task and the environment. Indeed, the postural control system adjusts its goal under different circumstances, such as longitudinal alignment of the whole body to maintain a steady, erect stance; remodeling of stance in preparation for a voluntary movement; shaping of the body for display purposes, as in dance; maintenance of balance, as on the gymnast's beam; or conservation of energy in a demanding task (Kandel et al., 2000; Shumway-Cook & Woolacott, 2007).

Biomechanically, a postural control position is achieved when the CoM is within the base of support and is aligned with the centre of pressure (CoP) (Winter, 1995). Any external or internal perturbation that changes the projection of the CoM to the limits of the base of support and the alignment between CoM and CoP may lead to postural challenge. The ability to maintain the body’s CoM within a specific boundary is dictated by the efficiency of the individual’s balance mechanisms (Raine et al., 2009) related to anticipatory postural adjustments (APA), triggered by feedforward mechanisms prior to the perturbation (Aruin & Latash, 1995; Belen'kii et al., 1967; Li & Aruin, 2007; Massion, 1992; Schepens & Drew, 2004), as well as to compensatory postural adjustments (CPA) that are initiated by sensory feedback signals (Alexandrov et al., 2005; Park et al., 2004). The process of generation of APA is likely to be affected by expected magnitude (Aruin & Latash, 1996; Bouisset et al., 2000) and direction (Aruin & Almeida, 1997; Santos & Aruin, 2008) of the perturbation, voluntary action associated to the perturbation (Arruin, 2003; Shiratori & Aruin, 2007), postural task and body configuration (Arruin, 2003; van der Fits et al., 1998). In conditions of high instability demands, the central nervous system (CNS) may suppress APA as a protection against their possible destabilising effects (Arruin et al., 1998). In fact, a relation between APA and CPA has been demonstrated (Santos et al., 2009), suggesting the existence of an optimal utilization of APA in postural control. The CPA response depends not only on the APA, but also on the direction and magnitude of the perturbation, the base of support dimension (Dimitrova et al., 2004; Henry et al., 1998; Horak & Nashner, 1986; Jones et al., 2008) and on the involvement in a secondary task (Bateni et al., 2004).

The main sensory systems involved in postural control are proprioception, the vestibular system and vision, and their afferent pathways within the CNS (Day & Cole, 2002; Shumway-Cook & Woolacott, 2007). Afferent and efferent pathways involve the spinal cord, the brain stem, the cerebellum, the midbrain, and the sensorimotor cortex. All of these contribute to the development of an internal representation of body posture that is continuously updated based on multisensory feedback and is used to forward commands to control body position in space (Massion, 1994; Mergner & Rosemeier, 1998). This provides a basis for all interactions involving perception and action with respect to the external world and is likely to be partly genetically determined and partly acquired through ongoing experiential learning. It is therefore adaptable and vulnerable, is dependent on the ongoing information that it receives (Meadows & Williams, 2009) and is related to human movement variability, allowing for adaptable functional behavior (Van Emmerik & Van Wegen, 2000).

The neural process involved on stability organisation and body orientation in space is necessary practically for all dynamic motor actions (Massion, 1998). Specifically, the control of balance during gait and while changing from one posture to another requires a complex control of a moving body CoM that is not within the base of foot support (Winter et al., 1993). In fact, human gait is influenced by a multifactorial interaction that results from neural and mechanical organisation, including musculoskeletal dynamics, a central pattern generator (CPG), based on a genetically determined spinal circuit, and peripheral and supraspinal inputs (Arechavaleta, 2008; Borghese et al., 1996; Horak & Macpherson, 1996; Mazzaro et al., 2005; McCollum et al., 1995; Segers, 2006). The CPG designs spinal networks than can generate patterns of rhythmic activity in the absence of external feedback or supraspinal control. However, these spinal networks are modulated by peripheral input and supraspinal control (Armstrong, 1986; Rossignol et al., 2006).

The present study aims to review the biomechanical and neurophysiological mechanisms related to postural control in both standing and walking based on movement efficiency. In the following sections, the neural mechanisms, the role of afferent information and biomechanical aspects will be considered to upright standing and human gait.

2. Postural and movement control

2.1 Neural mechanisms

Upright standing

The upright stance of the human is an unstable position (Peterka & Loughlin, 2004). Postural sway reflects noise and regulatory activity of the several control loops involved in maintenance of balance, which requires that the CoM never deviates beyond the support area. The control of the appropriate level of neuromuscular activity to produce rapid postural control strategies involves medial descending systems (Raine et al., 2009). The role of these systems is fundamental to the organisation of postural tone appropriately according to environment demands, gravity and base of support. The vestibular system action is related to postural tone adjustments to body weight support (Matsuyama & Drew, 2000). This system plays a major role in the antigravity function (Latash, 1998; Siegel & Sapru, 2011) as it is responsible, through lateral vestibulospinal tract, for the activation of ipsilateral extensor motor neurons and their associated gamma motor neurons (Latash, 1998; Rothwell, 1994; Siegel & Sapru, 2011). The reticular formation has an important role on APA production (Schepens & Drew, 2004) as it receives afferent input from all the sensory system and also from the pre-motor cortex and supplementary motor area (Brodal, 1981; Kiernan, 2005; Rothwell, 1994). The possible role of the cortex in postural control has been discussed and there is reference in literature to the pre-motor cortex influence in APA production (Massion, 1992) and to the supplementary motor area as potential focus of control for APA generation (Jacobs et al., 2009).

Human gait

Appropriate mechanisms for controlling muscle tone are essential to maintain stable postural and locomotor synergies in bipedal gait performance. The dependency between postural control and movement may be justified by the connection between the cortex and the reticular formation. In fact, muscle tone and the locomotor system can be controlled, in parallel, by a combined input to the brain stem of net inhibition from the basal ganglia, and net excitation from the motor cortex (Takakusaki et al., 2004). Specifically, an important neuronal circuit that allows the coexistence of postural adjustments and execution of movement is the cortico-ponto-cerebellar pathway, which allows the connection of the cortex with the nucleus of the brain stem and cerebellum (Ito, 2006). With this circuit the postural control can be organised ipsilateraly to the activated side with respect to the control of movement in the contralateral side. This relationship between movement and postural control through the activation of ventro-medial and dorso-lateral systems, as well as the importance of the coactivation mechanism between the two lower limbs (Dietz et al., 2002) to keep the body CoM over the feet (Dietz et al., 1992; Dietz et al., 1989), justify the study of mechanisms that occur in both sides of the body in relation to a unilateral movement like gait initiation as well as in relation to the gait cycle.

Basic structures involved in the control of locomotion and postural muscle tone are located in the midbrain (Takakusaki et al., 2004). Some circumscribed regions have been identified as relevant in activating and controlling the intensity of spinal locomotor CPG operation, maintaining equilibrium during locomotion, adapting limb movement to external conditions and coordinating locomotion to other motor acts (Armstrong, 1986; Jordan, 1986; Orlovsky, 1991). Among the main supraspinal centres involved are the sensorimotor cortex and the supplementary motor area (Kapreli et al., 2006; Mackay-Lyons, 2002; Miyai et al., 2002) the cerebellum, the basal ganglia (Garcia-Rill, 1986; Mackay-Lyons, 2002), the midbrain locomotor region (Kandel et al., 2000; Mileykovskiy et al., 2000) and the spinal cord (Dietz et al., 1992). The sensorimotor cortex is involved in the preparation for and execution of movement (Nelson, 1996). The cerebellum receives copies of CPG output to motoneurons via ventral spinocerebellar tract and spinoreticularcerebellar pathways, as well as information about the activity of the peripheral motor apparatus via the dorsal spinocerebellar tract (Orlovsky, 1991). Based on these, influences motoneurons indirectly via vestibulospinal, rubrospinal, reticulospinal and corticospinal pathways (Orlovsky, 1991). The cerebellum main role may be the timing of muscle activation, “fine-tuning” the output by adapting each step (Lansner & Ekeberg, 1994). Nevertheless, both the cerebellum and the basal ganglia seem to play an important role in timing of sequential muscle activation, with the basal ganglia operating at the level of planning, initiation, execution, and termination of motor programs as well motor learning (Mackay-Lyons, 2002; Wichmann & DeLong, 1996). The midbrain locomotor region activates “muscle tone excitatory system” and “rhythm generating system” (Takakusaki et al., 2004). Although not being relevant in gait, the motor cortex is involved in the modification of CPG activity in unstable surfaces or when gait needs a visual orientation (Mackay, 1999). The degree of supraspinal and spinal influences in movement generation is determined by context (Mackay-Lyons, 2002).

In Figure 1 are presented important structures involved in postural control as well the neural connection between postural control and movement control in functional tasks like walking and upright standing. The relevant role of afferent input, specifically the proprioceptive input, in postural and movement control justify the study of the role of Golgi tendon organ and muscle spindles and their afferences in standing and walking.

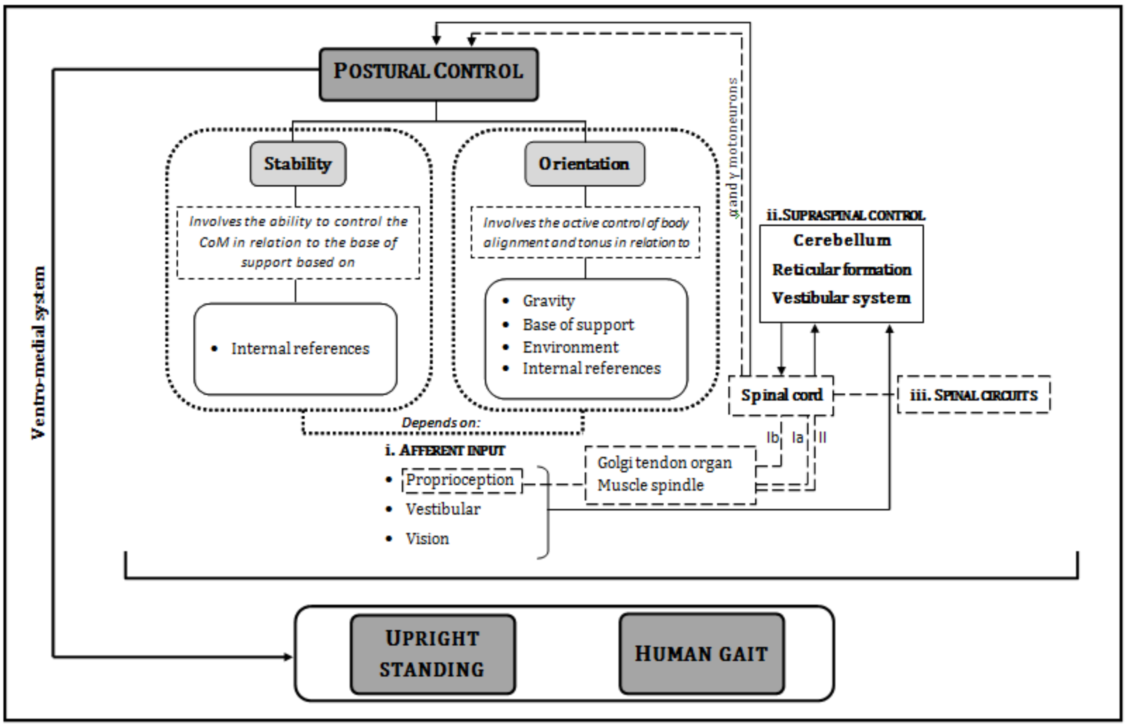

Figure 1: A conceptual schematic diagram illustrating the main structures involved postural control in both standing and walking.

2.2 The role of afferent information

Upright standing

The ability to reweight sensory information depending on the context is important to maintain stability when an individual moves from one context to another (Peterka, 2002). For instance, while vestibular information may not be a large contributor for the control of upright stance (Winter et al. 1998) and for triggering or coordinating muscle activation patterns associated to ankle strategy (Horak et al., 1990), it is likely to play a crucial role during moments of increased postural instability (Fitzpatrick and McCloskey 1994). As in normal conditions proprioceptive information assumes more relevance than other sources, in this paper focus has been given to the role of proprioceptive information.

It is well known that the mechanoreceptors (i.e. specialised sensorial receptors responsible for transduction of mechanic events into neural signs (Grigg, 1994)) accounting for proprioceptive information are primarily founded on muscles, tendons, ligaments and capsule (Hogersvorst & Brand, 1998; Jami, 1992; Johansson et al., 1991). Receptors located in the deeper skin tissue and fascia are traditionally associated with touch receptors, being categorised as additional sources (Edin & Johansson, 1995; Grigg, 1994; Macefield et al., 1990). Support has been given to the role of the Golgi tendon organs in providing afferent input from “gravity-dependent” receptors required to indicate the projection of the body’s CoM within the base of support (Dietz, 1996; Dietz, 1998; Dietz & Colombo, 1996; Dietz et al., 1992). Also, the small magnitudes of sway observed during quiet standing may be enough to alter muscle lengths, resulting in changes of Ia-afferent input onto the motoneuron pool of the lower limbs. Recent studies by (Loram et al., 2005b) have suggested this possibility, whereby muscle length changes in the gastrocnemius and soleus muscles during quiet standing have been detected within the range at which muscle spindles are sensitive to movement (Proske et al., 2000). Support has been given to the role of medium latency responses from group II during standing (Corna et al., 1996; Nardone et al., 1996; Schieppati et al., 1995). Indeed there is evidence that muscle spindle type II fibres play a more relevant role than group Ia fibres in the control of bipedal stance (Marchand-Pauvert et al., 2005) as only medium latency responses have a stabilising effect during perturbations of stance, and also because these fibres are more influenced by the “postural set” (Nardone et al., 1990). Findings obtained by Grasso et al, 1996, demonstrate the existence of crossed neural pathways fed by these fibres, which explains the bilateral electromyographic responses to unilateral perturbations during standing. This finding is supported by (Dietz, 1996), as this author argues that a complex bilateral coordination of leg muscle activation (mediated by a spinal mechanism (Dietz & Berger, 1984)) is needed for upright postural control during locomotion.

All the receptors mentioned above and the corresponding afferents input may allow a modulation of postural activity in relation to muscle length and tension variation, but only a combination of afferent inputs can provide the necessary information to control body equilibrium (Dietz, 1996). The role of proprioceptive information from ankle muscles has been highlighted in various studies (Fitzpatrick et al., 1994; Fitzpatrick et al., 1992a; Gatev et al., 1999; Loram et al., 2005a). Some authors go further, arguing that normal subjects can stand in a stable manner when receptors of the ankle muscles are the only source of information about postural sway (Fitzpatrick et al., 1994; Fitzpatrick et al., 1992a). The soleus and the gastrocnemius have traditionally been considered the source of muscle proprioceptive information signalling changes in body position (Fitzpatrick et al., 1992b; Loram & Lakie, 2002a; Loram et al., 2005b). These muscles act predominantly as active agonists and, because the foot is constrained on the ground, they prevent forward toppling of the body, whose centre of gravity is maintained in front of the ankle joint (Fitzpatrick et al., 1992a; Lakie et al., 2003; Loram & Lakie, 2002a; Loram et al., 2005a; Maki & Ostrovski, 1993). The problem with muscle spindles as position sensors is that they are able to generate impulses in response to muscle length changes as well as from fusimotor activity (Proske, 2006). According to (Di Giulio et al., 2009) the best proprioceptive information may come from un-modulated muscles crossing the joint in parallel with the active agonist. In fact earlier studies stated that, depending upon the stance conditions, muscle stretch does not necessarily result in a compensatory stretch reflex response but instead results in an antagonistic muscle activation (Gollhofer et al., 1989; Hansen et al., 1988). Based on this, it has been argued that reciprocal patterns of muscle activation are typically involved in postural control (Di Giulio et al., 2009; Latash, 1993). Neurophysiologically, reciprocal inhibition is mediated, at least in part, by a dysinaptic circuit in the spinal cord that is subject to several supraspinal as well as segmental modulator mechanisms (Jankowska, 1992) and varies according to the way in which antagonist muscles are activated (Lavoie et al., 1997). Synergies between antagonist muscles include simple patterns of reciprocal activation, co-contractions, and complex triphasic activation patterns (Lavoie et al., 1997). There is evidence that the strength of dysinaptic inhibition is reduced during co-contraction of antagonist muscles compared with reciprocal activation (Nielsen & Kagamihara, 1992). Another source of proprioceptive information may come from the cutaneous afferents of the feet as there is a large distribution of cutaneous receptors at various locations on the sole of the foot (Kennedy & Inglis, 2002). It has been suggested that this source of proprioceptive information contributes to both the coding and spatial representation of body posture during standing (Roll et al., 2002) and that the architecture and physiology of the foot appear to contribute to the task of bipedal postural control with great sensitivity (Wright et al., 2012).

Human gait

During gait afferent feedback adapts dynamically, through a reciprocal relationship, the response of the CPG to environmental requirements and assumes multiple roles in regulating the production of motor patterns, such as: (1) the production of detail in the temporal pattern of muscle activation sequence (Ivanenko et al., 2006; Pearson, 1993), (2) the reinforce of ongoing motor activity, particularly those involving load-bearing muscles, such as the extensor muscles during the stance phase of gait (Pearson, 1993; Sinkjær et al., 2000; Stephens & Yang, 1996), and (3) the control of transition from one phase of movement to another (Lacquaniti et al., 1999; Pearson, 1993). Swing is initiated when the leg is extended (stretching the flexor muscles) and unloaded (reduced force in extensor muscles sensed by the Golgi tendon organ of the extensor muscles) (Zehr & Duysens, 2004). Consequently gait cycles depend on the afferent input from peripheral receptors as the muscle force production at a given level of motor unit recruitment can change according to length (velocity) and tension variations (Frigo et al., 1996)

The monosynaptic excitation of spinal motoneurons from the large diameter group Ia afferent fibres related to short latency response (Matthews, 1991) has been demonstrated when an expected stretch of the ankle extensors is imposed during gait (Sinkjaer et al., 1996; Yang et al., 1991). Also a phasic modulation of Ia input has been demonstrated by changes in the magnitude of H-reflex over the course of the gait cycle, with the greatest attenuation occurring during flexion (Schneider et al., 2000; Yang & Whelan, 1993). This modulation is consistent with the fact that the maximum soleus length occurs during foot off, when maximum plantar flexion of the foot occurs, which is coincident with its maximum force production (Orendurff et al., 2005). The modulation of the H-reflex is a reflection of: (1) the background excitability of the motoneuron pool, (2) the modulation associated to the activation of the antagonist muscle, and (3) presynaptic inhibition of the primary afferents (Yang & Whelan, 1993) that seems to be related partially to Ia afferents from the hip and knee extensor muscles (Brooke et al., 1997). Medium latency response from group II has been demonstrated during gait (Dietz et al., 1985) and some authors argue that this group is more important to feedback in the stance phase than group Ia (Grey et al., 2001; Grey et al., 2002; Nielsen & Sinkjaer, 2002; Sinkjær et al., 2000). Earlier studies have suggested that strong central effects of group II muscle afferents are mediated via a complex neural pathway influenced by supraspinal input and peripheral input during walking (Dietz et al., 1987; Yang et al., 1991). Specifically, there is evidence for the role of vestibulo- and reticulo-spinal pathway (Davies & Edgley, 1994) which supports the hypothesize that the facilitation of the relevant lumbar propriospinal neurons by descending tracts neurons would be stronger over group II during maintenance of posture than during voluntary contractions (Marchand-Pauvert et al., 2005). Also, the role of group Ib load-sensitive afferences related to medium latency response has been reported to contribute to the regulation stance phase of gait (Stephens & Yang, 1999) associated to a disynaptic Ib reflex-reversal (Stephens & Yang, 1996). Findings obtained in (Grey et al., 2007) suggest that tendon organ feedback via an excitatory group Ib pathway contributes to the late stance phase enhancement of the soleus muscle activity. The combination of the different afferent inputs plays an important role on gait dynamics related to the ipsilateral limb but also on the contralateral limb, as it has been demonstrated that unilateral leg displacement during gait evokes a bilateral response pattern, with a similar onset on both sides (Dietz & Berger, 1984). From a functional point of view, this interlimb coordination is necessary to keep the body's CoM over the feet (Dietz, 1996).

Figure 2 summarizes the role of proprioceptive receptors and respective afferences in standing and walking. Important networks related to integration of proprioceptive information (cerebellum) and to the modulation of afferent information at spinal cord (reticulospinal and vestibulospinal tracts) are represented.

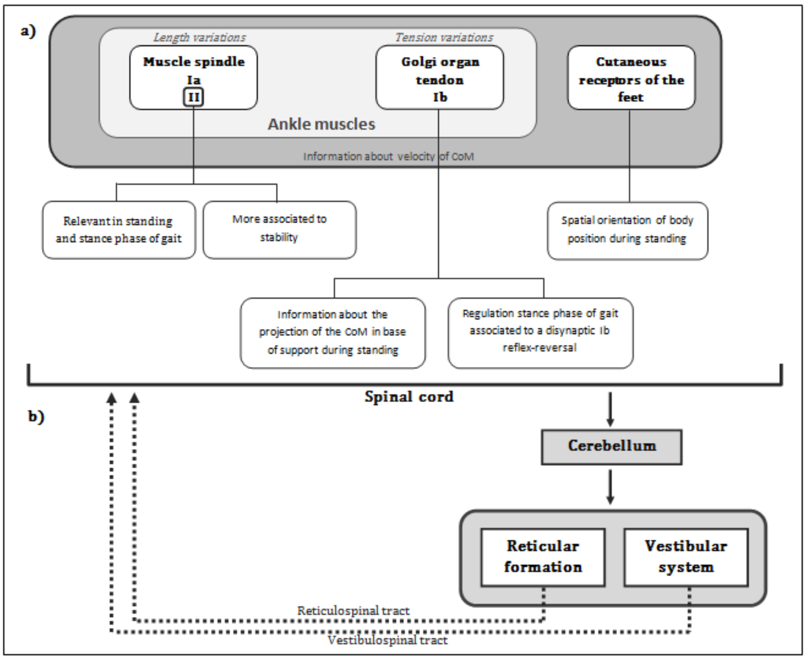

Figure 2: Representation of the most important proprioceptive receptors and afferences and their role in standing and stance phase of gait (a). There are also represented important networks related to proprioceptive information (b). In this part of illustration (b), dotted lines represent efferent pathways and solid lines represent afferent pathways.

2.3 Biomechanical aspects

Upright standing

Upright stance is associated with small deviations from an upright body position, which results in a gravity-induced torque acting on the body, causing it to accelerate further away from the upright position. Corrective torque must be generated to counter the destabilizing torque due to gravity. This process of continuous small body deviations countered by corrective torques creates a pattern known as spontaneous sway. The mechanisms underlying spontaneous sway are not fully understood, and controversy remains regarding the organisation of sensory and motor systems contributing to spontaneous sway. Numerous authors have suggested that active feedback control mechanisms contribute to the maintenance of upright stance (Fitzpatrick et al., 1996; Johanson & Magnusson, 1991; Peterka & Loughlin, 2004; Peterka & Benolken, 1995; van der Kooij et al., 2001). Recent studies have shown that a model based primarily on a feedback mechanism with a 150- to 200-ms time delay can account for postural control during a broad variety of perturbations (Peterka, 2002; Peterka & Loughlin, 2004; Peterka & Benolken, 1995) and can yield a spontaneous sway pattern that resembles normal (Peterka, 2000) or pathological spontaneous sway (Parkinson’s disease; (Maurer et al., 2003)). However, the relevance of feedback mechanisms for postural control is still debated. Some authors concluded from their experiments that corrective torque originating from feedback control is insufficient for stabilizing the body (Fitzpatrick et al., 1996). Others suggested additional sources for corrective torque, like prediction (Morasso et al., 1999; van der Kooij et al., 2001), or have proposed more complex concepts (Baratto et al., 2002; Collins & De Luca, 1993; Loram & Lakie, 2002b). Postural sway has been viewed as a result of a correlated random-walk process (Collins & De Luca, 1993), a result of computational noise (Kiemel et al., 2002), and/or a moving reference point (Zatsiorsky & Duarte, 1999). The possible importance of postural sway as a reflection of a hypothetical search process within the system of postural stabilization has been emphasised (Mochizuki et al., 2006; Riley et al., 1997).

From a functional point of view, the control of human upright posture stability is commonly viewed as a continuous stabilization process of a multilink inverted pendulum, where the main controlled parameter is the CoM position within the limits of the supporting base (Maurer & Peterka, 2005). This aspect as been described as biomechanical constraints that determines patterns of postural coordination (Buchanan & Horak, 2003). In stance, the limits of stability - that is, the area over which individuals can move their CoM and maintain equilibrium without changing the base of support - are shaped like a cone (McCollum & Leen, 1989a). Thus, equilibrium is not a particular position but a space determined by the size of the support base and the limitations on joint range, muscle strength and sensory information available to detect limits. The CNS has an internal representation of this cone of stability that it uses to determine how to move to maintain equilibrium (Horak, 2006). Gatev et al., 1999, reported a significant correlation between spontaneous body sway and the activity of the gastrocnemius muscle. They also discovered that gastrocnemius activity preceded temporally CoM displacement, suggesting a central program of control of the ankle joint stiffness working to predict the loading pattern. More recent studies proposed that the actual postural control system during quiet standing adopts a control strategy that relies notably on velocity information of CoM and that such a controller can modulate muscle activity in an anticipatory manner without using feedforward mechanisms (Masani et al., 2003). According to this view, velocity feedback can play a significant role in anticipating body position changes because it carries information about the subsequent state of the body, i.e., a change in CoM velocity indicates the direction and intensity with which the current CoM displacement will be changed in the following time instant. It has been hypothesised that the integration of proprioceptive and plantar cutaneous sensations would play a significant role in the velocity feedback mechanism (Masani et al., 2003). Another biomechanical constraint is related to frequency of postural sway (Nashner et al., 1989), as when postural sway is lower than 0.5 Hz the body can be compared to a simple inverted pendulum (McCollum & Leen, 1989b), and when it is higher than this value the body can be compared to a double inverted pendulum with the fulcrum at the hip level (Yang et al., 1990).

Human gait

The coordination between posture and movement involves the dynamic control of the CoM in the base of support (Stapley et al., 1999). Consequently, to access the simplified concept of locomotion it is necessary to consider the behavior of the CoM during gait cycles. The trajectory described by the CoM in the plan of progression is a sinusoidal curve that moves vertically twice during one cycle and laterally in the horizontal plan and that is similar in form to that found in the vertical displacement (Gard et al., 2004; Norkin & Levangie, 1992). Peak-to-peak amplitude is described as being about 4-5 cm for adults at freely chosen speed and has been used to estimate exchanges of mechanical energy, efficiency, work, and to describe the symmetry as an indicator of the quality of gait (for more information see (Gard et al., 2004)).

The human gait results from a complex interaction of muscle forces, joint movements and neural commands. Variables including electromyographic activity, muscle torque, ground reaction forces, kinematics and metabolic-energy costs have been measured and quantified. This data set requires an interpretation and organisation of the fundamental principles that elucidate the mechanisms of gait. Several models have been suggested to describe human gait mechanisms (Cavagna & Kaneko, 1977; Cavagna & Margaria, 1966; Donelan et al., 2002b; Kuo et al., 2005; Kuo et al., 2007; Saunders et al., 1953; Waters & Mulroy, 1999). The six determinants of gait theory (Saunders et al., 1953), based on the premise that vertical and horizontal CoM displacements are energetically costly, proposes a set of kinematic features that help to reduce CoM displacement. However, there is evidence that some determinants have a non-significant role on the CoM vertical displacement and that there is higher metabolic expenditure when subjects voluntary reduce vertical displacement of CoM (for review see (Kuo et al., 2007)). The inverted pendulum model proposes that most of the work during gait is performed by a passive mechanism of exchange of gravitational potential and kinetic energies (60-70%)(Cavagna et al., 1977; Griffin et al., 2003). However this model cannot reproduce the existence of two peaks in vertical ground reaction force (Pandy, 2003; Zajac et al., 2003) and does not account for the costs which are not considered responsible for work, like isometric force stabilisation and body weight support (Kuo et al., 2005). The difference in the percentage of energy recovery in relation to an ideal inverted pendulum has been related mostly to double support phase (McGeer, 1990). Indeed, it has been demonstrated a low percentage of energy recovery in the double support phase (Geyer et al., 2006) related to the interruption of the energy-conserving motion of single support by an inelastic collision of the swing leg with the ground, leading to changes in velocities of the legs and the CoM (Kuo et al., 2007). This energy loss can be reduced by 75% through the application of a propulsion impulse in the trailing leg immediately before collision of the leading leg (Kuo, 2002). Simulations suggest that the ankle plantar flexor (soleus, gastrocnemius) and the uni- and bi-articular hip extensors (gluteus maximus, hamstrings) dominate work output over the gait cycle (Neptune et al., 2004). These muscles, being active in the late stance and in the beginning of stance, are therefore restoring energy to the body near double-support (Zajac et al., 2003).

Ankle plantar flexors are the primary contributors for forward progression and vertical support (Kepple et al., 1997), before midstance they hinder progression (Neptune et al., 2001) and during midstance they maintain body support and the forward motion of the trunk and leg, which is consistent with inverted-pendulum-like ballistic walking as the synergy of these muscles in this subphase occurs with minimal metabolic energy expenditure, as expected in ballistic-like walking (Zajac et al., 2003). Biarticular hip extensors generate forward acceleration during the first half of stance, while uniarticular quadriceps muscles and the uniarticular hip extensors decelerate the body mass centre and provide body support (Liu et al., 2006; Neptune et al., 2004). The biarticular quadriceps muscle is a significant contributor to forward progression in late stance (Neptune et al., 2004).

According to Donelan, 2004, lateral stabilisation exacts a modest metabolic cost as walking requires active lateral stabilisation. It has been demonstrated that the gluteus medius, although acting primarily outside the sagittal plane in walking, contributes to support and slows progression (less than the other muscles) in the first half of stance and contributes to support in the second half (Liu et al., 2006). Also, is has been demonstrated that the body lateral motion is partially stabilised via medio-lateral foot placement (Donelan, 2004; Kuo, 1999).

3. Movement efficiency

The relationship between muscle activity and whole body mechanics it too variable and complex to allow direct control of the later without an intermediate kinematic representation (Lacquaniti et al., 1999). There is evidence that supports the idea that global kinematic gait is controlled (Ivanenko et al., 2004). Kinematics is relatively invariant in various modes of locomotion, while the electromyographic activity patterns to produce the required kinematic patterns can vary considerably (Grasso et al., 1998; Ivanenko et al., 2004). These findings suggest that neural circuitry can somehow specify limbs kinematics and the appropriate muscle synergies would be determined in a subordinate and flexible manner to adapt to the current mechanical constraints (Lacquaniti et al., 1999; Lacquaniti et al., 2002). The basic biomechanical control signal may exert its action through an appropriate model of inverse dynamics and feedback device that determines the muscle torque necessary to achieve kinematic patterns (Ivanenko et al., 2004). The significance of muscle redundancy would then be to allow the same movement to be carried out by means of different combinations of muscle activity under different environmental circumstances, for instance, to cope with fatigue or changes in load (Lacquaniti et al., 1999).

The major function of muscles in gait is to generate and absorb energy; such function is largely ignored in neurophysiological research (Winter & Eng, 1995). The body has the capacity of transferring energy between segments across the joint centres and can store and recover energy in the passive elastic tissues in the tendon and muscles. However, this last energy conserving mechanism is quite small in walking (Winter & Eng, 1995). The CNS has learnt how to create motor patterns to conserve much of the energy that was generated earlier in the gait cycle. It has been estimated that of the total energy changes of all body segments over the gait cycle only 33% are caused by active muscle generation and absorption, while 67% are due to the passive transfers between segments (Pierrynowski et al., 1980). Considering this it is important to quantify the movement also on the criterion of efficiency (Fetters & Holt, 1990; Sparrow & Newell, 1998).

In biomechanical and physiological research, efficiency of movement is normally defined as the ratio of the mechanical work performed and the metabolic cost of performing the work (Stainsby et al., 1980). Typically an efficiency formula will take the form:

$$Efficiency\ (\%) = \frac{Mechanical\ work}{Energy\ work} \times 100$$

Energy expenditure during walking can be characterised through mechanical energy estimations (Cavagna et al., 1963; Saibene & Minetti, 2003; Willems et al., 1995) or metabolic energy measurements (Waters, 1999). Mechanical energy is generally estimated by one of three approaches: (1) analysis of energy changes of the CoM relative to the surroundings (external work) and of the body segments relative to the CoM (internal work) (Cavagna & Margaria, 1966; Cavagna et al., 1963; Willems et al., 1995); (2) analysis of the energy changes of moving body segments (sum of segmental energies) or (3) measurement of muscle power around the joints (net joint work) (Winter, 2005). In all mechanical energy estimations the actual amount of work performed is underestimated as additional metabolic work resulting from isometric muscle contractions or antagonist co-contractions is not taken into account (Fetters & Holt, 1990; Winter, 2005). This problem is overcome when assessing metabolic energy i.e. measuring oxygen consumption during walking (Fetters & Holt, 1990; Vandewalle, 2004). The relation between metabolic cost and the mechanical work performed by stance limb muscles to lift and accelerate the CoM during walking has been already demonstrated (Donelan et al., 2001; Donelan et al., 2002a) and has been considered a valid predictor of walking performance (Anderson & Pandy, 2001). Metabolic energy expenditure can be accessed through indirect calorimetry, where oxygen consumption and/or carbon dioxide production is measured and converted into energy expenditure using formulae (Cunningham, 1990; Garby & Astrup, 1987) which have been reported as a valid method (Levine, 2005). Mechanical and metabolic energy analyses allow monitoring how the CNS takes advantage of energy conserving mechanisms in order to achieve a more efficient movement.

Summary/Concluding remarks

Postural control has been vastly explored in the scientific community. However, the complexity of the interrelations between neural and mechanical aspects and environment leads to the need of studying postural control in a holistic way. In addition, the study of postural control needs to reflect the dynamic inter-relation of the different components of human movement on the basis of movement efficiency. The adaptability, vulnerability and continuous dependency of afferent information on the postural control system turns this area a focus of clinical interest. Considering that the postural control system has the capacity of reorganization for higher movement performance, it is important to understand in detail the static and dynamic postural control mechanisms and strategies and how these mechanisms influence other systems and are influenced by changes in afferent and efferent information.

References

Alexandrov A, Frolov A, Horak F, Carlson-Kuhta P, Park S. 2005. Feedback equilibrium control during human standing. Biological Cybernetics 93:309-322.

Anderson FC, Pandy MG. 2001. Dynamic optimization of human walking. Journal of Biomechanical Engineering 123:5 381-390.

Arechavaleta GL, J; Hicheur, H; Berthoz, A. 2008. An Optimal Principle Governing Human Walking. IEEE Transactions on Robotics 24:1.

Armstrong M. 1986. Supraspinal contributions to the initiation and control of locomotion on the cat. Progress in Neurobiology 26:273-361.

Arruin A. 2003. The effect of changes in the body configuration on anticipatory postural adjustments. Motor Control 7:3 264-277.

Arruin A, Forrest W, Latash M. 1998. Anticipatory postural adjustments in conditions of postural instability. Electroencephalography and Clinical Neurophysiology 109:4 350-359.

Aruin A, Almeida G. 1997. A coactivation strategy in anticipatory postural adjustment in persons with Down syndrome. Motor Control 2:178-197.

Aruin A, Latash M. 1995. The role of motor action in anticipatory postural adjustments studied with self-induced and externally triggered perturbations. Experimental Brain Research 106:291-300.

Aruin A, Latash M. 1996. Anticipatory postural adjustments during self-initiated perturbations of different magnitude triggered by standard motor action. Electroencephalography and Clinical Neurophysiology 101:6 497-503.

Baratto L, Morasso PG, Re C, Spada G. 2002. A new look at posturographic analysis in the clinical context: sway-density versus other parameterization techniques. Motor Control 6:3 246-270.

Bateni H, Zecevi A, McIlroy W, Maki B. 2004. Resolving conflicts in task demands during balance recovery: does holding an object inhibit compensatory grasping? Experimental Brain Research 157:1 49-58.

Belen'kii V, Gurfinkel' V, Pal'tsev Y. 1967. Elements of control of voluntary movements. Biofizika 10:135-141.

Borghese N, Bianchi L, Lacquaniti F. 1996. Kinematic determinants of human locomotion. Journal of Physiology 494:3 863-869.

Bouisset S, Richardson J, Zattara M. 2000. Are amplitude and duration of anticipatory postural adjustments identically scaled to focal movements parameters in humans? Neuroscience Letters 278:3 153-156.

Brodal A. 1981. Neurological anatomy in relation to clinical medicine. 3 ed. New York: Oxford University Press.

Brooke JD, Cheng J, Collins DF, McIlroy WE, Misiaszek JE, Staines WR. 1997. Sensori-sensory afferent conditioning with leg movement: gain control in spinal reflex and ascending paths. Progress in Neurobiology 51:4 393-421.

Buchanan J, Horak F. 2003. Voluntary control of postural equilibrium patterns. Behavioral and Brain Sciences 143:121-140.

Cavagna G, Kaneko M. 1977. Mechanical work and efficiency in level walking and running. Journal of Physiology 268:2 467-481.

Cavagna G, Margaria R. 1966. Mechanics of walking. Journal of Applied Physiology 21:1 271-278.

Cavagna G, Saibene F, Margaria R. 1963. External work in walking. Journal of Applied Physiology 18:1-9.

Cavagna GA, Heglund NC, Taylor CR. 1977. Mechanical work in terrestrial locomotion: two basic mechanisms for minimizing energy expenditure. American Journal of Physiology - Regulatory, Integrative and Comparative Physiology 233:5 R243-R261.

Collins J, De Luca C. 1993. Open-loop and closed-loop control of posture: a random-walk analysis of center-of-pressure trajectories. Experimental Brain Research 95:2 308-318.

Corna S, Galante M, Grasso M, Nardone A, Schieppati M. 1996. Unilateral displacement of lower limb evokes bilateral EMG responses in leg and foot muscles in standing humans. Experimental Brain Research 109:1 83-91.

Cunningham J. 1990. Calculation of energy expenditure from indirecr calorimetry: assessment of the Weir equation. Nutrition 6:3 222-223.

Davies HE, Edgley SA. 1994. Inputs to group II-activated midlumbar interneurones from descending motor pathways in the cat. The Journal of Physiology 479:Pt 3 463-473.

Day B, Cole J. 2002. Vestibular-evoked postural responses in the absence of somatosensory information. Brain 125:9 2081-2088.

Di Giulio I, Maganaris C, Baltzopoulos V, Loram I. 2009. The proprioceptive and agonist roles of gastrocnemius, soleus and tibialis anterior muscles in maintaining human upright posture. Journal of Physiology 587:10 2399-2416.

Dietz V. 1996. Interaction between central programs and afferent input in the control of posture and locomotion. Journal of Biomechanics 29:7 841-844.

Dietz V. 1998. Evidence for a load receptor contribution to the control of posture and locomotion. Neuroscience & Biobehavioral Reviews 22:4 495-499.

Dietz V, Berger W. 1984. Interlimb coordination of posture in patients with spastic paresis. Brain 107:3 965-978.

Dietz V, Colombo G. 1996. Effects of body immersion on postural adjustments to voluntary arm movements in humans: role of load receptor input. The Journal of Physiology 497:Pt 3 849-856.

Dietz V, Gollhofer A, Kleiber M, Trippel M. 1992. Regulation of bipedal stance: dependency on “load” receptors. Experimental Brain Research 89:1 229-231.

Dietz V, Horstmann GA, Berger W. 1989. Interlimb coordination of leg-muscle activation during perturbation of stance in humans. Journal of Neurophysiology 62:3 680-693.

Dietz V, Muller R, Colombo G. 2002. Locomotor activity in spinal man: significance of afferent input from joint and load receptors. Brain 125:2626-2634.

Dietz V, Quintern J, Berger W. 1985. Afferent control of human stance and gait: evidence for blocking of group I afferents during gait. Experimental Brain Research 61:1 153-163.

Dietz V, Quintern J, Sillem M. 1987. Stumbling reactions in man: significance of proprioceptive and pre-programmed mechanisms. The Journal of Physiology 386:1 149-163.

Dimitrova D, Horak F, Nutt J. 2004. Postural muscle responses to multidirectional translations in patients with Parkinson's disease. Journal of Neurophysiology 91:1 489-501.

Donelan J, Kram R, Kuo A. 2001. Mechanical and metabolic determinants of the preferred step width in human walking. Proceedings of the Royal Society of London Series B-Biological Sciences 268:1985-1992.

Donelan J, Kram R, Kuo A. 2002a. Mechanical work for step-to-step transition is a major determinant of the metabolic cost. Journal of Experimental Biology 205:3717-3727.

Donelan J, Kram R, Kuo A. 2002b. A simultaneous positive and negative external mechanical work in human walking. Journal of Biomechanics 35:1 117-124.

Donelan JS, D; Kram, R; Kuo, A. 2004. Mechanical and metabolic requirements for active lateral stabilization in human walking. Journal of Biomechanics 37:827-835.

Edin B, Johansson N. 1995. Skin strain patterns provide kinaesthetic information to the human central nervous system. Journal of Physiology 487:243-251.

Fetters L, Holt K. 1990. Efficiency of movement: biomechanical and metabolic aspects. Pediatric Physical Therapy155-159.

Fitzpatrick R, Burke D, Gandevia SC. 1996. Loop gain of reflexes controlling human standing measured with the use of postural and vestibular disturbances. Journal of Neurophysiology 76:6 3994-4008.

Fitzpatrick R, Douglas K, McCloskey D. 1994. Stable human standing with lower-limb muscle afferents providing the only sensory input. Journal of Physiology 2536:395-403.

Fitzpatrick R, Gorman R, Burke D, Gandevia S. 1992a. Postural proprioceptive reflexes in standing human subjects: bandwidth of response and transmission characteristics. The Journal of Physiology 458:1 69-83.

Fitzpatrick R, Taylor J, McCloskey D. 1992b. Ankle stiffness of standing humans in response to imperceptible perturbation: reflex and task-dependent components. Journal of Physiology 454:533-547.

Frigo C, Nielsen J, Crenna P. 1996. Modelling the triceps surae muscle-tendon complex for the estimation of length changes during walking. Journal of Electromyography and Kinesiology 6:3 191-203.

Garby L, Astrup A. 1987. The relationship between the respiratory quotient and the energy equivalent of oxygen during simultaneous glucose and lipid oxidation and lipogenesis. Acta Physiologica Scandinavica 129:443-444.

Garcia-Rill E. 1986. The basal ganglia and the locomotor regions. Brain Research Reviews 11:1 47-63.

Gard S, Miff S, Kuo A. 2004. A comparison of kinematic and kinetic methods for computing the vertical motion of the body central mass during walking. Human Movement Science 22:597-610.

Gatev P, Thomas S, Kepple T, Hallet M. 1999. Feedforward ankle strategy of balance during quiet stance in adults. Journal of Physiology 514:3 915-928.

Geyer H, Seyfarth A, Blickhan R. 2006. Compliant leg behaviour explains basic dynamics of walking and running. Proceedings of the Royal Society B: Biological Sciences 273:1603 2861-2867.

Gollhofer A, Horstmann GA, Berger W, Dietz V. 1989. Compensation of translational and rotational perturbations in human posture: Stabilization of the centre of gravity. Neuroscience Letters 105:1–2 73-78.

Grasso R, Bianchi L, Lacquaniti F. 1998. Motor patterns for human gait: backward versus forward locomotion. Journal of Neurophysiology 80:4 1868-1885.

Grey MJ, Ladouceur M, Andersen JB, Nielsen JB, Sinkjær T. 2001. Group II muscle afferents probably contribute to the medium latency soleus stretch reflex during walking in humans. The Journal of Physiology 534:3 925-933.

Grey MJ, Nielsen JB, Mazzaro N, Sinkjaer T. 2007. Positive force feedback in human walking. Journal of Physiology 581:1 99-105.

Grey MJ, Van Doornik J, Sinkjær T. 2002. Plantar flexor stretch reflex responses to whole body loading/unloading during human walking. European Journal of Neuroscience 16:10 2001-2007.

Griffin T, Roberts T, Kam R. 2003. Metabolic of generation muscular force in human walking: insights from load-carring and speed experiments. Journal of Applied Physiology 95:1 172-183.

Grigg P. 1994. Peripheral neural mechanisms in proprioception. Journal of Sports and Rehabilitation 3:2-17.

Hansen PD, Woollacott MH, Debu B. 1988. Postural responses to changing task conditions. Experimental Brain Research 73:3 627-636.

Henry S, Fung J, Horak F. 1998. EMG responses to maintain stance during multidirectional surface translations. Journal of Neurophysiology 80:4 1939-1950.

Hogersvorst T, Brand R. 1998. Mechanoreceptors in joint function. Journal of Bone and Joint Surgery 80:1365-1378.

Horak B, Macpherson M. 1996. Postural orientation and equilibrium Handbook of Physiology. Exercise. Regulation and Integration of Multiple Systems (pp. 256-292). Washington DC: American Physiology Society.

Horak F. 2006. Mechanistic and physiological aspects. Postural orientation and equilibrium: what do we need to know about neural control of balance to prevent falls? Age and Ageing 35-S2:ii7-ii11.

Horak F, Nashner L. 1986. Central programming of postural movements: adaptation to altered support-surface configurations. Journal of Neurophysiology 55:6 1369-1381.

Horak F, Nashner L, Diener H. 1990. Postural strategies associated with somatosensory and vestibular loss. Experimental Brain Research 82:1 167-177.

Ito M. 2006. Cerebellar circuitry as a neuronal machine. Progress in Neurobiology 78:3–5 272-303.

Ivanenko Y, Poppele R, Macellari V, Lacquaniti F. 2004. Five basic muscle activation patterns account for muscle activity during human locomotion. Journal of Physiology 556:1 267-282.

Ivanenko YP, Poppele RE, Lacquaniti F. 2006. Spinal cord maps of spatiotemporal alpha-motoneuron activation in humans walking at different speeds. Journal of Neurophysiology 95:2 602-618.

Jacobs JV, Lou JS, Kraakevik JA, Horak FB. 2009. The supplementary motor area contributes to the timing of the anticipatory postural adjustment during step initiation in participants with and without Parkinson's disease. Neuroscience 164:2 877-885.

Jami L. 1992. Golgi tendon organs in mammalian skeletal muscle: functional properties and central actions. Physiology Review 72:623-666.

Jankowska E. 1992. Interneuronal relay in spinal pathways from proprioceptors. Progress in Neurobiology 38:4 335-378.

Johanson R, Magnusson M. 1991. Human postural dynamics. Critical Review in Biomedical Engineering 18:6 413-437.

Johansson H, Sjolander P, Sojka P. 1991. A sensory role for the crusiate ligaments. Clinical Orthopaedics 80:1365-1378.

Jones S, Henry S, Raasch C, Hitt J, Bunn J. 2008. Responses to multi-directional surface translations involve redistribution of proximal versus distal strategies to maintain upright posture Experimental Brain Research 187:3 407-417.

Jordan M. 1986. Initiation of locomotion in mammals. Annals of the New York Academy of Sciences 860:83-93.

Kandel E, Schwartz J, Jessell T. 2000. Principals of neural science. 4th ed. New York: McGraw-Hill.

Kapreli E, Athanasopoulos S, Papathanasiou M, Van Hecke P, Strimpakos N, Gouliamos A, et al. 2006. Lateralization of brain activity during lower limb joints movement. An fMRI study. NeuroImage 32:4 1709-1721.

Kennedy P, Inglis J. 2002. Distribution and behaviour of glabrous cutaneous receptors in the human foot sole. The Journal of Physiology 538:3 995-1002.

Kepple T, Siegel K, Stanhope S. 1997. Relative contributions of the lower extremity joint moments to forward progression and support during stance. Gait & Posture 6:1-8.

Kiemel T, Oie KS, Jeka JJ. 2002. Multisensory fusion and the stochastic structure of postural sway. Biological Cybernetics 87:4 262-277.

Kiernan J. 2005. The human nervous system. An anatomical viewpoint. 8 ed. London: Lippincott Williams & Wilkins.

Kuo A. 1999. Stabilization of lateral motion in passive dynamic walking. International Journal of Robotic Research 18:9 917-930.

Kuo A. 2002. Energetics of actively powered locomotion using the simplest walking model. Journal of Biomechanical Engineering 124:1 113-120.

Kuo A, Donelan M, Ruina A. 2005. Energetic consequences of walking like an inverted pendulum: step to step transitions. Exercise Sports Science Review 33:2 88-97.

Kuo A, Doneland M, Ruina A. 2007. The six determinants of gait in the inverted pendulum analogy: a dynamic walking perspective. Human Movement Science 26:4 617-656.

Lacquaniti F, Grasso R, Zago M. 1999. Mottor patterns in walking. News in Physiology Sciences 14:4 168-174.

Lacquaniti F, Ivanenko Y, Zago M. 2002. Kinematic control of walking. Archives Italian Biology 140:4 263-272.

Lakie M, Caplan N, Loram I. 2003. Human balancing of an inverted pendulum with a compliant linkage: neural control by anticipatory intermittent bias. Journal of Physiology 551:1 357-370.

Lansner A, Ekeberg Ö. 1994. Neuronal network models of motor generation and control. Current Opinion in Neurobiology 4:6 903-908.

Latash M. 1993. Control of Human Movement. Human Kinetics.

Latash M. 1998. Neurophysiological basis of movement. USA: Human Kinetics.

Lavoie B, Devanne H, Capaday C. 1997. Differential control of reciprocal inhibition during walking versus postural and voluntary motor tasks in humans. Journal of Neurophysiology 78:1 429-438.

Levine J. 2005. Measurement of energy expenditure. Public Health Nutrition 8:7A 1123-1132.

Li X, Aruin A. 2007. The effect of short-term changes in the body mass on anticipatory postural adjustments. Experimental Brain Research 181:333-346.

Liu M, Anderson F, Pandy M, Delp S. 2006. Muscles that support the body also modulate forward progression during walking. Journal of Biomechanics 39:14 2623-2630.

Loram I, Lakie M. 2002a. Direct measurement of human ankle stiffness during quiet standing: the intrinsic mechanical stiffness is insufficient for stability. Journal of Physiology 545:3 1041-1053.

Loram I, Lakie M. 2002b. Human balancing of an inverted pendulum: position control by small, ballistic-like, throw and catch movements. Journal of Physiology 540:3 1111-1124.

Loram I, Maganaris C, Lakie M. 2005a. Active, non-spring-like muscle movements in human postural sway: how might paradoxical changes in muscle length be produced? Journal of Physiology 564:1 283-293.

Loram I, Maganaris C, Lakie M. 2005b. Human postural sway results from frequent, ballistic bias impulses by soleus and gastrocnemius. Journal of Physiology 564:1 295-311.

Lundy-Ekman L. 2002. Neuroscience: fundamentals for rehabilitation. 2nd ed. Philadelphia.

Macefield G, Gandevia S, Burke D. 1990. Perceptual responses to microstimulation of single afferents innervating joints, muscles and skin of the human hand. Journal of Physiology 429:113-129.

Mackay-Lyons M. 2002. Central pattern generation of locomotion: a review of the evidence. Physical Therapy 82:69-83.

Maki B, Ostrovski G. 1993. Scaling of postural responses to transient and continuous perturbations. Gait & Posture 1:2 93-104.

Marchand-Pauvert V, Nicolas G, Marque P, Iglesias C, Pierrot-Deseilligny E. 2005. Increase in group II excitation from ankle muscles to thigh motoneurones during human standing. The Journal of Physiology 566:1 257-271.

Masani K, Popovic MR, Nakazawa K, Kouzaki M, Nozaki D. 2003. Importance of body sway velocity information in controlling ankle extensor activities during quiet stance. Journal of Neurophysiology 90:6 3774-3782.

Massion J. 1992. Movement, posture and equilibrium: interaction and coordination. Progress in Neurobiology 38:35-56.

Massion J. 1994. Postural control system. Current Opinion in Neurophysiology 4:877-887.

Massion J. 1998. Postural control systems in developmental perspective. Neuroscience Biobehavioral Review 4:465-472.

Matsuyama K, Drew T. 2000. Vestibulospinal and Reticulospinal Neuronal Activity During Locomotion in the Intact Cat.II. Walking on an Inclined Plane. Journal of Applied Physiology 84:2257-2276.

Matthews PBC. 1991. The human stretch reflex and the motor cortex. Trends in Neurosciences 14:3 87-91.

Maurer C, Mergner T, Xie J, Faist M, Pollak P, Lücking CH. 2003. Effect of chronic bilateral subthalamic nucleus (STN) stimulation on postural control in Parkinson's disease. Brain 126:5 1146-1163.

Maurer C, Peterka RJ. 2005. A new interpretation of spontaneous sway measures based on a simple model of human postural control. Journal of Neurophysiology 93:1 189-200.

Mazzaro M, Sznaier M, Camps O. 2005. A model (in) validation approach to gait classification. IEEE Transactions on Robotics 27:11.

McCollum G, Holroyd C, Castelfranco M. 1995. Forms of early walking. Journal of Theoretical Biology 176:373-390.

McCollum G, Leen T. 1989a. Form and exploration of mechanical stability limits in erect stance. Journal of Motor Behavior 21:3 225-244.

McCollum G, Leen T. 1989b. Form and exploration of mechanical stability limits in erect stance. Journal of Motor Behaviour 21:225-244.

McGeer T. 1990. Passive Dynamic Walking. International Journal of Robotic Research 9:62-82.

Meadows L, Williams J. 2009. An understanding of functional movement as a basis for clinical reasoning. In S. Raine, L. Meadows & M. Lynch-Ellerington (Eds.), Bobath Concept - theory and clinical practice in neurological rehabilitation: Wiley-Blackwell.

Mergner T, Rosemeier T. 1998. Interaction of vestibular, somatosensory and visual signals for postural control under terrestrial and microgravity conditions: a conceptual model. Brain Research Review118-135.

Mileykovskiy BY, Kiyashchenko LI, Kodama T, Lai Y-Y, Siegel JM. 2000. Activation of Pontine and Medullary Motor Inhibitory Regions Reduces Discharge in Neurons Located in the Locus Coeruleus and the Anatomical Equivalent of the Midbrain Locomotor Region. The Journal of Neuroscience 20:22 8551-8558.

Miyai I, Yagura H, Oda I, Konishi I, Eda H, Suzuki T, et al. 2002. Premotor cortex is involved in restoration of gait in stroke. Annals of Neurology 52:2 188-194.

Mochizuki L, Duarte M, Amadio A, Zatsiorsky V, Latash M. 2006. Changes in postural say and its fractions in conditions of postural instability. Journal of Applied Biomechanics 22:51-60.

Morasso PG, Baratto L, Capra R, Spada G. 1999. Internal models in the control of posture. Neural Networks 12:7–8 1173-1180.

Nardone A, Giordano A, Corrá T, Schieppati M. 1990. Responses of leg muscles in humans displaced while standing. Effects of types of perturbation and of postural set. Brain 113:1 65-84.

Nardone A, Grasso M, Giordano A, Schieppati M. 1996. Different effect of height on latency of leg and foot short- and medium-latency EMG responses to perturbation of stance in humans. Neuroscience Letters 206:2–3 89-92.

Nashner L, Shupert C, Horak F, Black F. 1989. Organization of posture controls: an analysis of sensory and mechanical constraints. In Elsevier (Ed.), Progress in brain research: afferent control of posture and locomotion (pp. 411-418). New York.

Nelson RJ. 1996. Interactions between motor commands and somatic perception in sensorimotor cortex. Current Opinion in Neurobiology 6:6 801-810.

Neptune R, Kautz A, Zajac E. 2001. Contributions of the individual ankle flexors to support, forward progression and swing initiation during normal walking. Journal of Biomechanics 34:11 1387-1398.

Neptune R, Kautz S, Zajac F. 2004. Muscle force redistributes segmental power for body progression during walking. Gait & Posture 19:2 194-205.

Nielsen J, Kagamihara Y. 1992. The regulation of disynaptic reciprocal Ia inhibition during co-contraction of antagonistic muscles in man. Journal of Physiology 456:373-391.

Nielsen JB, Sinkjaer T. 2002. Afferent feedback in the control of human gait. Journal of electromyography and kinesiology 12:3 213-217.

Norkin C, Levangie K. 1992. Joint Structure and Function. A Comprehensive Analysis. 2nd ed. EUA: Library of Congress.

Orendurff MS, Segal AD, Aiona MD, Dorociak RD. 2005. Triceps surae force, length and velocity during walking. Gait & Posture 21:2 157-163.

Orlovsky N. 1991. Cerebellum and locomotion. In M. Shimamura, S. Griller & V. Edgerton (Eds.), Neurobiologic Basis of Human Locomotion (pp. 187-199). Tokyo: Scientific Society Press.

Pandy MG. 2003. Simple and complex models for studying muscle function in walking. Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences 358:1437 1501-1509.

Park S, Horak F, Kuo A. 2004. Postural feedback responses scale with biomechanical constraints in human standing. Experimental Brain Research 154:417-427.

Pearson K. 1993. Common principals of motor control in vertebrates and invertebrates. Annual Review of Neuroscience 16:265-297.

Peterka R. 2002. Sensorimotor integration in human postural control. Journal of Neurophysiology 88:1097-1118.

Peterka R, Loughlin P. 2004. Dynamic regulation of sensorimotor integration in human postural control. Journal of Neurophysiology 91:410-423.

Peterka RJ. 2000. Postural control model interpretation of stabilogram diffusion analysis. Biological Cybernetics 82:4 335-343.

Peterka RJ, Benolken MS. 1995. Role of somatosensory and vestibular cues in attenuating visually induced human postural sway. Experimental Brain Research 105:1 101-110.

Pierrynowski MR, Winter DA, Norman RW. 1980. Transfers of mechanical energy within the total body and mechanical efficiency during treadmill walking. Ergonomics 23:2 147-156.

Proske U. 2006. Kinestesia: the role of muscle receptors. Muscle Nerve 34:5 545-558.

Proske U, Wise A, Gregory J. 2000. The role of muscle receptors in the detection of movements. Progress in Neurobiology 60:1 85-96.

Raine S, Meadows L, Lynch-Ellerington M. 2009. Bobath concept. Theory and clinical practice in neurological rehabilitation. United Kingdom: Wiley-Backwell.

Riley MA, Wong S, Mitra S, Turvey MT. 1997. Common effects of touch and vision on postural parameters. Experimental Brain Research 117:1 165-170.

Roll R, Kavounoudias A, Roll J-P. 2002. Cutaneous afferents from human plantar sole contribute to body posture awareness. NeuroReport 13:15 1957-1961.

Rossignol S, Dubuc R, Gossard P. 2006. Dynamic sensorimotor interactions in locomotion. Physiology Review 86:89-154.

Rothwell J. 1994. Control of Human Voluntary Movement. 2 ed. London: Chapman & Hall.

Saibene F, Minetti A. 2003. Biomechanical and physiological aspects of legged locomotion in humans. European Journal of Applied Physiology 88:4 297-316.

Santos M, Aruin A. 2008. Role of lateral muscles and body orientation in feedforward postural control. Experimental Brain Research 184:4 547-559.

Santos M, Kanekar N, Aruin A. 2009. The role of anticipatory postural adjustments in compensatory control of posture: 1. Electromyographic analysis. Journal of Electromyography and Kinesiology 20:3 388-397.

Saunders M, Inman T, Heberhart D. 1953. The major determinants in normal and pathological gait. The Journal of Bone and Joint Surgery 53:3 543-558.

Schepens B, Drew T. 2004. Independent and convergent signals from the pontomedullary reticular formation contribute to the control of posture and movement during reaching in the cat. Journal of Neurophysiology 92:2217-2238.

Schieppati M, Nardone A, Siliotto R, Grasso M. 1995. Early and late stretch responses of human foot muscles induced by perturbation of stance. Experimental Brain Research 105:3 411-422.

Schneider C, Lavoie BA, Capaday C. 2000. On the origin of the soleus H-reflex modulation pattern during human walking and its task-dependent differences. Journal of Neurophysiology 83:5 2881-2890.

Segers V. (2006). *A biomechanical analysis of the realization of actual human transition.* Ghent University, Ghent.

Shiratori T, Aruin A. 2007. Modulation of anticipatory postural adjustments associated with unloading perturbation: effect of characteristics of a motor action. Experimental Brain Research 178:2 206-215.

Shumway-Cook A, Woolacott M. 2000. Attentional demands and postural control: the effect of sensory context. Journal of Gerontology 55A:M10-16.

Shumway-Cook A, Woolacott M. 2007. Motor control: translating research into clinical practice. 3 ed.: Lippincott Williams & Wilkins.

Siegel A, Sapru H. 2011. Essential Neuroscience (2 ed.). China: Lippincott Williams & Wilkins.

Sinkjær T, Andersen JB, Ladouceur M, Christensen LOD, Nielsen JB. 2000. Major role for sensory feedback in soleus EMG activity in the stance phase of walking in man. The Journal of Physiology 523:3 817-827.

Sinkjaer T, Andersen JB, Larsen B. 1996. Soleus stretch reflex modulation during gait in humans. Journal of Neurophysiology 76:2 1112-1120.

Sparrow W, Newell. 1998. Metabolic energy expenditure and the regulation of movement economy. Psychonomic Bulletin & Review 5:2 173-196.

Stainsby W, Gladden L, Barclay J, Wilson B. 1980. Exercise efficiency: validity of base-line subtractions. Journal of Applied Physiology 48:518-522.

Stapley P, Pozzo T, Cheron G, Grishin A. 1999. Does the coordination between posture and movement during human whole-body reaching ensure center of mass stabilization. Experimental Brain Research 129:134-146.

Stephens MJ, Yang JF. 1996. Short latency, non-reciprocal group I inhibition is reduced during the stance phase of walking in humans. Brain Research 743:1–2 24-31.

Stephens MJ, Yang JF. 1999. Loading during the stance phase of walking in humans increases the extensor EMG amplitude but does not change the duration of the step cycle. Experimental Brain Research 124:3 363-370.

Takakusaki K, Saitoh K, Harada H, Kashiwayanagi M. 2004. Role of basal ganglia–brainstem pathways in the control of motor behaviors. Neuroscience Research 50:2 137-151.

van der Fits I, Klip A, van Eykern L, Hadders-Algra M. 1998. Postural adjustments accompanying fast pointing movements in standing, sitting and lying adults. Experimental Brain Research 120:2 202-216.

van der Kooij H, Jacobs R, Koopman B, van der Helm F. 2001. An adaptive model of sensory integration in a dynamic environment applied to human stance control. Biologic Cybernetics 84:2 103-115.

Van Emmerik R, Van Wegen E. 2000. On variability and stability in human movement. Journal of Applied Biomechanics 16:394-406.

Vandewalle H. 2004. Consommation d'oxygène et consommation maximale d'oxygène : intérêts et limites de leur mesure. Annales de Réadaptation et de Médecine Physique 47:6 243-257.

Waters L, Mulroy S. 1999. The energy expenditure of normal and pathological gait: relation to mechanical energy cost. Journal of Neurophysiology 9:3 207-231.

Waters RM, S. 1999. The energy expenditure of normal and pathologic gait. Gait Posture 9:3 207-231.

Wichmann T, DeLong MR. 1996. Functional and pathophysiological models of the basal ganglia. Current Opinion in Neurobiology 6:6 751-758.

Willems A, Cavagna A, Heglund C. 1995. External, internal and total work in human locomotion. The Journal of Experimental Biology 198:379-383.

Winter D. 1995. Human balance and posture control during standing and walking. Gait & Posture 3:193-214.

Winter D. 2005. Biomechanics and motor control of human movement. NJ: John Wiley and Sons, Inc.

Winter D, Patia A, Frank J. 1990. Assessment of balance control in humans. Medical Progress Through Technology 16:31-51.

Winter DA, Eng P. 1995. Kinetics: our window into the goals and strategies of the central nervous system. Behavioural Brain Research 67:2 111-120.

Winter DA, MacKinnon CD, Ruder GK, Wieman C. 1993. Chapter 32 An integrated EMG/biomechanical model of upper body balance and posture during human gait. In D. J. A.-M. F. P. H. J.H.J. Allum & R. Probst (Eds.), Progress in Brain Research (Vol. Volume 97, pp. 359-367): Elsevier.

Wright W, Ivanenko Y, Gurfinkel V. 2012. Foot anatomy specialization for postural sensation and control. Journal of Neurophysiology 107:5 1513-1521.

Yang J, Winter D, Wells R. 1990. Postural dynamics in standing human. Biological Cybernetics 62:4 309-320.

Yang JF, Stein RB, James KB. 1991. Contribution of peripheral afferents to the activation of the soleus muscle during walking in humans. Experimental Brain Research 87:3 679-687.

Yang JF, Whelan PJ. 1993. Neural mechanisms that contribute to cyclical modulation of the soleus H-reflex in walking in humans. Experimental Brain Research 95:3 547-556.

Zajac F, Neptune R, Kautz S. 2003. Biomechanics and muscle coordination of muscle walking Part II: Lessons from dinamic simulations and clinical implications. Gait & Posture 17:1 1-17.

Zatsiorsky V, Duarte M. 1999. Instant equilibrium point and its migration in standing tasks: rambling and trembling components of the stabilogram. Motor Control 3:1 28.

Zehr EP, Duysens J. 2004. Regulation of arm and leg movement during human locomotion. The Neuroscientist 10:4 347-361.