Disponible en ligne sur

ScienceDirect
www.sciencedirect.com

Elsevier Masson France

EM|consulte
www.em-consulte.com/en

REVIEW/MISE AU POINT

Gait post-stroke: Pathophysiology and rehabilitation strategies

La marche après accident vasculaire cérébral : physiopathologie et stratégies de rééducation

C. Beyaert [a,b,*], R. Vasa [c], G.E. Frykberg [d]

[a] EA3450, Université de Lorraine, Faculty of Medicine, 54500 Vandœuvre-lès-Nancy, France
[b] Motion Analysis Laboratory, L.-Pierquin Rehabilitation Center, 54000 Nancy, France
[c] RV Foundation, Centre for Brain and Spinal Injury Rehab, Mumbai, India
[d] Department of Neuroscience/Rehabilitation Medicine, Uppsala University, 75158 Uppsala, Sweden

Summary We reviewed neural control and biomechanical description of gait in both non-disabled and post-stroke subjects. In addition, we reviewed most of the gait rehabilitation strategies currently in use or in development and observed their principles in relation to recent pathophysiology of post-stroke gait. In both non-disabled and post-stroke subjects, motor control is organized on a task-oriented basis using a common set of a few muscle modules to simultaneously achieve body support, balance control, and forward progression during gait. Hemiparesis following stroke is due to disruption of descending neural pathways, usually with no direct lesion of the brainstem and cerebellar structures involved in motor automatic processes. Post-stroke, improvements of motor activities including standing and locomotion are variable but are typically characterized by a common postural behaviour which involves the unaffected side more for body support and balance control, likely in response to initial muscle weakness of the affected side. Various rehabilitation strategies are regularly used or in development, targeting muscle activity, postural and gait tasks, using more or less high-technology equipment. Reduced walking speed often improves with time and with various rehabilitation strategies, but asymmetric postural behaviour during standing and walking is often reinforced, maintained, or only transitorily decreased. This asymmetric compensatory postural behaviour appears to be robust, driven by support and balance tasks maintaining the predominant use of the unaffected side over the initially impaired affected side. Based on these elements, stroke

---

* Corresponding author. Laboratoire d'analyse du mouvement, Centre de Réadaptation L.-Pierquin, 75, boulevard Lobau, CS34209, 54042 Nancy cedex, France. Tel.: +33 3 83 52 98 22; fax: +33 3 83 52 98 89.
E-mail address: christian.beyaert@univ-lorraine.fr (C. Beyaert).

rehabilitation including affected muscle strengthening and often stretching would first need to correct the postural asymmetric pattern by exploiting postural automatic processes in various particular motor tasks secondarily beneficial to gait.

Résumé   Le contrôle nerveux et la biomécanique de la marche chez le sujet sain et le sujet hémiplégique ainsi que les stratégies de rééducation au regard de la physiopathologie de la marche après accident vasculaire cérébral (AVC) ont été passés en revue. Chez le sujet sain ou hémiplégique, un contrôle moteur de type modulaire tâche-dépendant assure simultanément le support antigravitaire, le contrôle de l'équilibre et la déambulation au cours de la marche. L'hémiparésie après AVC est due à une interruption de voies nerveuses descendantes habituellement sans lésion directe des structures du tronc cérébral et du cervelet impliqués dans le contrôle moteur automatique. Après AVC, la récupération d'une station debout et de la marche se fait habituellement avec un comportement postural impliquant davantage le côté non atteint dans le contrôle du support et de l'équilibre, probablement suite à la faiblesse musculaire initiale du côté atteint. Divers types de rééducation sont pratiqués, visant directement le comportement musculaire ou des tâches posturales ou de locomotion en utilisant un équipement plus ou moins sophistiqué. Bien que la faible vitesse de marche s'améliore souvent avec le temps ou après diverses rééducations, le comportement postural asymétrique est souvent renforcé, maintenu ou seulement réduit transitoirement. Ce comportement postural asymétrique apparaît robuste, avec utilisation prédominante du côté non atteint pour assurer les tâches posturales antigravitaires. Ainsi chez les patients post-AVC, il est suggéré que la rééducation, incluant des renforcements et étirements des muscles parétiques, associe aussi divers exercices impliquant une utilisation posturale automatique des muscles parétiques pour corriger le comportement postural asymétrique et en tirer un bénéfice secondaire sur la marche.
© 2015 Elsevier Masson SAS. Tous droits réservés.

Introduction

Walking dysfunction is a major problem for many subjects afflicted by stroke [2,109] and it causes difficulties in performing daily activities. Furthermore, there is a high risk for falls at all stages after stroke [6,59,254] and walking has been reported to be the event when falling most often occurs in community-dwelling stroke survivors [100]. Additionally, improving walking, with respect to safety and speed, is a major goal for stroke subjects in rehabilitation [59,193]. Discrimination between true recovered and compensatory movement patterns is increasingly emphasized in stroke rehabilitation nowadays (e.g. [20,140]), and for discriminative purposes, the quality of movement patterns needs to be observed, registered and analysed [146]. Moreover, our understanding of underlying mechanisms behind the emergence of both true recovery and compensation is still poor [20,140,248]. From the research perspective, the number of published neurophysiological studies regarding walking is rapidly expanding and is providing new knowledge about neural and biomechanical control including kinetic, muscle activation and kinematic data from typical and from post-stroke gait. These results might contribute to a deeper understanding of implicit factors influencing the mechanisms for true recovery post-stroke. From the clinical research side, the number of systematic reviews scrutinizing gait rehabilitation strategies post-stroke is also increasing, and there is an on-going discussion regarding what the optimal gait training models might be. The aims of the current review article are:

- to provide an update of the comprehensive new neurophysiological knowledge about typical and post-stroke gait reported in recent years;
- to give an overview of most of the gait rehabilitation strategies currently in use or in development;
- to supply a synthesis of research results and effects of current gait training models after stroke and to determine whether these models have implemented the new knowledge in practice.

Physiology of typical gait

Neural control of gait

In humans, bipedal locomotion is a motor task where the control system, in each step, needs to support body weight, provide forward and lateral stability and maintain forward progression. Thus, the postural antigravity control which provides body support and balance control to prevent falling is continuously associated with progression [165,177,260]. In addition, adaptation allows the adjustment of gait patterns to the environment [195]. The articulated body segments with quite different mass and inertia are linked by muscles with their own idiosyncratic viscoelastic characteristics, and their totality is responsible for the production of force and kinematics. The consequence is that each single joint movement involves dynamic interactions with the other segments of the kinematic chain inducing postural disturbance

[260]. Thus, the overall behaviour of the body and limbs during walking is determined by the net forces and torques resulting from the interplay of neural and mechanical factors [135].

The neural control of muscles has been shown to be modular, organized in functional groups often referred to as modules or muscle synergies [66,240]. Each module is defined by a fixed pattern of co-activation across multiple muscles at any given point in time. Modules have been used to describe muscle coordination during a variety of motor tasks, including balance control [242] and walking [35,101,187]. Modules may be organized to produce specific whole-limb or whole-body biomechanical functions during locomotion [4,35,102,187] such that altering the duration, phase or amplitude of muscle synergy recruitment may produce a variety of locomotor behaviours [168,239]. Thus, modules are suggested to allow the nervous system to produce consistent biomechanical functions. Given the specific sets of muscles investigated, four to five motor modules were sufficient to reconstruct a locomotor task with sufficient quality [36,101,168,187]. Neptune et al. [187] described that body support was provided by Module 1 (hip and knee extensors, hip abductors) in early stance and Module 2 (plantarflexors) in late stance. Forward propulsion was provided in early stance by Module 4 (hamstrings) and in late stance by Module 2, but net braking occurred in Modules 1 and 2. Module 3 (ankle dorsiflexors, rectus femoris) and 5 (hip flexors and adductors except adductor magnus) accelerate the ipsilateral leg forward in early swing whereas Module 4 decelerates the ipsilateral leg prior to heel-strike [4,187]. Recent studies have shown that modules identified in one task are also recruited by different neural pathways subserving other motor behaviours [35,36,192]. Thus, locomotor modules were also recruited in atypical phases of gait, accounting for both anticipatory gait modifications before perturbations and reactive feedback responses to perturbations [35,192]. A common set of modules has been shown to account for both locomotion and reactive balance control [36] suggesting that modules form a general repertoire of motor actions that can be recruited by a variety of different neural pathways for voluntary, rhythmic, and reactive motor behaviours [35,36].

The interconnected neural structures and their functionality underlying the biomechanical motor behaviour during gait has been described in increasing detail based on animal and human studies. The following simplified description is mainly based on cat, monkey and human studies (see Fig. 1). In mammals, spinal central pattern generators produce the basic command signals sent to muscles of the limbs for locomotor rhythm and pattern generation [89]. In humans, spinal locomotor pattern generator activity can be induced [58] and was recently described as a flexible organization of burst-generating elements [47]. Thus, 3 to 4 burst-generating elements driving the motor neuron pools are modularly organized and flexibly combined to produce a variety of rhythmic patterns including reciprocal or simultaneous activation of antagonist muscles [47]. Other studies in humans suggest that motor modules are largely controlled by the brainstem and spinal network [61,68,76,84] with cortical modulation during volitional locomotion [61,265]. During locomotion, the motor output achieves rhythmic patterns for progression and both support and balance functions.

Body support allows upright posture against gravity mainly by the action of trunk and lower limb extensors. The pontomedullary reticular formation (PMRF) and vestibular nuclei are the main brainstem structures involved in support and balance control under the regulating control of the cerebellum [46,65,75,155,164,177,223]. The PMRF contains in addition neurons, which activate the spinal rhythmic network under the influence of either the mesencephalic locomotor region, subthalamic locomotor region or cerebellum [65,177,223,237]. However, rhythmic activity can be induced only if an adequate level of postural muscle tone for support is developed [174]. On the other hand, suppression of postural muscle tone inhibits locomotor movements [175]. In other words, locomotion can be achieved only if prior adequate postural support is present. Locomotion initiation usually depends on either a ''volitional or cognitive reference'' involving the cerebral cortex or an ''emotional reference'' involving the limbic system [236]. Regardless of whether the locomotion initiation is volitional or emotional, it is accompanied by the above-mentioned automatic processing for support, balance and rhythmic activity [237]. The volitional process requires activation of various cortical areas and is executed by projections to the brainstem and spinal chord [166,237]. Locomotion after initiation is usually achieved in the absence of conscious awareness. However, intentional gait modification is needed when obstacles are encountered and requires motor programming in the premotor cortices [237]. The cerebellum regulates volitional and automatic processes by acting on the cerebral cortex and brainstem, respectively [164,177,237]. It likely takes into account simultaneously both feed-forward information from the cerebral cortex by the olivocerebellar tract and real-time sensory feedback by the spinocerebellar and vestibulocerebellar tracts [51,164,177,237]. In addition, the cerebellum is crucial for locomotor adaptation and learning processes [164,177]. The basal ganglia receive inputs from the cerebral cortex and influence volitional, emotional and automatic processes through projections to the cerebral cortex, limbic system and brainstem, respectively [235,237]. In summary, walking is mainly the result of automatic processing, which offers advantages compared to intentionally controlled cognitive processing [40,225].

Biomechanical description of gait

The biomechanical features of gait in non-disabled individuals are quite reproducible. The trajectories of the centre of mass (COM), i.e. the single point at which the whole mass of the body is imagined to be concentrated, and feet are highly regular and repeatable [260]. They are determined by the combined rotation and translation of the lower limb segments. The pelvis, thigh, shank and foot oscillate back and forth relative to the vertical with a similar waveform, time-shifted across different segments [14]. When the segment elevation angles are plotted one versus the others, they describe regular loops constrained close to a plane common to both the stance and swing phase [14]. The related planar law of intersegmental coordination is robust and applies for different gait velocities and different postures [88]. This planar law is not a simple consequence of biomechanics but
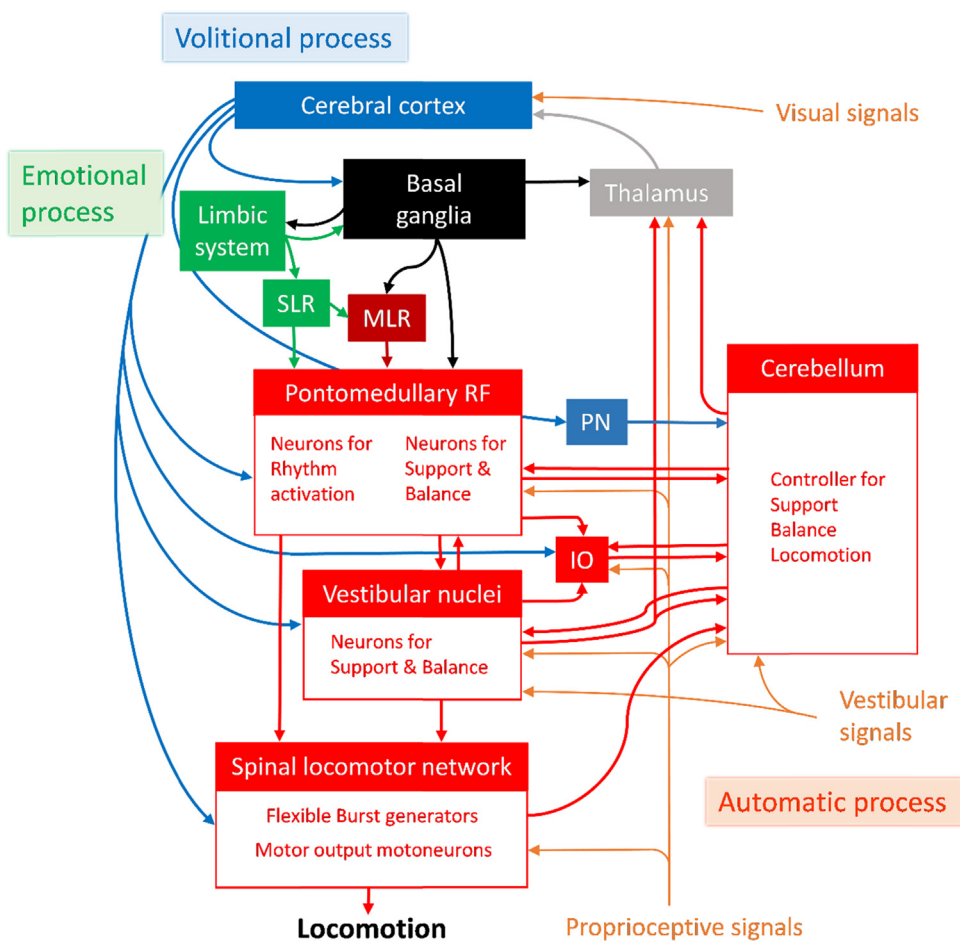
Figure 1 Schematic illustration of structures and processes involved in gait control. A volitional process, which involves the cerebral cortex, and an emotional process, which involves the limbic system, can both generate gait and are both accompanied by automatic processes involving the brainstem, cerebellum and spinal cord. An automatic process simultaneously controls support, balance and rhythmic activity. The basal ganglia and the cerebellum control volitional and automatic processes by thalamocortical projections and by direct projections to the brainstem, respectively. See text for further explanation. SLR: subthalamic locomotor region; MLR: mesencephalic locomotor region; RF: reticular formation; PN: pontine nuclei; IO: inferior olive.

likely related to central representation of kinematic components [103].

At optimal speed, walking saves energy by exchanging forward kinetic energy and gravitational potential energy of the COM during the inverted-pendulum oscillation of stance [26], and by oscillating the limb ballistically as an upright compound-pendulum during swing [173]. Mechanical (and metabolic) energy is mainly expended to redirect COM velocity during step-to-step transitions [132] and to force leg oscillation for swing [160]. During the step-to-step transition, mechanical work is required to redirect the COM velocity vector between the pendulum arcs of each limb [62,264]. Redirection comes from the net combination of (1) negative (or decelerating) work during the leading limb's double support (or loading response phase) and (2) positive (or accelerating) work during the trailing limb's double support (or pre-swing phase) [62]. Negative work during the leading limb's double support is used to change the direction of the COM velocity vector [62] and to stabilize gait in forward direction [133] and positive work is necessary to restore loss of energy. Minimizing total mechanical work is desirable to

minimize metabolic cost [63] and can occur when the timing and magnitude of the leading limb's negative work is equal to the trailing limb's positive work [232]. Divergence from metabolic optimization arises from inter-limb mechanical asymmetries during step-to-step transitions in both healthy [232] and clinical populations [72].

A brief and partial description of the functional phases and limb motions of normal gait will follow here (see Perry and Burnfield's book [201] for detailed description). After initial heel-strike with impact deceleration (initial contact), the loading response phase (LR) occurs during which sagittal deceleration is achieved by the knee and hip extensors for shock absorption and for restraining the trunk forward momentum (see Fig. 2). Once the foot is flat on the floor at late LR, it gives stability and plantarflexors contribute to restrain trunk forward momentum [185]. With the transition to single limb support, knee extension stability is provided by an anterior ground reaction force vector and by calf stabilization of the tibia. Single limb support is achieved during mid-stance (foot flat) and terminal stance (heel rise) with the ankle as fulcrum for continued progression associated
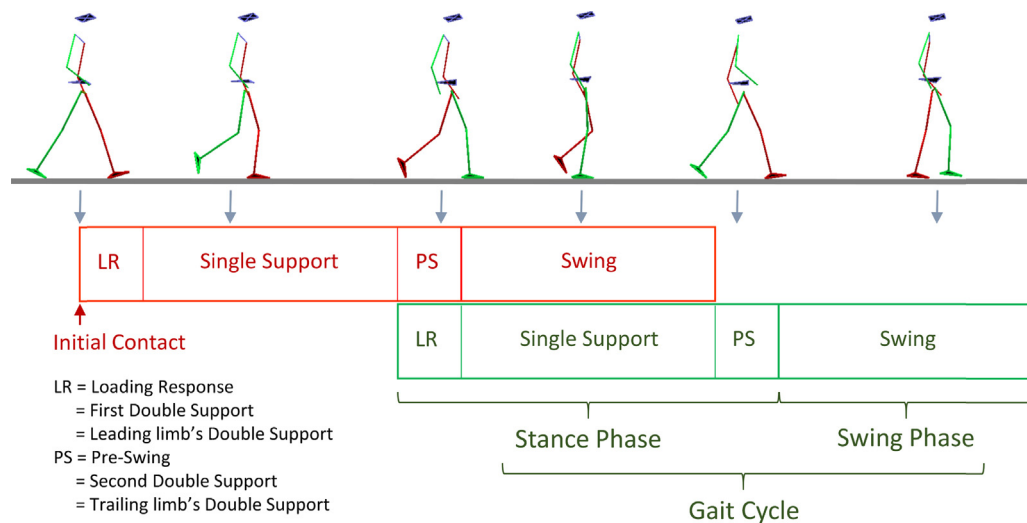
Figure 2   Temporal parameters of the gait.

with dorsiflexion movement. During single limb support, there is a forward motion of the body beyond the supporting foot with the main contribution of plantarflexors to body support and forward progression. At pre-swing phase (PS), the final arc of plantarflexion allows propulsion (push-off) and initiation of knee flexion for swing. During swing phase, foot clearance at mid swing is achieved by dorsiflexion, knee and hip flexion. Hip flexors advance the limb during swing but the demand is low. The tibialis anterior has a double-phase pattern of activity, the first occurring at pre-swing and initial-swing phases for dorsiflexion and foot clearance and the second at terminal swing and loading response for decelerating foot drop.

Gait adaptability

Adaptability to altered conditions of gait is essential, either to environmental characteristics or to body anatomical and/or functional alterations. For example, walking on a slippery walkway induces a specific gait mode including increased limb stiffness, shorter steps, initial and prolonged flat foot contact with the ground and toe grip allowing lower shear stress applying to the ground and greater available ground friction for braking purpose [24,74]. In order to have further insights for pathophysiology of post-stroke gait, the following sections will consider gait analyses in self-selected speed but also in comparison to additional conditions to study gait adaptability and the effect of various treatments.

Description and pathophysiology of gait post-stroke

About 80% of subjects afflicted by stroke experience walking problems 3 months after onset [2]. In a comprehensive prospective study of over 800 subjects with stroke, with a mortality rate of 21%, 18% could not walk at all, 11% walked when assisted and the remaining 50% walked independently at the end of rehabilitation [109]. Furthermore, 70% of community-dwelling post-stroke individuals fall during the

first year [254] and most falls occur due to loss of balance when walking [100]. Thus, there is a high fall risk in those subjects with post-stroke symptoms who are independently walking.

Neural impairment and neural control of gait post-stroke

Muscle weakness and loss of voluntary movements are predominant signs immediately following a stroke [184,193]. Acute stroke is defined by sudden neurologic deficit of vascular origin lasting more than 24 hours. The related hemiparesis is due to disruption of descending neural pathways, with no direct lesion on the musculoskeletal system or spinal circuit [44]. In the acute phase, there is a high degree of correlation between individual motor tests for both upper and lower limb on the affected side [44]. Similar patterns of motor deficits occur and result from lesion(s) in many regions including the motor cortex, putamen, insula, frontal and parietal cortex, and descending motor pathways [44]. These findings suggest that a small number of correlated components are represented in patterns of neural activity at multiple levels in the motor system [32,38]. Brainstem strokes essentially of ischemic origin account for 4−16% of total brain strokes [25,48], involving the pons (54%) more commonly than the medulla (28%) or midbrain (14%) [130]. The dorsal portion of pons and medulla is rarely (10%) affected by stroke usually then sparing both the pontomedullary reticular formation and vestibular nuclei and their connections to the cerebellum and the spine [21,49,118,131,207]. On the other hand, the inferior olive is frequently affected in case of medullary infarction resulting in cerebellar syndrome [21,118]. Cerebellar strokes essentially of ischemic origin account for 2−6% of total brain strokes [25,241]. In summary, the brainstem and cerebellar structures involved in automatic process of gait control (see Fig. 1) are intact in more than 90% of total brain strokes. In post-stroke subjects, these latter structures are particularly active while walking [153,263] whereas walking can be observed after complete lateral corticospinal tract injury

of the affected hemisphere [1]. In post-stroke subjects, the brainstem, spinal and cerebellar activities are obviously essential in gait generation and patterning but the question remains open about the pathophysiology of the gait dysfunction. Gait deviations consecutive to body anatomical and/or function alterations can be divided in principle into:

- primary deviations that are directly due to the pathology and usually present at the beginning of the pathology;
- secondary deviations which split into:
  - passive secondary effects that follow as a physical effect of the primary deviation,
  - active secondary deviations (i.e. compensatory mechanisms/compensations) that act in order to actively offset primary deviations and secondary physical effects [224].

In post-stroke subjects, gait deviations can be explicitly described compared to gait of able-bodied subjects but the underlying or implicit mechanisms still need to be further understood. In particular, distinguishing between primary and secondary deviations is of importance for treatment. Primary gait deviations would be mostly related to the initial disruption of descending neural pathways while secondary deviations are neural adaptive processes. Adaptive processes can be cognitive and/or automatic, involving in both cases the cerebellum, which is usually intact in stroke. These processes can use already learned compensations and/or learn new specific compensations. Common compensatory mechanisms that appear to be independent from the underlying disease have been described across different patient groups [224]. For example, reduced foot clearance in the swing phase could be compensated by either a pelvic hike, circumduction or vaulting as seen in post-stroke subjects [30,45,116,163,224]. On the other hand, in post-stroke gait, the asymmetric pattern with higher kinetic involvement of the unaffected side (see below for description) may be a specific adaption to the initial and on-going paresis of the affected side. This adaptation may involve both automatic and intentional cognitive processes, for support, balance and progression purposes. In general, there is an early activation of the contralesional cortex in the acute phase of stroke with a gradual return to normal ipsilesional activation during the subacute and chronic phases after stroke [82]. In chronic stroke, an increased fibre volume of the corticoreticular pathway observed in the unaffected hemisphere may be related to adaptive mechanisms [104].

Improvements in voluntary muscle activity and strength have been described to involve spasticity and stereotyped movements in accordance with Brunnström's notion of hemiplegic limb synergies [19]. However, spasticity prevalence is highly variable, ranging from 4% to 27% 1—4 weeks post-stroke and 17% to 43% after 3 months post-stroke [261]. In addition, only a minor proportion of subjects 3 months post-stroke have their voluntary movements restricted to the synergies described by Brunnström [255]. The role of spasticity on post-stroke gait patterns is controversial [22,57,150] and will not be debated in depth in this review. Spasticity is characterized by a velocity-dependent increase in resistance during passive stretch, resulting from hyperexcitability of the stretch reflex [138]. Exaggerated reflexes appear to have a minor role and secondary changes in mechanical muscle fibre properties, with increased resistance to stretch having a greater role in movement disorders during gait [57]. Indeed, immobilisation initially related to paresis can conduct quickly to muscle atrophy and contracture when in shortened position including then an increase in the ratio of collagen to muscle fibre tissue [87]. In addition, the medial reticulospinal tract, which plays an important role in maintaining joint position and posture against gravity, would increase both muscle tone and stretch reflex excitability of antigravity muscle groups, e.g. upper limb flexors and lower limb extensors in upright posture [65,150]. Thus, prolonged muscle tone would be included in the feed-forward motor control during gait [150,237] whereas stretch reflexes would be evoked as feedback postural reaction in response to balance perturbations particularly during slower and unstable gait post-stroke as observed during stance posture in non-disabled and post-stroke individuals [15,98,152,182]. Whereas reticulospinal pathways are largely bilateral in their anatomical spinal distribution, the unilateral nature of vestibulospinal pathways may give to the latter an important role in the sharply lateralized nature of muscle hypertonia and spasticity in stroke [171]. Interestingly in favour of a role of the vestibular pathways and postural orientation, Denny-Brown observed in the monkey that in the affected side, tonic posture of flexed upper limb and extended lower limb in upright posture was reversed to active extended upper limb and flexed lower limb when the animal was suspended in air by the pelvis with the head-down and the neck extended [54]. In addition, when the attitude of limbs changed as a result of head-down posture, the reflex pattern also changed so that the flexor spasticity disappeared and extensor spasticity appeared in upper limbs and vice-versa in lower limbs [54].

Activation of the affected muscles can be voluntary and/or automatic. Central disuse (learned non-use) of the paretic body parts contributes to reducing the ability to activate muscle by voluntary command [87]. On the other hand, automatic affected muscle over-activity usually emerges with difficulty in arresting motor unit activity in specific muscles, whether in situations of complete voluntary rest or during actions normally involving other muscles only [86]. In particular, spastic dystonia and spastic co-activation have been described and distinguished by their primary triggering factor, i.e., tonic muscle stretch and volitional command, respectively [86]. The muscle over-activity has been studied mostly in upper limbs but also in lower limbs in open chain unloaded position [86]. In the functional condition of walking in stroke subjects, muscle co-activation in lower limbs is particularly frequent in both the affected and unaffected limbs [215]. Muscle co-activation is a common compensatory strategy providing mechanical stability by stiffening joints, such as during very slow walking speeds [52] or walking on a slippery surface [24], in the elderly [114,205], in individuals who have undergone knee arthroplasty [70] and in individuals with cerebellar ataxia [162]. In individuals with stroke, muscle co-activation may represent an adaptation to compensate for impaired stability during step transition [33,136,215]. One can note that the duration of lower limbs muscle co-activation *per se* does not impede functional gait improvements in terms of maximum walking speed increase [53] although it would be associated with higher energy cost [136]. On the other hand, muscle co-activation timing can be

reduced as has been shown for thigh muscles in both limbs after a gait training programme on a treadmill with real-time feedback of the body's COM in stroke subjects [163].

A modular organization of muscle coordination underlying walking occurs in post-stroke and in non-disabled subjects. Clark and colleagues [38], in a study based on EMG signals from eight leg muscles in 55 adults with chronic stroke and controls, reported that four modules were required in non-disabled controls and in most of the unaffected legs (58%). However, most of affected legs required just two (45%) or three (36%) modules, resulting from merging of the modules observed in the controls. The number of modules (2, 3 or 4) was correlated to prefer walking speed, speed modulation, step length asymmetry, and propulsive asymmetry. Thus, persons with fewer modules on the affected limb walked more slowly and had more asymmetrical step lengths and propulsion generation. Post-stroke, the central nervous system (CNS) adapts the existing modules available for both postural and locomotor controls [36] rather than introducing new modules as also shown in non-disabled subjects [192]. Merging of existing modules on the affected side in post-stroke subjects may be generated by the CNS to allow automatic simplified body support of the affected side in response to the initial muscle weakness and lack of muscle voluntary control. This modification would be included in a larger adaptation of the CNS, reducing the contribution from the affected side and increasing the unaffected side's contribution to body support and balance control during standing and walking. However, as more modules are merged, greater interference between subtasks occurs, leading to poorer walking performance and limited mobility capability [217]. Improving walking ability with locomotor training including body weight support has been found to result in improvements in the number and quality of modules post-stroke [216]. It has been shown that the planar law of sagittal intersegmental coordination during gait was similarly followed by non-disabled individuals and both the affected and unaffected legs of individuals with chronic stroke walking at moderate or high speed [34]. This centrally controlled kinematic coordination [103,134] might be related to the modular organization of muscle coordination.

Spatio-temporal characteristics of gait post-stroke

Reduced walking speed
Reduced walking speed is a characteristic sign of post-stroke gait. The average self-selected gait velocity reported for post-stroke subjects is lower than values for able-bodied subjects ranging in 17 studies from 0.23 m/s (SD = 0.11 m/s) to 0.73 m/s (SD = 0.38 m/s) [193]. For high-functioning subjects, post-stroke gait speeds of 0.78 m/s [90] and 0.95 m/s [107] have been reported. A gait speed of more than 0.80 m/s has been suggested to be necessary for effective community ambulation, e.g. to cross a street in time [78]. Furthermore, a gait speed exceeding 0.83 m/s seems to be needed for pelvic rotation and subsequently for thorax rotation to occur [18]. Consistent with velocity decreases, both stride length and cadence are lower than values for able-bodied subjects. Nakamura and colleagues [181] reported the relationship between cadence and velocity to be linear up to a velocity of about 0.33 m/s and a cadence of about

90 steps/min, with further gains primarily attributable to increases in stride. However, when velocity is higher than about 0.33 m/s, post-stroke subjects at both their preferred and fast gait velocities have cadences equal to or higher than controls at the same velocity, while stride length is equal to or lower than controls [108]. Olney and colleagues performed principal component analysis of kinematic and kinetic variables of gait in chronic post-stroke subjects [194]. They reported that the first principal component was related to gait velocity and accounted for 41% of the variance, and the second component was related to inter-limb asymmetry and accounted for 13% of the variance. Over time after stroke, it has been reported that gait velocity can still increase from 3 months to 12—18 months after stroke, whereas the Fugl-Meyer sensorimotor scale and Barthel functional independence index improved up to 6 weeks and 3 months after stroke, respectively [213]. Thus, gait velocity has been proposed as an outcome measure of locomotor recovery for the post-stroke subjects who can walk faster than 0.33 m/s [212,213].

Temporal and spatial inter-limb asymmetries
Marked temporal and spatial inter-limb asymmetries occur frequently, in 48% to 82% and 44% to 62% of post-stroke subjects, respectively [149,196], with good repeatability of the measures [148,191]. Patterns of temporal asymmetry are often characterized by a shorter stance time and longer swing time of the affected limb, while spatial asymmetry is often characterized by a shorter step length of the unaffected limb, although the opposite occurs too [149,196]. Thus, the decreased unaffected swing time often results in a shorter unaffected step length [97]. The asymmetry ratios (affected/unaffected ratios) of stance time, swing time and step length have significant negative association with self-selected gait velocity [149,196]. Furthermore, it has been reported that post-stroke subjects with greater step length asymmetry at their self-selected velocity walked significantly more slowly at their highest-comfortable velocity [7]. In addition, on the affected side the PS phase is on average more prolonged than the LR phase [145] with very marked difference at the lowest velocities [50]. Gait symmetry, unlike velocity, is not related to age [198]. In a cross-sectional study from weeks to years after stroke, Patterson et al. [197] reported that swing time, stance time, and step symmetry demonstrated a systematic linear trend toward greater asymmetry in groups in the later stages post-stroke, whereas velocity, neurological deficit, and lower-extremity motor impairment demonstrated no significant linear trend. These results suggest that velocity and symmetry measure independent features.

Impaired standing balance control
Impaired standing balance control has been related to spatio-temporal asymmetry during gait in post-stroke subjects [91,149,183]. Lewek et al. [149] found that the Berg Balance Scale was negatively correlated with both step length asymmetry and swing time asymmetry during both comfortable and fast walking. However, weight distribution between limbs during quiet standing was negatively correlated with stance time asymmetry only in fast walking [149]. Hendrickson et al. [91] reported that

increased weight-bearing on the unaffected limb in quiet standing, increased contribution of the unaffected limb to fore-aft balance control, and reduced capacity to bear weight on the affected limb were related to increased asymmetry of swing time, stance time and to a lesser extent of step length. These associations were independent of underlying lower limb impairment. These results strongly suggest that impaired ability of the affected limb to control balance may contribute to gait asymmetry. The authors additionally reported that maximum weight-bearing on the affected limb during standing was only negligibly related to stance time symmetry [91]. Note that evidence for associations between weight-bearing asymmetry and performance on clinical balance tests or falls is weak [113]. This point may explain why prior work has established that practice to improve weight distribution during quiet standing does not transfer to improvements in gait [5,252,258].

Biomechanical description of gait post-stroke

Vertical and anterior-posterior components of the ground reaction force

Vertical and anterior-posterior components of the ground reaction force (GRF) have been described during gait in individuals with stroke. Beneath the affected limb, the vertical GRF is significantly decreased with a one-peak pattern, and the anterior-posterior component displays a higher braking than propulsive impulse, in particular at lower gait velocity in individuals with chronic stroke, using a cane or not [16,115,176]. Walking speed was strongly correlated to affected propulsive impulse and to unaffected braking impulse [16]. The affected propulsive impulse is strongly positively associated with plantarflexor activity, but also negatively associated with leg flexor (tibial anterior, rectus femoris) activity, especially in the severe post-stroke group [243]. Note that foot placement relative to the COM of the body accounts for propulsive and braking impulses in addition to the muscle-force produced [133]. Thus, a reduced posterior position of the affected foot at PS would tend to reduce the affected propulsive impulse, whereas a longer affected step at LR would tend to increase the affected braking impulse [16,133,203]. Further, the foot usually touches the ground either flat or by the forefoot (76—89% of prevalence) with anterior shift of the centre of pressure at initial contact [189] and reduction of its longitudinal course [29,110,189,262]. Thus, plantarflexors, which are active from initial contact [38,85], contribute early to higher ground friction [24], higher ankle negative work [71] and in part to body support and trunk forward deceleration [185]. Wong et al. [262] classified the foot contact pattern in three groups (forefoot-, flatfoot- and heel-initial contact) and reported a high correlation between the foot contact pattern and GRF pattern, gait velocity and Brunnström's recovery stages. Thus, a higher degree of neurological deficiency was associated with higher shift of the centre of pressure trajectory towards the front of the foot at initial contact [262]. In addition, the foot contact pattern categories are highly associated with three separate lower limb joint kinematic subgroups [110] and are early predictors of future functional outcome after rehabilitation in stroke patients [111].

Claw toes

Claw toes occurred in 46% of 39 inpatients in a rehabilitation department with claw toes onset taking place mostly before the end of the third month post-stroke [141]. When claw toes appeared during sitting, this remained present in loading and during gait; when this aspect appeared during standing up and loading it always aggravated during gait, although it always disappeared during rest. The occurrence of claw toes was higher in patients with worse postural control and was significantly correlated to equinus and/or varus foot but not to triceps surae spasticity [141]. Claw toes, which occur more frequently in patients with worse postural control and in more challenging tasks for balance, might have a functional base to increase ground friction as is suggested by claw toes occurring in non-disabled individuals walking on a slippery surface to increase toe grip [74]. Interestingly, the gait mode which is used on slippery surface for balance purposes shares several features with post-stroke gait such as gentle foot flat contact on the ground, toe grip, one-peak of GRF during single-support, and muscle co-activation allowing higher limb stiffness [24,74].

Biomechanical effects of motor control

Merging of modules during gait in post-stroke subjects has biomechanical consequences [38]. Module 1, involving mostly knee extensors and gluteus medius, merged frequently to both module 2, involving mostly plantarflexors, and module 4, involving mostly hamstrings, or merged only to module 2 or rarely only to module 4 [38]. Muscle activation spanned from late swing to almost the end of stance phase for both modules 1 and 4 and over the entire stance phase for module 2. Thus, the frequent extensive co-activation of gluteus medius, knee extensors, plantarflexors and hamstrings during stance likely contributes to body support and higher proximal joints stiffness [83,94] and might explain the one-peak pattern of vertical GRF usually observed post-stroke [16,29,176,262]. On the other hand, poorer generation of propulsion resulted if the early and late stance modules (modules 4 and 2, respectively) were combined, because this indicates interference between the weight acceptance and propulsion subtasks [38]. Interestingly, the second phase of activity of tibialis anterior occurring at terminal swing and loading response in non-disabled subjects [38,85,259] is not observed in affected legs [38,85]. In addition, plantarflexors are frequently not active during the swing phase and always active during the entire stance phase [38,85]. These muscle patterns at terminal swing allow the more frequent flatfoot- or forefoot-initial contact on the ground [29,110,262]. During loading response, tibialis anterior is not required to decelerate dorsiflexion [116] and plantarflexors early contribute to higher ground friction [24], higher deceleration of ankle dorsiflexion [71] and in part to body support and trunk forward deceleration [185]. These early actions of plantarflexors might be related to postural antigravity control. In a more general manner, the motor output complexity based on the number of modules might be strongly influenced by the involuntary postural component of motor control during

gait. This would explain in part that motor output complexity has been shown to be a far superior predictor of walking performance than the Fugl-Meyer assessment [17], which evaluates isolated voluntary movements.

Inter-limb asymmetry in mechanical power

Robust inter-limb asymmetry in mechanical power has been reported during walking in individuals with chronic stroke in a study based on the individual limb method [158]. The unaffected limb produced significantly more positive net mechanical power than the affected limb during all phases of a stride and over a complete stride. In detail, the average net mechanical power was: during PS less positive on affected than unaffected limb; during LR more negative on affected than unaffected limb; during single-support and over a complete stride negative on affected limb and positive on unaffected limb. On the affected limb, peak negative power during LR was inversely correlated to the affected limb's step length. In other words, over a complete stride the affected limb decelerates the COM including less push-off at PS and more braking at LR, while the unaffected limb accelerates the COM including more push-off at PS. One can note that the affected limb generates a high braking impulse also during gait initiation from sitting [79].

The ankle joint

The ankle joint is essential when characterizing gait patterns post-stroke. Using inverse dynamics analysis, a consistent decrease in plantarflexor work and moment impulse during PS has been reported in the affected limb in individuals with chronic stroke [3,31]. Those subjects who walk with symmetric steps were able to compensate for plantarflexor impairment by bilaterally increasing hip flexor moment to increase swing initiation [4] as commonly used to overcome plantarflexor weakness [172,180,219]. Those subjects who walk with longer affected than unaffected steps, on the other hand, typically rely on the unaffected leg with higher plantarflexor and knee extensor moment to forward propulsion [4]. In addition, on the unaffected side, the sagittal ankle power generation is highly correlated with speed whereas on the affected side, it is the sagittal hip power generation, which correlates [116].

Knee joint

The role of the knee joint, anatomically coupled to the ankle joint, is also essential to describe with respect to gait patterns post-stroke. In addition to gait velocity, knee sagittal pattern is a factor that permanently accounts for categories of gait patterns [50,110,116,120,178,194]. However, on the affected leg, knee sagittal pattern is highly correlated to the sagittal pattern of ankle or foot and to a lesser extent to the sagittal pattern of hip or thigh across categories [110,120,178,194]. Important and prolonged affected knee hyperextension during stance (see Fig. 3A) occurs usually in very slow walking, associated with forefoot- or flatfoot-initial contact, prolonged ankle plantarflexion and followed by low peak knee flexion during swing phase [116,178,194,262]. In that case, the anterior position of the ground reaction force vector pushes and maintains the knee in recurvatum [116], which in turn maintains the ankle in plantarflexion. This pattern is

regularly associated with plantarflexor weakness [43,124,178] whereas knee extensor weakness is much more irregular [43,128,179]. Important and prolonged affected knee flexion during stance (see Fig. 3B) occurs usually in very slow or slow walking, associated with forefoot- or flatfoot-initial contact, ankle dorsiflexion during most of the stance phase and followed by inconstant decrease in knee flexion during swing phase [178,194,262]. This pattern appears to be associated with plantarflexor and hip extensor weakness whereas knee extensors are solicited to oppose knee flexion [178]. Moderate or slight affected knee flexion or hyperextension during part of stance (see Fig. 3C) occurs usually in moderate or faster walking, associated with flatfoot- or heel-initial contact, variable ankle plantarflexion and dorsiflexion during stance phase and followed by inconstant moderate decrease in knee flexion during the swing phase [178,194,262]. In these patterns, apart from a lower plantarflexor weakness in the faster walking compared to slow walking, there is no clear tendency regarding muscle strength status [178].

Hip joint

The hip joint, as the more proximal joint linking the leg to the pelvis and trunk, is also essential to describe with regard to gait patterns post-stroke. In the sagittal plane, hip power generation of the affected leg is strongly correlated with walking speed and compensates for plantarflexor weakness [116]. Hip extensors generate forward propulsion during stance [45,186] and hip flexors pull up the leg during the swing phase [4]. In the frontal plane, hip abductor moment is present throughout stance on both sides and contributes to gait stability [69,116]. During the swing phase of the affected leg, hip abductors of the unaffected leg contribute to hip hiking, in which the affected hemipelvis is elevated [30,45] whereas on the affected side hip abductors contribute to hip circumduction [116]. Hip circumduction, which involves hip abduction from toe-off into swing, is more frequently present in forefoot landing and in the knee hyperextension gait pattern and is required for foot clearance [116].

Arm swing and trunk kinematics

Arm swing and trunk kinematics during gait post-stroke is also characterized by asymmetry, where the affected-arm has been reported to deviate significantly more as compared to both the unaffected-arm and to the non-dominant arm in subjects without disabilities [107]. Furthermore, the arm-to-leg swing ratio, typically expressed as a 1:1 relation during walking with normal velocity, switches to a 2:1 frequency relation during slow walking [129], which often is observed in the stroke population. Furthermore, trunk kinematics with respect to intersegmental coordination between thorax and pelvis are associated with walking speed [18,90,137,245]. Subjects with stroke walking slower with shorter and wider steps, as compared to non-disabled controls, used about 15% more thoracic than pelvis rotation, and the coordination between arms and legs was more in-phase than anti-phase [90]. This increased thoracic rotation was interpreted as a compensatory action for the decreased affected-arm swing, aiming for a
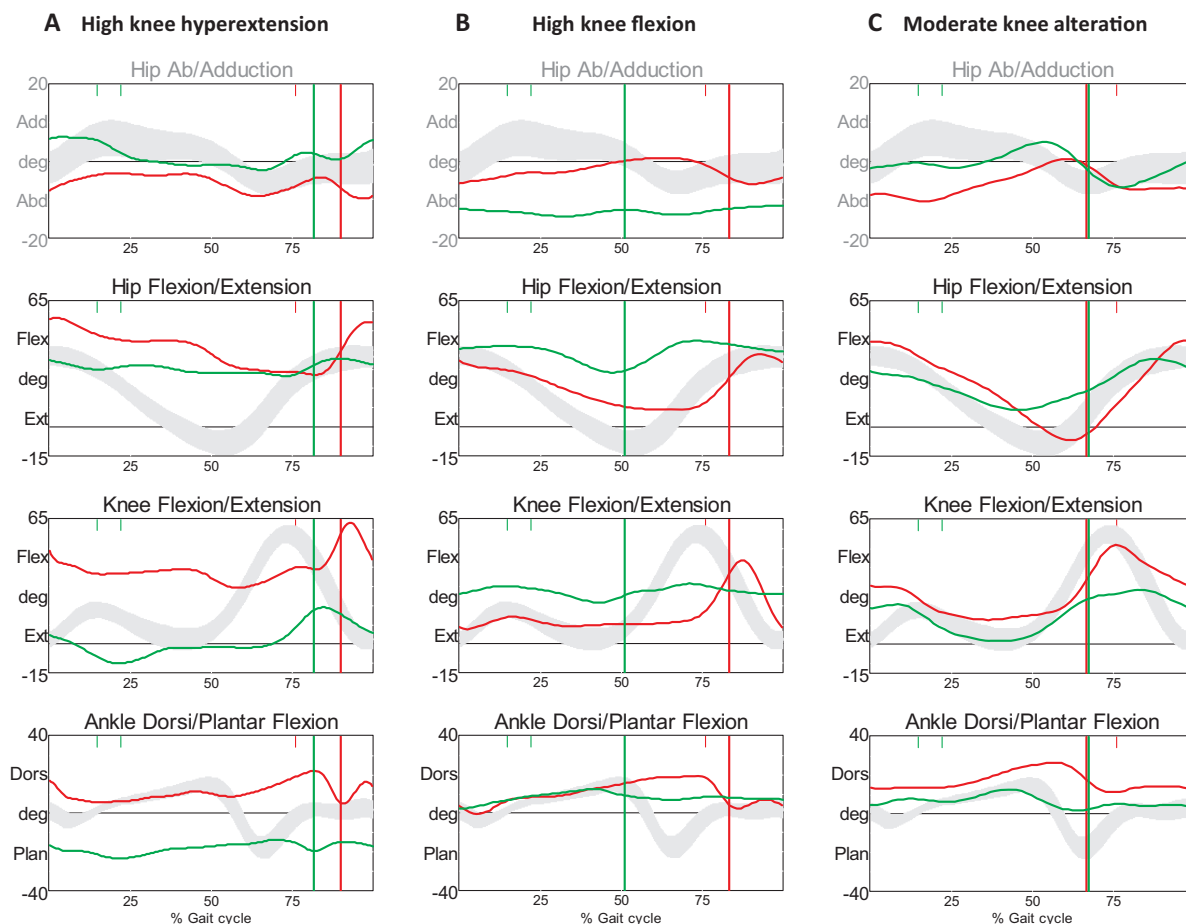
A **High knee hyperextension** **B** **High knee flexion** **C** **Moderate knee alteration**

Figure 3 Examples of lower limbs kinematics representative of the main types of post-stroke knee sagittal patterns. The three examples were taken from individuals with chronic stroke and right hemiparesis, walking spontaneously very slowly with a cane (A), or quite slowly without (B and C). In A, important and prolonged affected knee hyperextension during stance is associated to ankle plantarflexion, hip flexion related to pelvis and trunk forward inclination. On the non-affected left side, a triple flexion contributes to stability and hip abduction during stance contributes to slight right hip hiking during the right swing phase. In B, important and prolonged affected knee flexion during stance is associated with ankle dorsiflexion. During the right swing phase, right hip abduction contributes to slight circumduction and left hip abduction to slight right hip hiking. In C, moderate knee sagittal alteration during stance is associated to slight continuous ankle dorsiflexion, in this case, and hip flexion related in part to pelvis and trunk forward inclination. During the right swing phase, right hip abduction contributes to important circumduction and left hip abduction to important right hip hiking. Flatfoot-initial contact occurs for the affected foot in B, C and almost in A and for the unaffected foot in C (lack of foot motion towards plantarflexion during LR). Colours depicting the right (green), left (red) sides post-stroke and non-disabled individuals (grey, mean ± 1SD). Vertical bars depict the limit between stance and swing phases.

facilitated forward displacement of COM to enhance gait progression.

Gait adaptability

Gait adaptability in subjects with stroke can give insights about neural control. Except for calf muscle contracture in equinus position, ankle sagittal orientation is adjustable during stair ambulation. Thus, community-dwelling post-stroke and non-disabled individuals have at initial contact a similar ankle position either in plantarflexion during stair descent or in more than 10° of dorsiflexion during stair ascent allowing adapted foot landing [190]. Since the ankle sagittal orientation is quite flexible during stair ambulation in post-stroke individuals, the frequent plantarflexion

with forefoot- or flatfoot-initial contact during level walking may have a functional component in its origin. At loading response of post-stroke walking, the early triceps surae activity is associated with dorsiflexion deceleration [71] and trunk forward deceleration [185] as the foot is touching the ground early, either flat or by the forefoot [29,110,262]. This early braking action of triceps might have a non-specific beneficial postural stabilizing effect on the forward progression of gait in individuals with balance challenge or difficulties. This hypothesis would help to explain the usual early triceps action occurring in balance-challenging conditions or pathologies such as when walking on a slippery surface [24,73], in individuals with cerebellar ataxia [162] or cerebral palsy [151]. Interestingly, in children with cerebral palsy wearing negative-heeled shoes compared to barefoot condition, an immediate shift

from ankle plantarflexion to dorsiflexion occurs at initial contact, allowing flatfoot contact on the ground and early braking effect on gait [9]. On the other hand, calf muscle contracture and spastic hypertonia account for equinus gait occurring in part of the individuals with stroke [86,87,120,249].

The split-belt treadmill, until now mostly used in research studies, has two independently controlled belts making it possible to force one foot for example to travel at double the speed of the other. This offers interesting insights into asymmetrical gait and inter-limb coordination in post-stroke persons. This perturbing locomotor environment has been shown to induce locomotor adaptation and after-effects in both non-disabled persons and stroke survivors who are free from cerebellar dysfunction [208,209]. The intact basic capacity for locomotor adaptation after stroke is thereby demonstrated. In particular, when belt speeds are set to augment step length asymmetry, after-effects result in reduced step length asymmetry during overground walking practice [209] with even a long-term effect after repeated split-belt treadmill training [211]. Other setups, such as unilateral step training of the unaffected leg [112] or swing phase perturbation of the leg having the shorter step length [222], also have overground after-effects demonstrating decreasing step length asymmetry. In all the previous setups, balance could be secured by at least light touch on a front handrail [112,211,222] and likely did not interfere with inter-limb adaptation during treadmill training. Interestingly, limping-like walking of non-disabled persons on split-belt treadmill presents major similarities of muscle contributions to COM acceleration in post-stroke persons [105]. This report provides additional insights into the functionally organized pattern of asymmetrical gait in stroke survivors.

Gait rehabilitation strategies post-stroke

Evolution and principles of rehabilitation strategies

Apace with the development of theories of motor control, there has been a change of focus in rehabilitation strategies following neurological disease and injury. Assumptions from motor control theories (such as the reflex theory [229], the hierarchical theory [157], and systems theory [8]) have strongly influenced treatment principles in neurological rehabilitation [95]. One of those theories of motor control, the Dynamic Systems Theory, includes concepts such as 'self-organization of a dynamic system', 'degrees of freedom', 'movement variability', 'non-linearity' and 'stable and unstable movement patterns' [202,238] with suggested potential to understand underlying mechanisms in the CNS and altered movement patterns post-stroke [80].

Based on recent research regarding experience-dependent neural plasticity several treatment principles after brain damage have been suggested [122]. Animal and human studies have demonstrated that the principle of ''Use it or lose it'' is the result of lack of use, e.g. of an extremity, leading to degradation of neural circuits (e.g. [214]), whereas motor skill training, illustrating the principle of ''Use it and improve it'' has been shown to drive restorative neural plasticity with respect to neuromotor processes and to advance motor function regarding action [10].

In a rehabilitation context, the principle of ''Specificity'' implies that skilled movements might lead to enhanced corticospinal activity, whereas practising unskilled movements fails to show improvements [200]. Whether specific kinds of training experience may lead to changes in subsets of neural circuitries and thereby influence the potential for improvements in non-trained activities, according to the principle of ''Transference'', remains an open question [122].

Neural plasticity, often thought of as implying beneficial changes, might lead to maladaptive changes resulting in compensatory movement patterns, ''bad habits'' [122]. This principle of ''Interference'' illustrates how recovery might be impeded and will need to be overcome [27], if possible. There is an on-going discussion with respect to upper limb function post-stroke whether allowing compensatory movement strategies will prevent recovery or not [20,154].

Additionally, for the patient's ability to continue to use the trained function outside of therapy, treatment principles such as ''Repetition'' and ''Intensity'' seem to be critical for augmenting neural plasticity into recovery [122] or perhaps into compensatory movement behaviour [121,146]. However, further results from research studies are warranted in order to draw conclusions about the degree of intensity [42,140].

Recovery and compensation

The discrimination between recovery and compensation is increasingly highlighted in stroke rehabilitation [20,37,125,127,140,146,154,248], even leading to discussions that allowing compensatory strategies early post-stroke might prevent the possibility of true recovery [20,37,154]. The concept of recovery has been defined as ''the re-emergence of movement kinematics similar to those of healthy age-matched controls, resulting from a decrease in impairments, whereas compensation involves the use of the unaffected limb or alternative muscle groups on the affected side to accomplish the task'' [121]. This discrimination between recovery and compensation has most recently been applied to rehabilitation of the upper extremity post-stroke [20,154], but is hereby now proposed to be implemented also in the on-going discussion about optimal gait training models following stroke.

One aspect of recovery is whether gait post-stroke is achieved through automatic processes, i.e. typical movement behaviour, or through more or less compensatory attention-demanding executive control processes [40]. Novel rehabilitation strategies are called for where an up-regulation of CNS circuits for automaticity (see Fig. 1) should be targeted in order to achieve adaptive neuroplastic changes.

Below, gait rehabilitation strategies post-stroke will be described in sections with either a mainly muscular or a task-oriented focus. This division is not to be taken too literally, as most gait rehabilitation strategies nowadays cover both perspectives.

Muscular focus

Neurofacilitation approaches

''Traditional therapies'' in gait rehabilitation post-stroke (original Bobath [11], Brunnström [19], Proprioceptive Neuromuscular Facilitation [PNF] [123]) comprise methods where the muscular dysfunction, such as abnormal muscle tone, flexor and extensor synergies, muscle weakness, and as a consequence dysfunctional movement patterns, has been a main target. None of these so-called neurofacilitation approaches has been able to show better effect on motor performance after stroke than any other treatment method, and permanent improvements in walking performance are lacking [12,126,139,251]. The Bobath concept has continuously been redefined since the 1990s, nowadays incorporating a task-oriented approach. Directions for future research have recently been outlined in order to assess its effectiveness for improvements in stroke rehabilitation [147,246,247].

Functional electrical stimulation (FES)

Functional electrical stimulation (FES), where predetermined frequencies and amplitudes of electrical currents are applied to nerves or myoneural junctions, has been shown to augment muscle-force production, but with fatigue noticed as a problem [12]. According to a Cochrane review there is insufficient robust data, mainly due to methodological differences across studies, for formulating recommendations regarding FES for clinicians [206].

Muscle strength training

Muscle strength training is achieving increasing attention among potential gait rehabilitation strategies [231], as impairment of muscle strength (including endurance and power aspects [234]) is an initial deficit related to a stroke [13]. There is a strong relationship between muscle strength and function [64,188], thus research is now addressing the optimal model for strength training in stroke rehabilitation. In a study by Clark and Patten [39], subjects with stroke practised either concentric or eccentric resistance training for 5 weeks, followed by 3 weeks of gait training, in a high-intensity rehabilitation programme. The greatest gains in neuromuscular activation and power of affected leg muscles were achieved in the group practising eccentric training. However, during and after the gait training many of these improvements in muscular function were lost, in spite of increased walking speed. Furthermore, whereas marked improvements in muscle strength have been reported (e.g. [144,218]), particularly specific to the muscles and actions being trained, results regarding carry-over effects from strength training to functional abilities, such as walking, are inconclusive (for details, see Signal, 2014 [231]).

Task-oriented focus

A task-oriented focus in post-stroke rehabilitation is built upon theories of motor learning and motor control. In interventions, the aims are to prevent impairments, promote task-specific strategies and adapt goal-oriented tasks to continuously shifting environmental circumstances [230].

Repetitive task gait training after stroke has been analysed in a Cochrane review (14 studies; 659 participants) [77]. The results revealed short-term modest improvements regarding lower limb functions, exemplified by walking speed and distance. However, at long-term follow-up (after 6 and 12 months) evidence for maintained improvements was lacking.

In the coming paragraphs, different modes of gait training in stroke rehabilitation with predominantly a task-oriented focus will be described.

Overground physical therapy gait training

Overground physical therapy gait training, with simple aids, such as e.g. parallel bars but without high-technology equipment, is the most common form of clinical practice, at any stage post-stroke [106]. In a Cochrane review [233] (9 studies; 499 participants) it was found that there is insufficient evidence to demonstrate a significant effect of this kind of rehabilitation strategy on overall post-test gait function in subjects in the late phase after stroke. Single measures of gait performance, such as gait speed, time to perform 'Timed Up and Go', and distance walked in six minutes, showed small and short-term statistically significant improvements. However, the clinical relevance of those improvements [81] has to be questioned.

Treadmill training

Treadmill training with body weight support (BWS) (using a harness) enables the patient to practise coordinated stepping:

- during progressively increased demands for postural control;
- with control over gait speed;
- with potential to stimulate normal walking pattern [28,230];
- with reduced oxygen demand [156].

Increased self-selected walking speed under BWS (5—20%) has been demonstrated [99], although this is probably related to strengthening of existing compensatory strategies, rather than to recovery of normal kinetics, taking into account the relative proportion to propulsion from the affected leg [41].

Various supplements to gait training on a treadmill have been investigated. One example is real-time feedback of COM, which was added during a 6-week gait training programme for subjects with chronic stroke. The results showed 10% reduction of vertical COM displacement, 45% increase of affected knee flexion in swing, and 30% diminished energy cost [163].

In spite of the above-mentioned improvements on gait post-stroke the evidence to support treadmill training with or without use of partial BWS is inconclusive [60,227]. Additionally, in a Cochrane Review [170] (44 studies; 2658 participants) it was shown that the intervention was not likely to improve the ability to walk independently.

However, walking speed and walking capacity might be improved.

Electromechanical and robot-assisted gait training

Electromechanical and robot-assisted gait training is increasingly being investigated in stroke studies [93,199,226,257]. Suggested advantages are reduced need for physical input [256] and consequently the possibility of extending training duration [253]. Furthermore, robotic devices have the potential to collect joint kinetic and kinematic data simultaneously with walking [92,250].

Results regarding robot-based therapy as compared to manual therapist-assisted gait training are inconclusive [93,96,167]. Thus, further research with high-quality designed studies is needed in order to determine the efficacy of electromechanical and robot-assisted gait training in order to draw reliable conclusions [169,253].

Ankle-foot orthoses (AFOs)

Ankle-foot orthoses (AFOs) are used in stroke rehabilitation as a strategy to control spasticity, to reduce excessive plantar flexion and to improve balance. Different kinds of AFOs exist, including solid and dynamic models [230]. However, solid AFOs have been shown to result in a reduced use of normal ankle strategy for stability control and to prevent the normal distal-to-proximal sequencing and timing of leg muscles during walking [23]. A recent systematic analysis of the effect of ankle-foot orthoses on gait biomechanics following stroke [244] (20 studies; 314 participants) revealed statistically confirmed positive effects on ankle kinematics, knee kinematics during stance, on weight transfer over the affected leg, and on energy cost. A crossover design was used in most of the studies, with no information about long-term effects.

Virtual reality

In recent years, virtual reality training with interactive video gaming has entered into stroke rehabilitation. Efforts to determine its efficacy have been performed in Cochrane reviews [142,143]. In spite of presenting the user with enriched environment and a high dosage of training, the effects on gait speed and on global motor function post-stroke have shown no significant improvements. Furthermore, concerns have been raised related to the low eligibility rates (26%) and the lack of long-term follow-up studies [143].

Mental practice with motor imagery (MI)

Mental practice with motor imagery (MI), when an individual imagines an action without its physical execution [159], is founded on assumptions that activation of motor brain areas using MI would facilitate brain plasticity (e.g. [161,228]). A few studies have investigated MI for gait rehabilitation following stroke [55,56,67], showing increases in gait speed [55,67] as well as increased step length and single-leg stance time [67]. However, the improvements should be interpreted with caution, as there was no control of how much the participants increased their actual walking over the 6 weeks of treatment [67].

Training with expected transfer effect on gait performance

Some treatment approaches, where motor tasks other than gait have been practised, have shown to have effects on walking ability.

Sit-to-stand training

Sit-to-stand training on either a stable (SSS) or on an unstable (USS) support surface (using balance pads) has shown statistically significant effects on step length on both the affected and non-affected side (USS) as well as on distance walked during the 6-minute walk test (6MWT) (SSS and USS) [179]. The clinical relevance of improvements gained, however, was not discussed.

Mechanical horse-riding

Mechanical horse-riding also has reported effects on gait speed [119]. The authors discuss the possibility of enhanced trunk motor skills and balance through this kind of training.

Fitness-training programmes

Physical fitness is often reduced in individuals post-stroke and therefore fitness-training programmes, including cardiorespiratory training, resistance training and/or mixed training, have been implemented [221]. Gait speed (maximum or preferred) and gait capacity (6MWT) improved in most of the reported studies. However, many of the studies included interventions that were walking-specific [220].

Standing exercises on a supplementary tilt table

Standing exercises on a supplementary tilt table have been used to investigate the effect of different tilt table applications on muscle strength and on gait parameters in post-stroke subjects [117]. The participants in the group performing progressive task-oriented training showed the greatest improvements regarding muscle strength, gait velocity, cadence, stride length, reduction in double limb support period as well as on gait asymmetry.

Synthesis

During typical walking, the CNS simultaneously achieves body support, balance control, and forward progression [165,260]. Adaptation allows adjustment of gait patterns to the environment [195,210] and/or to compensate for body anatomical and function alterations [224]. Motor control is organized on a task-oriented basis using a common set of few modules (or muscle synergies) which can be recruited by different neural pathways for voluntary or automatic motor behaviours to achieve the different biomechanical functions mentioned above [36,38,187,217]. Brain stroke provokes disruption of descending neural pathways inducing hemiparesis [44] but usually no direct lesion of the structures in brainstem [21,49,118,131,207] and cerebellum [25,241] that are involved in automatic processes controlling gait. In normal gait, these structures and the spinal cord are essential for the task-dependent motor modular organisation associating rhythmic patterns to the fundamental postural support and balance control (see Fig. 1). In individuals with stroke, when an

upright posture is achieved, while standing or walking, it is usually characterized by an asymmetric pattern involving more the unaffected side for body support and balance [91,115,149,176,182]. In addition during walking, the usual inter-limb spatio-temporal and kinetic asymmetries have been related to both impaired standing balance control [91,149,183] and hemiparesis [158,243]. Post-stroke gait remains centrally highly organized (task-oriented modular organisation [36,38,217] and planar law of intersegmental coordination [34]) with ecologic adaptability (stair ambulation [190], walking on split-belt treadmill [208,209]). However, merging of existing modules on the affected side for support and balance tasks leads to poor walking performance and limited mobility capability [38,83,94,217]. Lower limb muscle co-activation has been mainly related to spastic mechanisms [86] whereas its neural origin as a compensatory strategy providing mechanical stability by stiffening joints is increasingly considered [33,136,215]. Neural adaptations during gait are mainly automatic under the influence of the cerebellar controller but also involve intentional voluntary processes [40,177,237]. In post-stroke gait, the asymmetric pattern involving more the unaffected side for support, balance and forward progression is increasingly suggested to be mainly a neural adaptation to the initial paretic affected side [38,83,91,94,149,204,217]. However, this asymmetric pattern of motor output would tend to be automatically maintained or reinforced with time [197] due to the maintained neural disuse of the affected side. In more detail, postural asymmetry, instability and weakness on the affected side, in particular long-lasting plantarflexor weakness, contribute to slow walking, with asymmetric kinetic, kinematic as well as step-to-step transition patterns [3,16,31,91,158,180,262]. Unfortunately, this asymmetric motor behaviour has functional limitations leading to high occurrence of falls while walking. That is why recovering a symmetric motor behaviour is an important goal for motor activities, including walking, in post-stroke individuals.

Various rehabilitations strategies are regularly used or in development, targeting muscle activity, postural and gait tasks, using more or less high-technology equipment [12,80,95,143,169,230,231]. Reduced walking speed often improves with time and with various rehabilitation strategies [39,77,99,233] but the asymmetric postural behaviour during walking is often reinforced [41,91], maintained, or only transitorily decreased [222], thus the compensatory gait strategies most often remain. This asymmetric compensatory postural behaviour appears to be robust, driven by support and balance tasks maintaining the predominant use of the unaffected side over the initially impaired affected side [16,91,149,158,183]. True recovery of typical-like motor patterns would need a neural reorganisation that allows equal use of both affected and non-affected sides in the priority postural antigravity function to control the COM for balance safety during all postural and locomotor tasks. The postural and gait improvements obtained even transitorily by some rehabilitation setups [91,112,113,117,119,179,211,222] provide further insights for suggestions to adapt rehabilitation to neural function in post-stroke subjects. For example, training in particular postural designs [117,119,179] could transfer beneficial effects on gait performance. Bipedal locomotion is very challenging in regard to balance control. Post-stroke rehabilitation including affected muscle strengthening and often stretching would need first to correct the asymmetric pattern of the permanently on-going postural antigravity control in various motor tasks, which are less challenging for balance control than overground gait, before allowing the latter. Since self-postural control is an automatic process, various setups of exercise may be designed and associated with safely and automatically involving the affected side more in support and balance. The subject would preferably achieve these exercises by autonomously controlling his own support and balance. In particular, part of the exercises may be designed to automatically involve the affected side for support and balance while achieving a voluntary movement on the unaffected side. In such postural tasks, proprioceptive sensory input from affected segments are capitalized to support and balance the body by exploiting postural automatic processes that also would benefit gait secondarily automatically. Thus walking, like other motor tasks, would benefit from higher involvement of the affected side in postural function.

Recently, increasing knowledge about pathophysiology of motor function in typical and post-stroke individuals provides a better understanding of the insufficient results of rehabilitation strategies. Further research on therapeutics in subjects post-stroke is needed to provide if possible permanent true recovery.

Disclosure of interest

The authors declare that they have no competing interest.

References

[1] Ahn YH, Ahn SH, Kim H, Hong JH, Jang SH. Can stroke patients walk after complete lateral corticospinal tract injury of the affected hemisphere? Neuroreport 2006;17(10):987—90.

[2] Alguren B, Lundgren-Nilsson A, Sunnerhagen KS. Functioning of stroke survivors — a validation of the ICF core set for stroke in Sweden. Disabil Rehabil 2010;32(7):551—9.

[3] Allen JL, Kautz SA, Neptune RR. Step length asymmetry is representative of compensatory mechanisms used in post-stroke hemiparetic walking. Gait Posture 2011;33(4):538—43.

[4] Allen JL, Neptune RR. Three-dimensional modular control of human walking. J Biomech 2012;45(12):2157—63.

[5] Awad LN, Reisman DS, Binder-Macleod SA. Do improvements in balance relate to improvements in long-distance walking function after stroke? Stroke Res Treat 2014;2014:646230.

[6] Batchelor FA, Mackintosh SF, Said CM, Hill KD. Falls after stroke. Int J Stroke 2012;7(6):482—90.

[7] Beaman CB, Peterson CL, Neptune RR, Kautz SA. Differences in self-selected and fastest-comfortable walking in post-stroke hemiparetic persons. Gait Posture 2010;31(3):311—6.

[8] Bernstein N. The coordination and regulation of movements. Oxford, UK: Pergamon Press Ltd; 1967.

[9] Beyaert C, Caudron S, Avila F, Billon C, Paysant J. Use of negative heel shoes and pathophysiology of equinus gait in children with cerebral palsy. Gait Posture 2014;39(Suppl. 1(0)):S59—60.

[10] Biernaskie J, Corbett D. Enriched rehabilitative training promotes improved forelimb motor function and enhanced dendritic growth after focal ischemic injury. J Neurosci 2001;21(14):5272—80.

[11] Bobath B. Adult hemiplegia: evaluation and treatment. 3rd edn London: Butterworth-Heinemann; 1990.

[12] Bogey R, Hornby GT. Gait training strategies utilized in post-stroke rehabilitation: are we really making a difference? Top Stroke Rehabil 2007;14(6):1—8.

[13] Bohannon RW. Muscle strength and muscle training after stroke. J Rehabil Med 2007;39(1):14—20.

[14] Borghese NA, Bianchi L, Lacquaniti F. Kinematic determinants of human locomotion. J Physio 1996;494(Pt 3):863—79.

[15] Bove M, Trompetto C, Abbruzzese G, Schieppati M. The posture-related interaction between Ia-afferent and descending input on the spinal reflex excitability in humans. Neurosci Lett 2006;397(3):301—6.

[16] Bowden MG, Balasubramanian CK, Neptune RR, Kautz SA. Anterior-posterior ground reaction forces as a measure of paretic leg contribution in hemiparetic walking. Stroke 2006;37(3):872—6.

[17] Bowden MG, Clark DJ, Kautz SA. Evaluation of abnormal synergy patterns poststroke: relationship of the Fugl-Meyer Assessment to hemiparetic locomotion. Neurorehabil Neural Repair 2010;24(4):328—37.

[18] Bruijn SM, Meijer OG, van Dieen JH, Kingma I, Lamoth CJ. Coordination of leg swing, thorax rotations, and pelvis rotations during gait: the organisation of total body angular momentum. Gait Posture 2008;27(3):455—62.

[19] Brunnström S. Movement therapy in hemiplegia: a neuropsychological approach. New York: Harper and Row; 1970.

[20] Buma F, Kwakkel G, Ramsey N. Understanding upper limb recovery after stroke. Restor Neurol Neurosci 2013;31(6):707—22.

[21] Burger KM, Tuhrim S, Naidich TP. Brainstem vascular stroke anatomy. Neuroimaging Clin N Am 2005;15(2):297—324 [x].

[22] Burne JA, Carleton VL, O'Dwyer NJ. The spasticity paradox: movement disorder or disorder of resting limbs? J Neurol Neurosurg Psychiatry 2005;76(1):47—54.

[23] Burtner PA, Woollacott MH, Qualls C. Stance balance control with orthoses in a group of children with spastic cerebral palsy. Dev Med Child Neurol 1999;41(11):748—57.

[24] Cappellini G, Ivanenko YP, Dominici N, Poppele RE, Lacquaniti F. Motor patterns during walking on a slippery walkway. J Neurophysiol 2010;103(2):746—60.

[25] Carrera E, Maeder-Ingvar M, Rossetti AO, Devuyst G, Bogousslavsky J, Lausanne Stroke R. Trends in risk factors, patterns and causes in hospitalized strokes over 25 years: The Lausanne Stroke Registry. Cerebrovasc Dis 2007;24(1):97—103.

[26] Cavagna GA, Margaria R. Mechanics of walking. J Appl Physiol 1966;21(1):271—8.

[27] Celnik PA, Cohen LG. Modulation of motor function and cortical plasticity in health and disease. Restor Neurol Neurosci 2004;22(3—5):261—8.

[28] Cernak K, Stevens V, Price R, Shumway-Cook A. Locomotor training using body-weight support on a treadmill in conjunction with ongoing physical therapy in a child with severe cerebellar ataxia. Phys ther 2008;88(1):88—97.

[29] Chen CY, Hong PW, Chen CL, Chou SW, Wu CY, Cheng PT, et al. Ground reaction force patterns in stroke patients with various degrees of motor recovery determined by plantar dynamic analysis. Chang Gung Med J 2007;30(1):62—72.

[30] Chen G, Patten C, Kothari DH, Zajac FE. Gait differences between individuals with post-stroke hemiparesis and non-disabled controls at matched speeds. Gait Posture 2005;22(1):51—6.

[31] Chen G, Patten C. Joint moment work during the stance-to-swing transition in hemiparetic subjects. J Biomech 2008;41(4):877—83.

[32] Cheung VC, Turolla A, Agostini M, Silvoni S, Bennis C, Kasi P, et al. Muscle synergy patterns as physiological markers of motor cortical damage. Proc Natl Acad Sci U S A 2012;109(36):14652—6.

[33] Chow JW, Yablon SA, Stokic DS. Coactivation of ankle muscles during stance phase of gait in patients with lower limb hypertonia after acquired brain injury. Clin Neurophysiol 2012;123(8):1599—605.

[34] Chow JW, Stokic DS. Intersegmental coordination of gait after hemorrhagic stroke. Exp Brain Res 2015;233(1):125—35.

[35] Chvatal SA, Ting LH. Voluntary and reactive recruitment of locomotor muscle synergies during perturbed walking. J Neurosci 2012;32(35):12237—50.

[36] Chvatal SA, Ting LH. Common muscle synergies for balance and walking. Front Comput Neurosci 2013;7:48.

[37] Cirstea MC, Levin MF. Compensatory strategies for reaching in stroke. Brain 2000;123(Pt 5):940—53.

[38] Clark DJ, Ting LH, Zajac FE, Neptune RR, Kautz SA. Merging of healthy motor modules predicts reduced locomotor performance and muscle coordination complexity post-stroke. J Neurophysiol 2010;103(2):844—57.

[39] Clark DJ, Patten C. Eccentric versus concentric resistance training to enhance neuromuscular activation and walking speed following stroke. Neurorehabil Neural Repair 2013;27(4):335—44.

[40] Clark DJ. Automaticity of walking: functional significance, mechanisms, measurement and rehabilitation strategies. Front Hum Neurosci 2015;9:246.

[41] Combs SA, Dugan EL, Ozimek EN, Curtis AB. Effects of body-weight supported treadmill training on kinetic symmetry in persons with chronic stroke. Clin Biomech (Bristol, Avon) 2012;27(9):887—92.

[42] Cooke EV, Mares K, Clark A, Tallis RC, Pomeroy VM. The effects of increased dose of exercise-based therapies to enhance motor recovery after stroke: a systematic review and meta-analysis. BMC Med 2010;8:60.

[43] Cooper A, Alghamdi GA, Alghamdi MA, Altowaijri A, Richardson S. The relationship of lower limb muscle strength and knee joint hyperextension during the stance phase of gait in hemiparetic stroke patients. Physiother Res Int 2012;17(3):150—6.

[44] Corbetta M, Ramsey L, Callejas A, Baldassarre A, Hacker CD, Siegel JS, et al. Common behavioral clusters and subcortical anatomy in stroke. Neuron 2015;85(5):927—41.

[45] Cruz TH, Lewek MD, Dhaher YY. Biomechanical impairments and gait adaptations post-stroke: multi-factorial associations. J Biomech 2009;42(11):1673—7.

[46] Cullen KE. The vestibular system: multimodal integration and encoding of self-motion for motor control. Trends Neurosci 2012;35(3):185—96.

[47] Danner SM, Hofstoetter US, Freundl B, Binder H, Mayr W, Rattay F, et al. Human spinal locomotor control is based on flexibly organized burst generators. Brain 2015;138(Pt 3):577—88.

[48] de Freitas GR, Devuyst G, van Melle G, Bogousslavsky J. Motor strokes sparing the leg: different lesions and causes. Arch Neurol 2000;57(4):513—8.

[49] De Mendivil AO, Alcalá-Galiano A, Ochoa M, Salvador E, Millán JM. Brainstem stroke: anatomy, clinical and radiological findings. Semin Ultrasound CT MRI 2013;34(2):131—41.

[50] De Quervain IA, Simon SR, Leurgans S, Pease WS, McAllister D. Gait pattern in the early recovery period after stroke. J Bone Joint Surg Am 1996;78(10):1506—14.

[51] De Zeeuw CI, Hoogenraad CC, Koekkoek SKE, Ruigrok TJH, Galjart N, Simpson JI. Microcircuitry and function of the inferior olive. Trends Neurosci 1998;21(9):391—400.

[52] den Otter AR, Geurts ACH, Mulder T, Duysens J. Speed related changes in muscle activity from normal to very slow walking speeds. Gait Posture 2004;19(3):270—8.

[53] Den Otter AR, Geurts AC, Mulder T, Duysens J. Gait recovery is not associated with changes in the temporal patterning of

muscle activity during treadmill walking in patients with post-stroke hemiparesis. Clin Neurophysiol 2006;117(1):4—15.

[54] Denny-Brown D. The cerebral control of movement. Liverpool University Press; 1966. p. 222.

[55] Dickstein R, Dunsky A, Marcovitz E. Motor imagery for gait rehabilitation in post-stroke hemiparesis. Phys Ther 2004;84(12):1167—77.

[56] Dickstein R, Deutsch JE. Motor imagery in physical therapist practice. Phys Ther 2007;87(7):942—53.

[57] Dietz V, Sinkjaer T. Spastic movement disorder: impaired reflex function and altered muscle mechanics. Lancet Neurol 2007;6(8):725—33.

[58] Dimitrijevic MR, Gerasimenko Y, Pinter MM. Evidence for a spinal central pattern generator in humans. Ann N Y Acad Sci 1998;860:360—76.

[59] Dobkin BH. Clinical practice. Rehabilitation after stroke. N Engl J Med 2005;352(16):1677—84.

[60] Dobkin BH, Nadeau SE, Behrman AL, Wu SS, Rose DK, Bowden M, et al. Prediction of responders for outcome measures of locomotor Experience Applied Post Stroke trial. J Rehabil Res Dev 2014;51(1):39—50.

[61] Dominici N, Ivanenko YP, Cappellini G, d'Avella A, Mondi V, Cicchese M, et al. Locomotor primitives in newborn babies and their development. Science 2011;334(6058):997—9.

[62] Donelan JM, Kram R, Kuo AD. Simultaneous positive and negative external mechanical work in human walking. J Biomech 2002;35(1):117—24.

[63] Donelan JM, Kram R, Kuo AD. Mechanical work for step-to-step transitions is a major determinant of the metabolic cost of human walking. J Ex Biol 2002;205(Pt 23):3717—27.

[64] Dorsch S, Ada L, Canning CG, Al-Zharani M, Dean C. The strength of the ankle dorsiflexors has a significant contribution to walking speed in people who can walk independently after stroke: an observational study. Arch Phys Med Rehabil 2012;93(6):1072—6.

[65] Drew T, Prentice S, Schepens B. Cortical and brainstem control of locomotion. Prog Brain Res 2004;143:251—61.

[66] Drew T, Kalaska J, Krouchev N. Muscle synergies during locomotion in the cat: a model for motor cortex control. J Physiol 2008;586(5):1239—45.

[67] Dunsky A, Dickstein R, Marcovitz E, Levy S, Deutsch JE. Home-based motor imagery training for gait rehabilitation of people with chronic poststroke hemiparesis. Arch Phys Med Rehabil 2008;89(8):1580—8.

[68] Duysens J, De Groote F, Jonkers I. The flexion synergy, mother of all synergies and father of new models of gait. Front Comput Neurosci 2013;7:14.

[69] Eng JJ, Winter DA. Kinetic analysis of the lower limbs during walking: what information can be gained from a three-dimensional model? J Biomech 1995;28(6):753—8.

[70] Fallah-Yakhdani HR, Abbasi-Bafghi H, Meijer OG, Bruijn SM, van den Dikkenberg N, Benedetti MG, et al. Determinants of co-contraction during walking before and after arthroplasty for knee osteoarthritis. Clin Biomech (Bristol, Avon) 2012;27(5):485—94.

[71] Farris DJ, Hampton A, Lewek MD, Sawicki GS. Revisiting the mechanics and energetics of walking in individuals with chronic hemiparesis following stroke: from individual limbs to lower limb joints. J Neuroeng Rehabil 2015;12:24.

[72] Feng J, Pierce R, Do KP, Aiona M. Motion of the center of mass in children with spastic hemiplegia: balance, energy transfer, and work performed by the affected leg vs. the unaffected leg. Gait Posture 2014;39(1):570—6.

[73] Fong DT, Hong Y, Li JX. Lower-extremity gait kinematics on slippery surfaces in construction worksites. Med Sci Sports Exerc 2005;37(3):447—54.

[74] Fong DT, Mao DW, Li JX, Hong Y. Greater toe grip and gentler heel strike are the strategies to adapt to slippery surface. J Biomech 2008;41(4):838—44.

[75] Forbes PA, Siegmund GP, Schouten AC, Blouin JS. Task, muscle and frequency dependent vestibular control of posture. Front Integr Neurosci 2014;8:94.

[76] Fox EJ, Tester NJ, Kautz SA, Howland DR, Clark DJ, Garvan C, et al. Modular control of varied locomotor tasks in children with incomplete spinal cord injuries. J Neurophysiol 2013;110(6):1415—25.

[77] French B, Thomas LH, Leathley MJ, Sutton CJ, McAdam J, Forster A, et al. Repetitive task training for improving functional ability after stroke. Cochrane Database Syst Rev 2007;4:Cd006073.

[78] Fritz S, Lusardi M. White paper: ''walking speed: the sixth vital sign''. J Geriatr Phys Ther 2009;32(2):46—9.

[79] Frykberg GE, Thierfelder T, Aberg AC, Halvorsen K, Borg J, Hirschfeld H. Impact of stroke on anterior-posterior force generation prior to seat-off during sit-to-walk. Gait Posture 2012;35(1):56—60.

[80] Frykberg GE, Vasa R. Neuroplasticity in action post-stroke: challenges for physiotherapists. Eur J Physiother 2015;17(2):56—65.

[81] Fulk GD, Ludwig M, Dunning K, Golden S, Boyne P, West T. Estimating clinically important change in gait speed in people with stroke undergoing outpatient rehabilitation. J Neurol Phys Ther 2011;35(2):82—9.

[82] Gale SD, Pearson CM. Neuroimaging predictors of stroke outcome: implications for neurorehabilitation. NeuroRehabilitation 2012;31(3):331—44.

[83] Garland SJ, Gray VL, Knorr S. Muscle activation patterns and postural control following stroke. Motor Control 2009;13(4):387—411.

[84] Giszter SF, Hart CB. Motor primitives and synergies in the spinal cord and after injury — the current state of play. Ann N Y Acad Sci 2013;1279:114—26.

[85] Gizzi L, Nielsen JF, Felici F, Ivanenko YP, Farina D. Impulses of activation but not motor modules are preserved in the locomotion of subacute stroke patients. J Neurophysiol 2011;106(1):202—10.

[86] Gracies JM. Pathophysiology of spastic paresis. II: emergence of muscle overactivity. Muscle Nerve 2005;31(5):552—71.

[87] Gracies JM. Pathophysiology of spastic paresis. I: paresis and soft tissue changes. Muscle Nerve 2005;31(5):535—51.

[88] Grasso R, Zago M, Lacquaniti F. Interactions between posture and locomotion: motor patterns in humans walking with bent posture versus erect posture. J Neurophysiol 2000;83(1):288—300.

[89] Guertin PA. The mammalian central pattern generator for locomotion. Brain Res Rev 2009;62(1):45—56.

[90] Hacmon RR, Krasovsky T, Lamontagne A, Levin MF. Deficits in intersegmental trunk coordination during walking are related to clinical balance and gait function in chronic stroke. J Neurol Phys Ther 2012;36(4):173—81.

[91] Hendrickson J, Patterson KK, Inness EL, McIlroy WE, Mansfield A. Relationship between asymmetry of quiet standing balance control and walking post-stroke. Gait Posture 2014;39(1):177—81.

[92] Hidler J, Nichols D, Pelliccio M, Brady K. Advances in the understanding and treatment of stroke impairment using robotic devices. Top Stroke Rehabil 2005;12(2):22—35.

[93] Hidler J, Nichols D, Pelliccio M, Brady K, Campbell DD, Kahn JH, et al. Multicenter randomized clinical trial evaluating the effectiveness of the Lokomat in subacute stroke. Neurorehabil Neural Repair 2009;23(1):5—13.

[94] Higginson JS, Zajac FE, Neptune RR, Kautz SA, Delp SL. Muscle contributions to support during gait in an individual with post-stroke hemiparesis. J Biomech 2006;39(10):1769–77.

[95] Horak F. Assumptions underlying motor control for neurologic rehabilitation. In: Lister M, editor. Contemporary management of motor control problems. Foundations for physical therapy. Alexandris, VA: APTA: II Step Conference; 1991. p. 11–27.

[96] Hornby TG, Campbell DD, Kahn JH, Demott T, Moore JL, Roth HR. Enhanced gait-related improvements after therapist- versus robotic-assisted locomotor training in subjects with chronic stroke: a randomized controlled study. Stroke 2008;39(6):1786–92.

[97] Hsu AL, Tang PF, Jan MH. Analysis of impairments influencing gait velocity and asymmetry of hemiplegic patients after mild to moderate stroke. Arch Phys Med Rehabil 2003;84(8):1185–93.

[98] Hsu WL, Scholz JP, Schoner G, Jeka JJ, Kiemel T. Control and estimation of posture during quiet stance depends on multi-joint coordination. J Neurophysiol 2007;97(4):3024–35.

[99] Hurt CP, Burgess JK, Brown DA. Limb contribution to increased self-selected walking speeds during body weight support in individuals poststroke. Gait Posture 2015;41(3):857–9.

[100] Hyndman D, Ashburn A, Stack E. Fall events among people with stroke living in the community: circumstances of falls and characteristics of fallers. Arch Phys Med Rehabil 2002;83(2):165–70.

[101] Ivanenko YP, Poppele RE, Lacquaniti F. Five basic muscle activation patterns account for muscle activity during human locomotion. J Physiol 2004;556(Pt 1):267–82.

[102] Ivanenko YP, Poppele RE, Lacquaniti F. Motor control programs and walking. Neuroscientist 2006;12(4):339–48.

[103] Ivanenko YP, d'Avella A, Poppele RE, Lacquaniti F. On the origin of planar covariation of elevation angles during human locomotion. J Neurophysiol 2008;99(4):1890–8.

[104] Jang SH, Chang CH, Lee J, Kim CS, Seo JP, Yeo SS. Functional role of the corticoreticular pathway in chronic stroke patients. Stroke 2013;44(4):1099–104.

[105] Jansen K, De Groote F, Duysens J, Jonkers I. Muscle contributions to center of mass acceleration adapt to asymmetric walking in healthy subjects. Gait Posture 2013;38(4):739–44.

[106] Jette DU, Latham NK, Smout RJ, Gassaway J, Slavin MD, Horn SD. Physical therapy interventions for patients with stroke in inpatient rehabilitation facilities. Phys Ther 2005;85(3):238–48.

[107] Johansson GM, Frykberg GE, Grip H, Brostrom EW, Hager CK. Assessment of arm movements during gait in stroke — the Arm Posture Score. Gait Posture 2014;40(4):549–55.

[108] Jonsdottir J, Recalcati M, Rabuffetti M, Casiraghi A, Boccardi S, Ferrarin M. Functional resources to increase gait speed in people with stroke: strategies adopted compared to healthy controls. Gait Posture 2009;29(3):355–9.

[109] Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Recovery of walking function in stroke patients: the Copenhagen Stroke Study. Arch Phys Med Rehabil 1995;76(1):27–32.

[110] Kaczmarczyk K, Wit A, Krawczyk M, Zaborski J. Gait classification in post-stroke patients using artificial neural networks. Gait Posture 2009;30(2):207–10.

[111] Kaczmarczyk K, Wit A, Krawczyk M, Zaborski J, Gajewski J. Associations between gait patterns, brain lesion factors and functional recovery in stroke patients. Gait Posture 2012;35(2):214–7.

[112] Kahn JH, Hornby TG. Rapid and long-term adaptations in gait symmetry following unilateral step training in people with hemiparesis. Phys Ther 2009;89(5):474–83.

[113] Kamphuis JF, de Kam D, Geurts AC, Weerdesteyn V. Is weight-bearing asymmetry associated with postural instability after stroke? A systematic review. Stroke Res Treat 2013;2013:692137.

[114] Kang HG, Quach L, Li W, Lipsitz LA. Stiffness control of balance during dual task and prospective falls in older adults: the MOBILIZE Boston Study. Gait Posture 2013;38(4):757–63.

[115] Kim CM, Eng JJ. Symmetry in vertical ground reaction force is accompanied by symmetry in temporal but not distance variables of gait in persons with stroke. Gait Posture 2003;18(1):23–8.

[116] Kim CM, Eng JJ. Magnitude and pattern of 3D kinematic and kinetic gait profiles in persons with stroke: relationship to walking speed. Gait Posture 2004;20(2):140–6.

[117] Kim CY, Lee JS, Kim HD, Kim JS. The effect of progressive task-oriented training on a supplementary tilt table on lower extremity muscle strength and gait recovery in patients with hemiplegic stroke. Gait Posture 2015;41(2):425–30.

[118] Kim JS. Pure lateral medullary infarction: clinical-radiological correlation of 130 acute, consecutive patients. Brain 2003;126(Pt 8):1864–72.

[119] Kim YN, Lee DK. Effects of horse-riding exercise on balance, gait, and activities of daily living in stroke patients. J Phys Ther Sci 2015;27(3):607–9.

[120] Kinsella S, Moran K. Gait pattern categorization of stroke participants with equinus deformity of the foot. Gait Posture 2008;27(1):144–51.

[121] Kitago T, Liang J, Huang VS, Hayes S, Simon P, Tenteromano L, et al. Improvement after constraint-induced movement therapy: recovery of normal motor control or task-specific compensation? Neurorehabil Neural Repair 2013;27(2):99–109.

[122] Kleim JA, Jones TA. Principles of experience-dependent neural plasticity: implications for rehabilitation after brain damage. J Speech Lang Hear Res 2008;51(1):S225–39.

[123] Knott M, Voss D. Proprioceptive neuromuscular facilitation. New York, NY: Harper & Row Publishers Inc; 1968.

[124] Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiparetic patients. Brain 1979;102(2):405–30.

[125] Kollen B, van de Port I, Lindeman E, Twisk J, Kwakkel G. Predicting improvement in gait after stroke: a longitudinal prospective study. Stroke 2005;36(12):2676–80.

[126] Kollen BJ, Lennon S, Lyons B, Wheatley-Smith L, Scheper M, Buurke JH, et al. The effectiveness of the Bobath concept in stroke rehabilitation: what is the evidence? Stroke 2009;40(4):e89–97.

[127] Krakauer JW, Carmichael ST, Corbett D, Wittenberg GF. Getting neurorehabilitation right: what can be learned from animal models? Neurorehabil Neural Repair 2012;26(8):923–31.

[128] Krasovsky T, Lamontagne A, Feldman AG, Levin MF. Reduced gait stability in high-functioning poststroke individuals. J Neurophysiol 2013;109(1):77–88.

[129] Kubo M, Wagenaar RC, Saltzman E, Holt KG. Biomechanical mechanism for transitions in phase and frequency of arm and leg swing during walking. Biol Cybern 2004;91(2):91–8.

[130] Kuker W, Weise J, Krapf H, Schmidt F, Friese S, Bahr M. MRI characteristics of acute and subacute brainstem and thalamic infarctions: value of T2- and diffusion-weighted sequences. J Neurol 2002;249(1):33–42.

[131] Kumral E, Bayulkem G, Evyapan D. Clinical spectrum of pontine infarction. Clinical-MRI correlations. J Neurol 2002;249(12):1659–70.

[132] Kuo AD, Donelan JM, Ruina A. Energetic consequences of walking like an inverted pendulum: step-to-step transitions. Exerc Sport Sci Rev 2005;33(2):88–97.

[133] Kuo AD, Donelan JM. Dynamic principles of gait and their clinical implications. Phys Ther 2010;90(2):157–74.

[134] Lacquaniti F, Ivanenko YP, Zago M. Kinematic control of walking. Arch Ital Biol 2002;140(4):263—72.

[135] Lacquaniti F, Ivanenko YP, Zago M. Patterned control of human locomotion. J Physiol 2012;590(Pt 10):2189—99.

[136] Lamontagne A, Richards CL, Malouin F. Coactivation during gait as an adaptive behavior after stroke. J Electromyogr Kinesiol 2000;10(6):407—15.

[137] Lamoth CJ, Beek PJ, Meijer OG. Pelvis-thorax coordination in the transverse plane during gait. Gait Posture 2002;16(2):101—14.

[138] Lance J. Symposium synopsis. In: Feldman R, Young R, Koella W, editors. Spasticity: disordered motor control. Chicago: Year Book Medical Publishers; 1980. p. 485—95.

[139] Langhammer B, Stanghelle JK. Can physiotherapy after stroke based on the Bobath concept result in improved quality of movement compared to the motor relearning programme. Physiother Res Int 2011;16(2):69—80.

[140] Langhorne P, Bernhardt J, Kwakkel G. Stroke rehabilitation. Lancet 2011;377(9778):1693—702.

[141] Laurent G, Valentini F, Loiseau K, Hennebelle D, Robain G. Claw toes in hemiplegic patients after stroke. Ann Phys Rehabil Med 2010;53(2):77—85.

[142] Laver KE, George S, Thomas S, Deutsch JE, Crotty M. Virtual reality for stroke rehabilitation. Cochrane Database Syst Rev 2011;9:Cd008349.

[143] Laver KE, George S, Thomas S, Deutsch JE, Crotty M. Virtual reality for stroke rehabilitation. Cochrane Database Syst Rev 2015;2:CD008349.

[144] Lee MJ, Kilbreath SL, Singh MF, Zeman B, Davis GM. Effect of progressive resistance training on muscle performance after chronic stroke. Med Sci Sports Exerc 2010;42(1):23—34.

[145] Lehmann JF, Condon SM, Price R, deLateur BJ. Gait abnormalities in hemiplegia: their correction by ankle-foot orthoses. Arch Phys Med Rehabil 1987;68(11):763—71.

[146] Levin MF, Kleim JA, Wolf SL. What do motor ''recovery'' and ''compensation'' mean in patients following stroke? Neurorehabil Neural Repair 2009;23(4):313—9.

[147] Levin MF, Panturin E. Sensorimotor integration for functional recovery and the Bobath approach. Motor Control 2011;15(2):285—301.

[148] Lewek MD, Randall EP. Reliability of spatiotemporal asymmetry during overground walking for individuals following chronic stroke. J Neurol Phys Ther 2011;35(3):116—21.

[149] Lewek MD, Bradley CE, Wutzke CJ, Zinder SM. The relationship between spatiotemporal gait asymmetry and balance in individuals with chronic stroke. J Appl Biomech 2014;30(1):31—6.

[150] Li S, Francisco GE. New insights into the pathophysiology of post-stroke spasticity. Front Hum Neurosci 2015;9:192.

[151] Lin JP, Brown JK. Peripheral and central mechanisms of hindfoot equinus in childhood hemiplegia. Dev Med Child Neurol 1992;34(11):949—65.

[152] Loram ID, Maganaris CN, Lakie M. Active, non-spring-like muscle movements in human postural sway: how might paradoxical changes in muscle length be produced? J Physiol 2005;564(Pt 1):281—93.

[153] Luft AR, Macko RF, Forrester LW, Villagra F, Ivey F, Sorkin JD, et al. Treadmill exercise activates subcortical neural networks and improves walking after stroke: a randomized controlled trial. Stroke 2008;39(12):3341—50.

[154] Lum PS, Mulroy S, Amdur RL, Requejo P, Prilutsky BI, Dromerick AW. Gains in upper extremity function after stroke via recovery or compensation: potential differential effects on amount of real-world limb use. Top Stroke Rehabil 2009;16(4):237—53.

[155] Luu BL, Inglis JT, Huryn TP, Van der Loos HF, Croft EA, Blouin JS. Human standing is modified by an unconscious integration of congruent sensory and motor signals. J Physiol 2012;590(Pt 22):5783—94.

[156] Macko RF, DeSouza CA, Tretter LD, Silver KH, Smith GV, Anderson PA, et al. Treadmill aerobic exercise training reduces the energy expenditure and cardiovascular demands of hemiparetic gait in chronic stroke patients. A preliminary report. Stroke 1997;28(2):326—30.

[157] Magnus R. Some results of studies in the physiology of posture. Lancet 1926;2(531):85.

[158] Mahon CE, Farris DJ, Sawicki GS, Lewek MD. Individual limb mechanical analysis of gait following stroke. J Biomech 2015;48(6):984—9.

[159] Malouin F, Richards CL. Mental practice for relearning locomotor skills. Phys Ther 2010;90(2):240—51.

[160] Marsh RL, Ellerby DJ, Carr JA, Henry HT, Buchanan CI. Partitioning the energetics of walking and running: swinging the limbs is expensive. Science 2004;303(5654):80—3.

[161] Marshall RS, Perera GM, Lazar RM, Krakauer JW, Constantine RC, DeLaPaz RL. Evolution of cortical activation during recovery from corticospinal tract infarction. Stroke 2000;31(3):656—61.

[162] Martino G, Ivanenko YP, Serrao M, Ranavolo A, d'Avella A, Draicchio F, et al. Locomotor patterns in cerebellar ataxia. J Neurophysiol 2014;112(11):2810—21.

[163] Massaad F, Lejeune TM, Detrembleur C. Reducing the energy cost of hemiparetic gait using center of mass feedback: a pilot study. Neurorehabil Neural Repair 2010;24(4):338—47.

[164] Massaquoi SG. Physiology of clinical dysfunction of the cerebellum. Handb Clin Neurol 2011;103:37—62.

[165] Massion J, Alexandrov A, Frolov A. Why and how are posture and movement coordinated? Prog Brain Res 2004;143:13—27.

[166] Matsuyama K, Mori F, Nakajima K, Drew T, Aoki M, Mori S. Locomotor role of the corticoreticular-reticulospinal-spinal interneuronal system. Prog Brain Res 2004;143:239—49.

[167] Mayr A, Kofler M, Quirbach E, Matzak H, Frohlich K, Saltuari L. Prospective, blinded, randomized crossover study of gait rehabilitation in stroke patients using the Lokomat gait orthosis. Neurorehabil Neural Repair 2007;21(4):307—14.

[168] McGowan CP, Neptune RR, Clark DJ, Kautz SA. Modular control of human walking: adaptations to altered mechanical demands. J Biomech 2010;43(3):412—9.

[169] Mehrholz J, Elsner B, Werner C, Kugler J, Pohl M. Electromechanical-assisted training for walking after stroke. Cochrane Database Syst Rev 2013;7:CD006185.

[170] Mehrholz J, Elsner B, Pohl M. Treadmill training for improving walking function after stroke: a major update of a Cochrane review. Stroke 2014;45(5):e76—7.

[171] Miller DM, Klein CS, Suresh NL, Rymer WZ. Asymmetries in vestibular evoked myogenic potentials in chronic stroke survivors with spastic hypertonia: evidence for a vestibulospinal role. Clin Neurophysiol 2014;125(10):2070—8.

[172] Milot MH, Nadeau S, Gravel D. Muscular utilization of the plantarflexors, hip flexors and extensors in persons with hemiparesis walking at self-selected and maximal speeds. J Electromyogr Kinesiol 2007;17(2):184—93.

[173] Mochon S, McMahon TA. Ballistic walking. J Biomech 1980;13(1):49—57.

[174] Mori S, Nishimura H, Kurakami C, Yamamura T, Aoki M. Controlled locomotion in the mesencephalic cat: distribution of facilitatory and inhibitory regions within pontine tegmentum. J Neurophysiol 1978;41(6):1580—91.

[175] Mori S. Contribution of postural muscle tone to full expression of posture and locomotor movements: multi-faceted analyses of its setting brainstem-spinal cord mechanisms in the cat. Jpn J Physiol 1989;39(6):785—809.

[176] Morita S, Yamamoto H, Furuya K. Gait analysis of hemiplegic patients by measurement of ground reaction force. Scand J Rehabil Med 1995;27(1):37—42.

[177] Morton SM, Bastian AJ. Cerebellar control of balance and loco-motion. Neuroscientist 2004;10(3):247—59.

[178] Mulroy S, Gronley J, Weiss W, Newsam C, Perry J. Use of cluster analysis for gait pattern classification of patients in the early and late recovery phases following stroke. Gait Posture 2003;18(1):114—25.

[179] Mun BM, Lee YS, Kim TH, Lee JH, Sim SM, Park IM, et al. Study on the usefulness of sit to stand training in self-directed treatment of stroke patients. J Phys Ther Sci 2014;26(4):483—5.

[180] Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. Clin Biomech (Bristol, Avon) 1999;14(2):125—35.

[181] Nakamura R, Handa T, Watanabe S, Morohashi I. Walking cycle after stroke. Tohoku J Exp Med 1988;154(3):241—4.

[182] Nardone A, Galante M, Lucas B, Schieppati M. Stance control is not affected by paresis and reflex hyperexcitability: the case of spastic patients. J Neurol Neurosurg Psychiatry 2001;70(5):635—43.

[183] Nardone A, Godi M, Grasso M, Guglielmetti S, Schieppati M. Stabilometry is a predictor of gait performance in chronic hemiparetic stroke patients. Gait Posture 2009;30(1):5—10.

[184] Neckel N, Pelliccio M, Nichols D, Hidler J. Quantification of functional weakness and abnormal synergy patterns in the lower limb of individuals with chronic stroke. J Neuroeng Rehabil 2006;3:17.

[185] Neptune RR, Kautz SA, Zajac FE. Contributions of the individual ankle plantar flexors to support, forward progression and swing initiation during walking. J Biomech 2001;34(11):1387—98.

[186] Neptune RR, Zajac FE, Kautz SA. Muscle force redistributes segmental power for body progression during walking. Gait Posture 2004;19(2):194—205.

[187] Neptune RR, Clark DJ, Kautz SA. Modular control of human walking: a simulation study. J Biomech 2009;42(9):1282—7.

[188] Ng SS, Hui-Chan CW. Contribution of ankle dorsiflexor strength to walking endurance in people with spastic hemiplegia after stroke. Arch Phys Med Rehabil 2012;93(6):1046—51.

[189] Nolan KJ, Yarossi M, McLaughlin P. Changes in center of pressure displacement with the use of a foot drop stimulator in individuals with stroke. Clin Biomech (Bristol, Avon) 2015;30(7):755—61.

[190] Novak AC, Brouwer B. Kinematic and kinetic evaluation of the stance phase of stair ambulation in persons with stroke and healthy adults: a pilot study. J Appl Biomech 2013;29(4):443—52.

[191] Öken Ö, Yavuzer G, Ergöçen S, Yorgancıoğlu ZR, Stam HJ. Repeatability and variation of quantitative gait data in subgroups of patients with stroke. Gait Posture 2008;27(3):506—11.

[192] Oliveira AS, Gizzi L, Kersting UG, Farina D. Modular organization of balance control following perturbations during walking. J Neurophysiol 2012;108(7):1895—906.

[193] Olney SJ, Richards C. Hemiparetic gait following stroke. Part I: characteristics. Gait Posture 1996;4(2):136—48.

[194] Olney SJ, Griffin MP, McBride ID. Multivariate examination of data from gait analysis of persons with stroke. Phys Ther 1998;78(8):814—28.

[195] Patla AE, Prentice SD, Robinson C, Neufeld J. Visual control of locomotion: strategies for changing direction and for going over obstacles. J Exp Psychol Hum Percept Perform 1991;17(3):603—34.

[196] Patterson KK, Gage WH, Brooks D, Black SE, McIlroy WE. Evaluation of gait symmetry after stroke: a comparison of current methods and recommendations for standardization. Gait Posture 2010;31(2):241—6.

[197] Patterson KK, Gage WH, Brooks D, Black SE, McIlroy WE. Changes in gait symmetry and velocity after stroke: a cross-sectional study from weeks to years after stroke. Neurorehabil Neural Repair 2010;24(9):783—90.

[198] Patterson KK, Nadkarni NK, Black SE, McIlroy WE. Gait symmetry and velocity differ in their relationship to age. Gait Posture 2012;35(4):590—4.

[199] Pennycott A, Wyss D, Vallery H, Klamroth-Marganska V, Riener R. Towards more effective robotic gait training for stroke rehabilitation: a review. J Neuroeng Rehabil 2012;9:65.

[200] Perez MA, Lungholt BK, Nyborg K, Nielsen JB. Motor skill training induces changes in the excitability of the leg cortical area in healthy humans. Exp Brain Res 2004;159(2):197—205.

[201] Perry J, Burnfield JM. Gait analysis: normal and pathological function. 2nd edn Thorofare, NJ: Slack Incorporated; 2010.

[202] Perry SB. Clinical implications of a dynamical systems theory. Neurol Rep 1998;22(1):4—10.

[203] Peterson CL, Cheng J, Kautz SA, Neptune RR. Leg extension is an important predictor of paretic leg propulsion in hemiparetic walking. Gait Posture 2010;32(4):451—6.

[204] Peterson CL, Hall AL, Kautz SA, Neptune RR. Pre-swing deficits in forward propulsion, swing initiation and power generation by individual muscles during hemiparetic walking. J Biomech 2010;43(12):2348—55.

[205] Peterson DS, Martin PE. Effects of age and walking speed on coactivation and cost of walking in healthy adults. Gait Posture 2010;31(3):355—9.

[206] Pomeroy VM, King L, Pollock A, Baily-Hallam A, Langhorne P. Electrostimulation for promoting recovery of movement or functional ability after stroke. Cochrane Database Syst Rev 2006;2:CD003241.

[207] Querol-Pascual MR. Clinical approach to brainstem lesions. Semin Ultrasound CT MRI 2010;31(3):220—9.

[208] Reisman DS, Wityk R, Silver K, Bastian AJ. Locomotor adaptation on a split-belt treadmill can improve walking symmetry post-stroke. Brain 2007;130(Pt 7):1861—72.

[209] Reisman DS, Wityk R, Silver K, Bastian AJ. Split-belt treadmill adaptation transfers to overground walking in persons poststroke. Neurorehabil Neural Repair 2009;23(7):735—44.

[210] Reisman DS, Bastian AJ, Morton SM. Neurophysiologic and rehabilitation insights from the split-belt and other locomotor adaptation paradigms. Phys Ther 2010;90(2):187—95.

[211] Reisman DS, McLean H, Keller J, Danks KA, Bastian AJ. Repeated split-belt treadmill training improves post-stroke step length asymmetry. Neurorehabil Neural Repair 2013;27(5):460—8.

[212] Richards CL, Malouin F, Dumas F, Tardif D. Gait velocity as an outcome measure of locomotor recovery after stroke. In: Craik RL, Oatis C, editors. Gait analysis: theory and application. St-Louis: Mosby; 1995. p. 355—64.

[213] Richards CL, Olney SJ. Hemiparetic gait following stroke. Part II: recovery and physical therapy. Gait Posture 1996;4(2):149—62.

[214] Robbins J, Butler SG, Daniels SK, Diez Gross R, Langmore S, Lazarus CL, et al. Swallowing and dysphagia rehabilitation: translating principles of neural plasticity into clinically oriented evidence. J Speech Lang Hear Res 2008;51(1):S276—300.

[215] Rosa MC, Marques A, Demain S, Metcalf CD. Lower limb co-contraction during walking in subjects with stroke: a systematic review. J Electromyogr Kinesiol 2014;24(1):1—10.

[216] Routson RL, Clark DJ, Bowden MG, Kautz SA, Neptune RR. The influence of locomotor rehabilitation on module quality and post-stroke hemiparetic walking performance. Gait Posture 2013;38(3):511—7.

[217] Routson RL, Kautz SA, Neptune RR. Modular organization across changing task demands in healthy and poststroke gait. Physiol Rep 2014;2(6):e12055.

[218] Ryan AS, Ivey FM, Prior S, Li G, Hafer-Macko C. Skeletal muscle hypertrophy and muscle myostatin reduction after resistive training in stroke survivors. Stroke 2011;42(2):416—20.

[219] Sadeghi H, Allard P, Duhaime PM. Muscle power compensatory mechanisms in below-knee amputee gait. Am J Phys Med Rehabil 2001;80(1):25—32.

[220] Saunders DH, Sanderson M, Brazzelli M, Greig CA, Mead GE. Physical fitness training for stroke patients. Cochrane Database Syst Rev 2013;10:CD003316.

[221] Saunders DH, Greig CA, Mead GE. Physical activity and exercise after stroke: review of multiple meaningful benefits. Stroke 2014;45(12):3742—7.

[222] Savin DN, Morton SM, Whitall J. Generalization of improved step length symmetry from treadmill to overground walking in persons with stroke and hemiparesis. Clin Neurophysiol 2014;125(5):1012—20.

[223] Schepens B, Stapley P, Drew T. Neurons in the pontomedullary reticular formation signal posture and movement both as an integrated behavior and independently. J Neurophysiol 2008;100(4):2235—53.

[224] Schmid S, Schweizer K, Romkes J, Lorenzetti S, Brunner R. Secondary gait deviations in patients with and without neurological involvement: a systematic review. Gait Posture 2013;37(4):480—93.

[225] Schneider W, Chein JM. Controlled & automatic processing: Behavior, theory, and biological mechanisms. Cogn Sci 2003;27(3):525—59.

[226] Schwartz I, Sajin A, Fisher I, Neeb M, Shochina M, Katz-Leurer M, et al. The effectiveness of locomotor therapy using robotic-assisted gait training in subacute stroke patients: a randomized controlled trial. PM & R 2009;1(6):516—23.

[227] Schwartz I, Meiner Z. [The influence of locomotor treatment using robotic body-weight-supported treadmill training on rehabilitation outcome of patients suffering from neurological disorders]. Harefuah 2013;152(3):166—71. [81,82].

[228] Sharma N, Baron JC. Does motor imagery share neural networks with executed movement: a multivariate fMRI analysis. Front Hum Neurosci 2013;7:564.

[229] Sherrington CS. The integrative action of the nervous system. New York, NY: Cambridge University Press; 1947.

[230] Shumway-Cook A, Woollacott MH. Clinical management of the patient with a mobility disorder. In: Shumway-Cook A, Woollacott MH, editors. Motor control — translating research into clinical practice. 4th edn Baltimore, MD: Lippincott Williams & Wilkins; 2012. p. 415—73.

[231] Signal NEJ. Strength training after stroke: rationale, evidence and potential implementation barriers for physiotherapists. N Z J Physiother 2014;42(2):101—7.

[232] Soo CH, Donelan JM. Coordination of push-off and collision determine the mechanical work of step-to-step transitions when isolated from human walking. Gait Posture 2012;35(2):292—7.

[233] States RA, Pappas E, Salem Y. Overground physical therapy gait training for chronic stroke patients with mobility deficits. Cochrane Database Syst Rev 2009;3:CD006075.

[234] Stavric VA, McNair PJ. Optimizing muscle power after stroke: a cross-sectional study. J Neuroeng Rehabil 2012;9:67.

[235] Takakusaki K, Saitoh K, Harada H, Kashiwayanagi M. Role of basal ganglia-brainstem pathways in the control of motor behaviors. Neurosci Res 2004;50(2):137—51.

[236] Takakusaki K. Forebrain control of locomotor behaviors. Brain Res Rev 2008;57(1):192—8.

[237] Takakusaki K. Neurophysiology of gait: from the spinal cord to the frontal lobe. Mov Disord 2013;28(11):1483—91.

[238] Thelen E, Ulrich BD, Niles D. The developmental origins of locomotion. In: Woollacott MH, Shumway-Cook A, editors. Development of posture and gait across the life span. Columbia, SC: University of South Carolina Press; 1989.

[239] Ting LH, Kautz SA, Brown DA, Zajac FE. Phase reversal of biomechanical functions and muscle activity in backward pedaling. J Neurophysiol 1999;81(2):544—51.

[240] Ting LH, McKay JL. Neuromechanics of muscle synergies for posture and movement. Curr Opin Neurobiol 2007;17(6):622—8.

[241] Tohgi H, Takahashi S, Chiba K, Hirata Y. Cerebellar infarction. Clinical and neuroimaging analysis in 293 patients. Stroke 1993;24(11):1697—701.

[242] Torres-Oviedo G, Ting LH. Muscle synergies characterizing human postural responses. J Neurophysiol 2007;98(4):2144—56.

[243] Turns LJ, Neptune RR, Kautz SA. Relationships between muscle activity and anteroposterior ground reaction forces in hemiparetic walking. Arch Phys Med Rehabil 2007;88(9):1127—35.

[244] Tyson SF, Sadeghi-Demneh E, Nester CJ. A systematic review and meta-analysis of the effect of an ankle-foot orthosis on gait biomechanics after stroke. Clin Rehabil 2013;27(10):879—91.

[245] van Emmerik RE, Wagenaar RC. Effects of walking velocity on relative phase dynamics in the trunk in human walking. J Biomech 1996;29(9):1175—84.

[246] Vaughan-Graham J, Cott C, Wright FV. The Bobath (NDT) concept in adult neurological rehabilitation: what is the state of the knowledge? A scoping review. Part II: intervention studies perspectives. Disabil Rehabil 2015;37(21):1909—28.

[247] Vaughan-Graham J, Cott C, Wright FV. The Bobath (NDT) concept in adult neurological rehabilitation: what is the state of the knowledge? A scoping review. Part I: conceptual perspectives. Disabil Rehabil 2015;37(20):1793—807.

[248] Veerbeek JM, van Wegen E, van Peppen R, van der Wees PJ, Hendriks E, Rietberg M, et al. What is the evidence for physical therapy poststroke? A systematic review and meta-analysis. PLoS One 2014;9(2):e87987.

[249] Verdié C, Daviet JC, Borie MJ, Popielarz S, Munoz M, Salle JY, et al. Epidemiology of varus equinus one year after an hemispheral stroke. Ann Readapt Med Phys 2004;47(2):81—6.

[250] Volpe BT, Krebs HI, Hogan N. Is robot-aided sensorimotor training in stroke rehabilitation a realistic option? Curr Opin Neurol 2001;14(6):745—52.

[251] Wagenaar RC, Meijer OG, van Wieringen PC, Kuik DJ, Hazenberg GJ, Lindeboom J, et al. The functional recovery of stroke: a comparison between neuro-developmental treatment and the Brunnstrom method. Scand J Rehabil Med 1990;22(1):1—8.

[252] Walker C, Brouwer BJ, Culham EG. Use of visual feedback in retraining balance following acute stroke. Phys Ther 2000;80(9):886—95.

[253] Wall A, Borg J, Palmcrantz S. Clinical application of the Hybrid Assistive Limb (HAL) for gait training-a systematic review. Front Syst Neurosci 2015;9:48.

[254] Weerdesteyn V, de Niet M, van Duijnhoven HJ, Geurts AC. Falls in individuals with stroke. J Rehabil Res Dev 2008;45(8):1195—213.

[255] Welmer AK, Holmqvist LW, Sommerfeld DK. Hemiplegic limb synergies in stroke patients. Am J Phys Med Rehabil 2006;85(2):112—9.

[256] Werner C, Von Frankenberg S, Treig T, Konrad M, Hesse S. Treadmill training with partial body weight support and an electromechanical gait trainer for restoration of gait in subacute stroke patients: a randomized crossover study. Stroke 2002;33(12):2895—901.

[257] Westlake KP, Patten C. Pilot study of Lokomat versus manual-assisted treadmill training for locomotor recovery post-stroke. J Neuroeng Rehabil 2009;6:18.

[258] Winstein CJ, Gardner ER, McNeal DR, Barto PS, Nicholson DE. Standing balance training: effect on balance and locomotion in hemiparetic adults. Arch Phys Med Rehabil 1989;70(10):755—62.

[259] Winter DA, Yack HJ. EMG profiles during normal human walking: stride-to-stride and inter-subject variability. Electroencephalogr Clin Neurophysiol 1987;67(5):402—11.

[260] Winter DA. Biomechanics and motor control of human movement. Third edition Wiley; 2004.

[261] Wissel J, Manack A, Brainin M. Toward an epidemiology of poststroke spasticity. Neurology 2013;80(3 Suppl. 2):S13—9.

[262] Wong AM, Pei YC, Hong WH, Chung CY, Lau YC, Chen CP. Foot contact pattern analysis in hemiplegic stroke patients: an implication for neurologic status determination. Arch Phys Med Rehabil 2004;85(10):1625—30.

[263] Yeo SS, Ahn SH, Choi BY, Chang CH, Lee J, Jang SH. Contribution of the pedunculopontine nucleus on walking in stroke patients. Eur Neurol 2011;65(6):332—7.

[264] Yeom J, Park S. A gravitational impulse model predicts collision impulse and mechanical work during a step-to-step transition. J Biomech 2011;44(1):59—67.

[265] Zelik KE, La Scaleia V, Ivanenko YP, Lacquaniti F. Can modular strategies simplify neural control of multidirectional human locomotion? J Neurophysiol 2014;111(8):1686—702.