Gait post-stroke: Pathophysiology and rehabilitation strategies

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

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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

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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 [59,193]. Walking is rapidly expanding and is providing 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.
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.

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 pre-motor 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 via 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
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...
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 corticoretic-plegic limb synergies [19]. However, spasticity prevalence is during the subacute and chronic phases after stroke [82].

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 antigraft 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 laterialized 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 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 Brunström’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 flat-foot- or firstfoot-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...
Pathophysiology of post-stroke gait and rehabilitation

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].

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, use about 15% more thoracic than pelvic 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
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 [99]. 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 pre-determined 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 resistance training for 5 weeks, followed by 3 weeks of gait training, in high-intensity rehabilitation programme. The greatest gains in neuromuscular activation and power of affected leg muscles were achieved in the group practising eccentric resistance 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 [233]. 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 rehabilitation 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 transiently 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 [91,112,113,117,119,179,217,222] 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.

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