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REVIEW/MISE AU POINT

Why we should study gait initiation in Parkinson's disease

Pourquoi étudier l'initiation de la marche dans la maladie de Parkinson

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Summary The gait initiation process is of particular interest in Parkinson's disease because it combines motor and cognitive components of movement preparation (referred to as anticipatory postural adjustments) and movement execution (the step by itself). Moreover, gait initiation in Parkinson's disease is often affected by motor blocks (a subtype of the ''freezing of gait'' phenomenon). Gait initiation disturbances in Parkinson's disease include delayed release of anticipatory postural adjustments, hypokinetic anticipatory postural adjustments (reduced scaling) and bradykinetic anticipatory postural adjustments (abnormal timing). The most extreme form is freezing of gait with sometimes the absence of anticipatory postural adjustments. Other phenomena can be also described in some freezing patients (such as multiple anticipatory postural adjustments, described clinically as ''knee trembling''). The fact that emotion, attention, external triggers and dopaminergic drugs can all modify this motor program suggests the existence of a complex pathophysiological mechanism that involves not only locomotor networks but also cortical areas and the basal ganglia system. Abnormal coupling between standing posture and anticipatory postural adjustments and between the latter and step execution appears to be a crucial part of the pathophysiological mechanism. Although external cueing appears to be of interest, few studies have provided evidence of the efficacy of various rehabilitation methods in routine care.
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Résumé Un des intérêts de l'étude de l'initiation de la marche chez le patient parkinsonien est que cette tâche combine un contrôle volontaire et cognitif du programme moteur. Elle est souvent perturbée par des enrayages cinétiques aboutissant dans leur forme extrême à un blocage complet du programme moteur appelé *freezing* de la marche. Un retard de déclenchement des ajustements posturaux anticipés, une réduction de l'amplitude (hypokinésie) de ces derniers ainsi qu'une augmentation de leur durée (bradykinésie) sont notés, l'absence totale d'ajustements posturaux adaptés représentant la forme la plus pure d'akinésie vue dans le phénomène de *freezing* de la marche. Cet enrayage peut se traduire également par des ajustements posturaux multiples précédant le pas, visible cliniquement sous forme d'un tremblement des genoux précédant le pas. L'émotion l'attention, des indiçages externes mais aussi les traitements dopaminergiques peuvent modifier le déclenchement du programme moteur, soulignant des mécanismes physiopathologiques multiples impliquant les circuits locomoteurs mais aussi différentes aires corticales en relation avec le système des ganglions de la base. Un couplage anormal non seulement entre la posture debout et les ajustements posturaux anticipés, mais aussi entre ces derniers et le premier pas apparaissent déterminants dans la physiopathologie de l'initiation de la marche chez le parkinsonien. Bien que l'indiçage externe apparaisse intéressant pour améliorer le déficit d'initiation du pas, l'utilisation de ce dernier en routine clinique n'est pas encore usuel du fait du peu de résultats probants dans la littérature spécifiques à la rééducation de l'initiation de la marche.
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Falls are a debilitating problem in Parkinson's disease (PD); people with PD are twice as likely to fall as people with other neurological conditions [61]. Falling is more common during transition movements, in which momentum must be generated or restrained and/or the base of support changes (i.e. when the centre of mass (CoM) moves outside the base of support) [56]. During gait initiation (GI), postural instability increases dramatically as the base of support changes and the centre of gravity (CoG) moves forward and outside the latter [72]. Decoupling the centre of pressure (CoP), the barycentre of the ground reaction forces and the vertical projection of the CoM enables the CoG to move forwards. This initial phase in the GI process (before foot-off) can be considered as a forward fall that must be restrained but is necessary for subsequent forward body movement [5]. In the present review, we shall describe the different aspects of GI in PD, by describing the characteristics and substrates for GI and improvement/rehabilitation strategies.

Description of GI in PD

Gait initiation is accompanied by anticipatory postural adjustments (APAs). These adjustments are essential because they unload the swing leg and thus create the conditions required for progression [6]. Indeed, the amplitude and duration of APAs are predictive of the subsequent peak step velocity [7]. During GI, healthy subjects always follow a highly stereotypical preparation pattern. Foot-off of the swing leg is preceded by co-activation of the tibialis anterior (TA). This shift in body weight generates a displacement of the CoP backwards and towards the swing leg. Next, a CoP displacement towards the stance leg and then forwards is observed. Heel-off of the swing leg occurs at the start of the second phase of the CoP displacement (i.e. with a lateral shift towards the stance leg), with toe-off just before the forward CoP displacement (Fig. 1) [17]. Brunt et al. [8] suggested that GI resulted from two highly coordinated motor programs, with heel-off of the stance leg marking the division between the two.

Impaired GI in patients with PD is a typical functional sign of akinesia (defined as a failure or slowness of willed movement) [26]. According to Halliday et al. [27], akinesia can been classified into three categories on the basis of when and how the symptoms present themselves:

- slow and non-dexterous movement;
- poverty of movement in the absence of muscle rigidity or weakness;
- difficulty initiating or maintaining movement (referred to as ''freezing of gait'' (FoG) or ''motor block'').

Failure of GI is a complex problem in advanced PD patients and is sometimes refractory to treatment with medications. Impairment of the archetypal APAs is considered to be a major pathophysiological mechanism underlying impaired GI in PD [27]. In patients with PD, the timing and size of bilateral TA excitation during GI are often abnormal. When initiating gait, PD patients spend a greater amount of time with low or no TA excitation [21]. As a consequence, the mediolateral and anteroposterior ground reaction forces and CoP changes that characterize APAs in PD patients are longer and weaker, with prolonged delays between APA onset and step onset [9,19,27,28,35,67]. The APAs for voluntary step initiation are often absent in PD patients, who display either hesitation [9] or very slow progression. Multiple APAs can also occur and correspond to a subtype of FoG referred to as ''knee trembling'' [32] (Fig. 1). Patients with PD also display impaired stepping performance, with a shorter first step length and a lower initial stepping speed.

These abnormalities can occur very early in the course of PD. Low-magnitude APAs (measured from peak CoP displacements and accelerations) have already been observed in untreated early-to-moderate-stage patients [38] in whom start hesitation may not be clinically detectable.

Other aspects of GI control have been addressed in healthy controls and/or parkinsonian patients. During the

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