



# Upright standing after stroke: How loading-unloading mechanism participates to the postural stabilization



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## ABSTRACT

Postural strategies employed by hemiparetic stroke patients need to be better understood to guide rehabilitation. Of the two complementary mechanisms used to stabilize the standing posture, loading-unloading (LU) and pressure distribution (PD), it is hypothesized that the former would be predominantly used. To this aim, posturographic assessments, through a dual force-platform, were performed in 30 Hemiparetics tested 3 months after a unilateral stroke, and 30 matched healthy Controls. Original indices (from 0 to 1) were calculated to assess LU and PD contributions. The results show that along the mediolateral axis, the LU contribution was very high and similar in Hemiparetics and in Controls ( $0.80 \pm 0.07$  vs  $0.76 \pm 0.09$  a.u.;  $p > 0.05$ ), indicating a predominant hip involvement. Along the anteroposterior axis, the PD contribution was very close to 1 in controls ( $0.96 \pm 0.03$  a.u.) indicating an exclusive ankle involvement. Despite a lower contribution in Hemiparetics ( $0.88 \pm 0.11$  a.u.;  $p < 0.01$ ), the indices were surprisingly always above 0.5, meaning that ankle movements remain predominant for controlling postural sways along the anteroposterior axis in all patients even those with severe clinical deficits. However the PD contribution appeared larger in patients with light or moderate deficits of the sensitivity ( $r = -0.532$ ;  $p < 0.01$ ) or the motor command ( $r = -0.513$ ;  $p < 0.01$ ). These results indicate that postural stabilization of hemiparetic persons remains controlled by a PD mechanism along the anteroposterior axis, even in those combining poor distal motor command and deep sensory loss. This ankle control, piloted by the more-loaded non-paretic limb, would therefore be preferred to a hip control through lateral trunk motion. This should be considered when defining the objectives of the postural rehabilitation after stroke.

## 1. Introduction

Most individuals with hemiparesis show postural disorders that are obvious in erect stance, due either to impaired control of body stabilization or orientation with respect to gravity (Barra, Oujamaa, Chauvineau, Rougier, & Pérennou, 2009; Pérennou et al., 2008). The stabilization impairment has been better understood since the recent use of separate measurements of the reaction forces under each foot (using a dual-force platform). This approach has highlighted the respective role played by each lower limb, with a key contribution of the non-paretic lower limb to compensate for the inability of the paretic limb to control the upright stance (de Haart, Geurts, Huidekoper, Fasotti, & van Limbeek, 2004; Genthon et al., 2008; Mansfield, Mochizuki, Inness, & McIlroy, 2012; Singer, Mansfield, Danells, McIlroy, & Mochizuki, 2013; van Asseldonk et al., 2006). Indeed, even though in the most impaired patients, the

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control of the center-of-pressure (CP) displacements under both feet were largely deteriorated, only the one measured under the more loaded foot play a significant role in the resultant center-of-pressure ( $CP_{Res}$ ) production. In other words, the increased  $CP_{Res}$  movements observed in hemiparetic patients, especially along the ML axis, could be explained by their relative incapacity to control their non-paretic lower limb. Interestingly, from a biomechanical perspective, this altered control could in turn affect in different ways one of the two basic mechanisms involved in balance control. Indeed, as initially underlined by Winter, Prince, Frank, Powell, and Zabjek (1996), upright standing mainly relies on loading-unloading (LU) and pressure distribution (PD) to control the displacements of the controlling variable, i.e. the  $CP_{Res}$ , along medio-lateral (ML) and antero-posterior (AP) axes, respectively when both feet are positioned side-by-side. Even though the concept of ankle and hip strategies proposed by Nashner and McCollum (1985) and extended by Winter et al. (1996) to describe the control of the quiet stance along the AP axis has been widely accepted for several decades, it has so far aroused little clinical interest, limited to vestibular or peripheral somatosensory deficits (Horak, Nashner, & Diener, 1990; Lafond, Corriveau, & Prince, 2004). To our knowledge, this objective to differentiate the respective contribution of these two main joints in the postural control, never applied on individuals with a brain lesion, appears especially relevant for at least three reasons. First, the integration of vestibular or somaesthetic information may be altered depending on the brain areas encroached on by the lesion. This might influence the selection of the predominant mechanism involved in the stabilization of the erect stance and therefore the joint: either the hip or the ankle. Second, the motor deficit is generally more severe for distal muscles (ankle) than for proximal muscles (hip) (Adams, Gandevia, & Skuse, 1990), which should favor a predominant role of the LU mechanisms in stabilizing the erect stance. Third, the weight-bearing asymmetry often observed in these patients induces opposite shifts of the plantar CPs, forward for the unloaded paretic limb and backward for the loaded nonparetic limb (Genthon et al., 2008; Geurts, de Haart, van Nes, & Duysens, 2005). Regardless of clinical deficits, this forward-backward gap creates the unique condition for  $CP_{Res}$  displacements to covariate along both ML and AP axes through a simplified motor command involving in larger proportions the hip muscles. To be more precise, we hypothesized that, in hemiparetic patients, less impaired motor command of hip muscles might play a predominant role in controlling upright stance along both the ML and AP axes, with a relationship between this predominance and the severity of motor and somatosensory deficits. This type of balance control strategy would be in accordance with the hypothesis that patients suffering from disease-related deficiencies, at their feet and legs, may exaggeratedly solicit proximal control mechanisms involving their hip (Bonnet & Lepeut, 2011).

This hypothesis was tested by comparing posturographic measurements of hemiparetic patients and matched healthy controls, and by analyzing LU and PD contributions through original indices allowing a quantification of their respective involvement (Rougier, 2007).

## 2. Methods

### 2.1. Subjects

Thirty hemiparetic patients, with a first hemispheric stroke and impaired balance control, and 30 healthy subjects gave informed consent to participate in accordance with the guidelines of the local ethics committee. In order to be eligible for the study, patients had to have the inclusion criteria of a first and unique hemisphere stroke. Exclusion criteria included unstable status, neuropathy, psychiatric disorders, major comprehension problems due to aphasia, dementia, signs of vestibular disorders and significant orthopaedic disease. As proposed in our previous studies (Genthon et al., 2008; Pérennou et al., 2001), clinical assessments were performed as follows by a trained practitioner who examine patients at the time of posturography. Motor weakness of eight muscle groups of the lower limb was assessed manually at hip (flexion, extension, abduction, adduction), knee (flexion, extension), and ankle (flexion, extension) levels through a five-point scale adapted to patients with central neurological disorders (Held, Pierrot-Desselligny, Bussel, Perrigot, & Mahler, 1975). Spasticity of 5 muscle groups of the lower limbs (psoas, hip adductors, quadriceps, hamstring, triceps surae) was assessed using the Ashworth scale (Ashworth, 1964). Hypoesthesia of the paretic side was assessed through pressure sensitivity at the pulp of the big toe using the Semmes-Weinstein aesthesiometer (Semmes, Weinstein, Ghent, & Teuber, 1960). The value of the force needed for a given patient to perceive the stimulus applied to the skin was then submitted to a log transform to obtain a 20-point linear increasing scale. The severity of the spatial neglect was quantified by a standard scale of behavioral neglect assessing both body and nonbody spaces (Azouvi et al., 2003). Balance disorders and gait ability were assessed by the Postural Assessment Scale for Stroke (PASS, Benaim, Perennou, Villy, Rousseaux, & Pelissier, 1999) and a six-point walking score (Lindmark & Hamrin, 1988), respectively. All these clinical characteristics, displayed through Table 1 with anthropometric information, are those usually found in series of patients with a recovery compatible with the maintenance of the standing posture without help for several minutes after a hemisphere stroke of various size and location.

### 2.2. Procedure

Postural movements were measured with barefoot subjects placing their feet (heels 9 cm apart, toes pointed out at 30°) on a dual force platform (PF02-Equi+, France). Hemiparetic patients adopted a spontaneous and comfortable stance, whereas controls stood asymmetrically to obtain matched weight-bearing distributions. Beforehand, controls performed one practice trial to be trained to stand asymmetrically at a required body weight distribution (about two third of body-weight on the right leg). Auditory feedback was used to guide them to reach this required body-weight distribution. Then, no feedback or information was provided during the trials collection. All participants were recorded during four 32-s trials (64 Hz). The subjects stood barefoot, each foot placed on one of the two side-by-side platforms (arms relaxed and hanging freely at the sides without ankle-foot orthoses, gazing at a fixed target

**Table 1**  
Features of participants (mean  $\pm$  s.d.).

	Healthy subjects	Hemiparetic patients
Sex	20 M; 10F	22 M; 8F
Age (years)	59.7 $\pm$ 12.2	60.1 $\pm$ 14.7
Height (m)	171.9 $\pm$ 8.7	168.7 $\pm$ 8.9
Weight (kg)	68.8 $\pm$ 12.9	66.0 $\pm$ 13.1
Time elapsed from stroke (days)		92.0 $\pm$ 48
Stroke side		20 left; 10 right
Motor weakness in lower legs 0 (normal) to 40 (severe weakness)		16.2 $\pm$ 11.6
Spasticity 0 (normal tone) to 20 (severe spasticity)		2.4 $\pm$ 3.2
Hypoesthesia using Semmes-Weinstein filaments (log F-mg) 1.65 (normal pressure sensitivity) to 6.65 (severe sensory loss)		5.0 $\pm$ 1.4
Behavioral neglect 0 (no neglect) to 30 (severe neglect)		3.3 $\pm$ 4.0
Postural control in daily life (PASS) 0 (severe postural disorders) to 36 (satisfactory erect stance)		30.2 $\pm$ 4.8
Gait autonomy (Lindmark test) 0 (no gait) to 6 (autonomous gait)		3.2 $\pm$ 1.5

(diameter, 30 mm, positioned one meter ahead) in an environment composed of vertical lines.

### 2.3. Signal processing

CP displacements under each foot and body-weight distribution were recorded and used to compute, along the AP and ML axes, displacements of the resultant CP ( $CP_{Res}$ ) according to the following relation

$$CP_{Res} = CP_{LF}(F_{LF}/F_{LF} + F_{RF}) + CP_{RF}(F_{RF}/F_{LF} + F_{RF})$$

where  $CP_{LF}$  and  $CP_{RF}$  are the CP displacements under the left and right feet, respectively, and  $F_{LF}$  and  $F_{RF}$  the reaction forces exerted by the left and right feet, respectively.

The postural behavior of patients and subjects was assessed by (1) the mean and dispersion (standard deviation) of the successive positions of  $CP_{Res}$ , (2) the sagittal distance between the mean positions of the two  $CP_{LF}$  and  $CP_{RF}$  trajectories (AP-dist), and (3) the relative contribution to the  $CP_{Res}$  displacements of LU and PD mechanisms ( $Contr_{LU}$  and  $Contr_{PD}$ ) (Rougier, 2007).

To disentangle the relative mechanism contribution, we recomputed  $CP_{Res}$  displacements once the CP displacements under each foot had been neutralized, then once the body-weight distribution had been neutralized (Rougier, 2007; Winter et al., 1996). This was done by interchanging each time series value by the mean of the corresponding series. At this stage, a  $CP_{Res}$  trajectory could be split into two elementary trajectories,  $CP_{LU}$  and  $CP_{PD}$ , which were intended to correspond to the  $CP_{Res}$  displacements that would have been obtained if the LU or the PD mechanisms only were involved in the control. As seen in Fig. 1, when the feet of a healthy subject are positioned side by side, the  $CP_{LU}$  and  $CP_{PD}$  displacements were orthogonal to each other. To assess their relative contribution to balance control along a given axis,  $Contr_{LU}$  and  $Contr_{PD}$  indices were then computed from the relative dispersions (standard deviation, s.d.) of these  $CP_{LU}$  and  $CP_{PD}$  with respect to the sum  $CP_{LU} + CP_{PD}$  (Rougier, 2007).

$$Contr_{LU} = s. d. CP_{LU} / (s.d. CP_{LU} + s. d. CP_{PD})$$

$$Contr_{PD} = s. d. CP_{PD} / (s.d. CP_{LU} + s. d. CP_{PD})$$

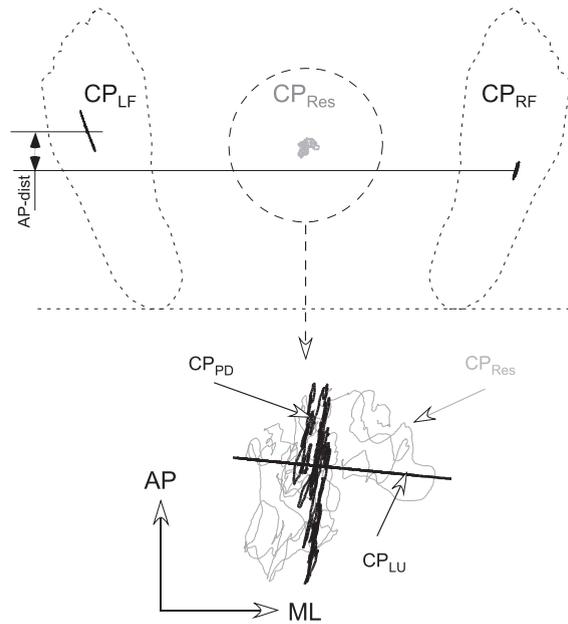
Since by definition,  $Contr_{LU} + Contr_{PD} = 1$ , only the highest index, expressing the most involved mechanism in the control (PD and LU along the AP and ML axes, respectively) was presented. The higher the index, the greater the mechanism contribution.

### 2.4. Statistics

ANOVAs bearing on a within-subject factor (ML and AP axes) and a between-subject factor (hemiparetics and controls) were carried out to assess the potential effects for the  $CP_{Res}$  dispersion (s.d.) and contribution indices (with Newman-Keuls post-hoc tests used when necessary). Because of their 0 to 1 range, statistical tests for the  $Contr_{LU}$  and  $Contr_{PD}$  indices were done on z transforms to normalize these distributions such that:

$$Z = 1/2 \text{Log}[\log(1 + Contr_{LU}) / \log(1 - Contr_{LU})] \text{ or } Z = 1/2 \text{Log}[\log(1 + Contr_{PD}) / \log(1 - Contr_{PD})]$$

Mean body-weight distribution, mean position of the  $CP_{Res}$  relative to the foot length and AP-dist were compared through nonparametric Mann-Whitney tests. Spearman rank correlation coefficients were computed to assess the linear relationships between clinical and posturographic parameters. The first level of significance for all tests was set at  $p < .05$ . All data are presented as mean  $\pm$  s.d.



**Fig. 1.** Top: The two plantar CP displacements, associated with the body-weight distribution (not displayed on the graph) can be used for computing a resultant center-of-pressure (CP<sub>Res</sub>) trajectory. Bottom: In turn, a CP<sub>Res</sub> trajectory can be split into two elementary CP<sub>PD</sub> and CP<sub>LU</sub> trajectories, each expressing the main contribution of ankle and hip joints to this movement.

### 3. Results

#### 3.1. Mean body-weight distribution

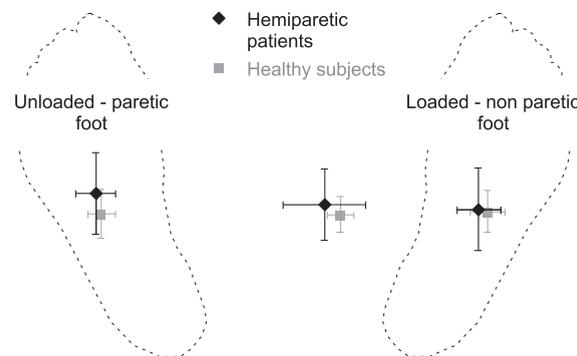
Hemiparetic patients adopted a spontaneous distribution of  $63.0 \pm 11.3\%$  over the loaded nonparetic limb. This value was matched by controls who voluntarily stood asymmetrically ( $65.7 \pm 3.0\%$ ;  $U = 319$ ;  $p > 0.05$ ).

#### 3.2. CP<sub>Res</sub> mean positions along the AP axis

CP<sub>Res</sub> mean positions along the AP axis, expressed relative to the foot length, were similar in both groups (hemiparetic patients:  $0.44 \pm 0.10$ ; controls:  $0.41 \pm 0.05$ ;  $U = 405$ ;  $p > 0.05$ ). Taken together, these two results demonstrate controls' ability to adopt a posture similar to that of hemiparetic patients with respect to the base of support (Fig. 2).

#### 3.3. CP<sub>Res</sub> dispersion

CP<sub>Res</sub> dispersion differed between groups [ $F(1,58) = 39.50$ ,  $p < 0.01$ ] but not between the two axes [ $F(1,58) = 0.23$ ,  $p > 0.05$ ], with an interaction between the two factors [ $F(1,58) = 9.38$ ,  $p < 0.01$ ]. Post-hoc analyses indicated greater dispersions, thus



**Fig. 2.** Mean positions (with standard deviations) of the plantar (CP<sub>LF</sub> and CP<sub>RF</sub>) and CP<sub>Res</sub> displacements for the two groups. Note the asymmetric body-weight distribution for both groups (inferring a lateral shift of the CP<sub>Res</sub>) and the tendency in both groups to shift forwardly and backwardly on their two plantar CP positions (AP-dist).

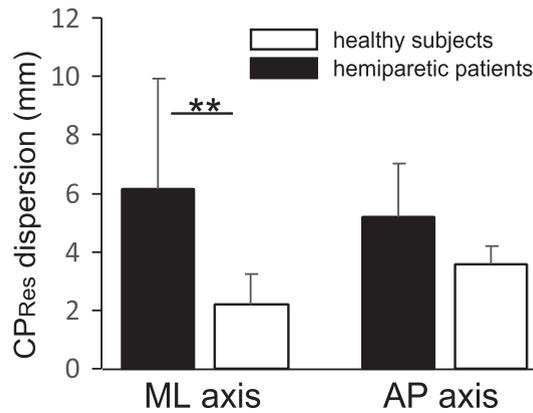


Fig. 3. Mean standard deviations of the CP<sub>Res</sub> movements along both ML and AP axes for the two groups of subjects (\*\*:  $p < 0.01$ ).

instability, in hemiparetic patients than in controls along the ML axis ( $p < 0.01$ ) but not along the AP axis (Fig. 3). CP<sub>Res</sub> dispersion along the ML and AP axes were correlated in controls ( $r = 0.366$ ;  $p < 0.05$ ) but not in hemiparetic patients ( $r = 0.278$ ,  $p > 0.05$ ).

### 3.4. The AP-dist

The AP-dist, which is the distance between mean CP positions under each foot along the AP axis, was twice greater in hemiparetic patients ( $28.3 \pm 23.8$  mm) than in controls ( $13.7 \pm 10.2$  mm,  $U = 297$ ;  $p < 0.05$ ). This difference was mainly due to a more forward position of the CP under the paretic limb (Fig. 2).

### 3.5. Contribution indices

Typical combinations of CP<sub>Res</sub>, CP<sub>LU</sub>, and CP<sub>PD</sub> trajectories are represented in Fig. 4, both for an asymmetric healthy subject and a hemiparetic patient. The ANOVA computed for the contribution indices showed a group effect [ $F(1,58) = 25.71$ ,  $p < 0.01$ ], an axis effect [ $F(1,58) = 668.50$ ,  $p < 0.01$ ], as well as an interaction between these factors [ $F(1,58) = 17.69$ ,  $p < 0.01$ ].

Along the ML axis, the Contr<sub>LU</sub> indices were high in both groups, indicating the predominance of LU mechanism. However, and contrary to what was noted along the AP axis, the values reported for the hemiparetic patients were close to those found in controls ( $0.80 \pm 0.07$  a.u.; range 0.59–0.89 vs  $0.76 \pm 0.09$  a.u.; range 0.49–0.88;  $p > 0.05$ ). No correlation was found between Contr<sub>LU</sub> indices and clinical deficits. These results indicate that the part devoted to hip and ankle strategies for controlling body stabilization in the frontal plane was unaffected by clinical deficits in hemiparetic patients.

Along the AP axis, Contr<sub>PD</sub> indices were very close to 1 in controls ( $0.96 \pm 0.03$  a.u.; range, 0.87–1.00), meaning that CP<sub>Res</sub> displacements were almost exclusively produced by PD mechanism. Although less PD involvement was found in hemiparetic patients ( $0.88 \pm 0.11$  a.u.;  $p < 0.01$ ), the indices were surprisingly always above 0.5 (range, 0.53–0.98). This meant that the PD mechanism predominantly controls AP postural sways in all patients, even those with severe clinical deficits. Contr<sub>PD</sub> indices were negatively correlated with hypoesthesia ( $r = -0.532$ ;  $p < 0.01$ ), motor weakness ( $r = -0.513$ ;  $p < 0.01$ ), and spasticity ( $r = -0.388$ ;  $p < 0.05$ ), indicating that the more severe the weakness and sensory loss, the less the PD mechanism contributed to body stabilization (Fig. 5A–C). Conversely, no significant linear correlation was found for behavioral neglect ( $r = -0.255$ ;  $p > 0.05$ ). In addition, Contr<sub>PD</sub> was negatively correlated with the AP-dist parameter for both controls ( $r = -0.859$ ;  $p < 0.01$ ) and hemiparetic patients ( $r = -0.816$ ;  $p < 0.01$ ): the greater the AP-dist, the smaller the PD contribution (Fig. 5D). This meant that the LU contribution to body stabilization increased with the distance between the average positions of the CP under each foot. Conversely, positive correlations were found with the PASS ( $r = 0.305$ ,  $p < 0.05$ ) and Lindmark scores ( $r = 0.514$ ,  $p < 0.01$ ), indicating that a

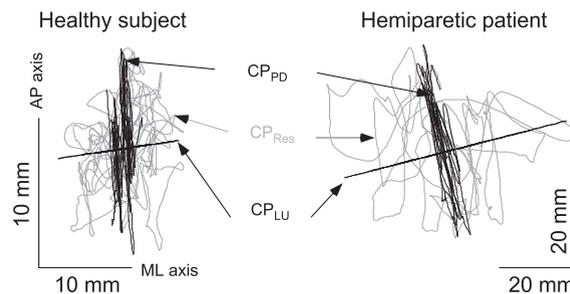
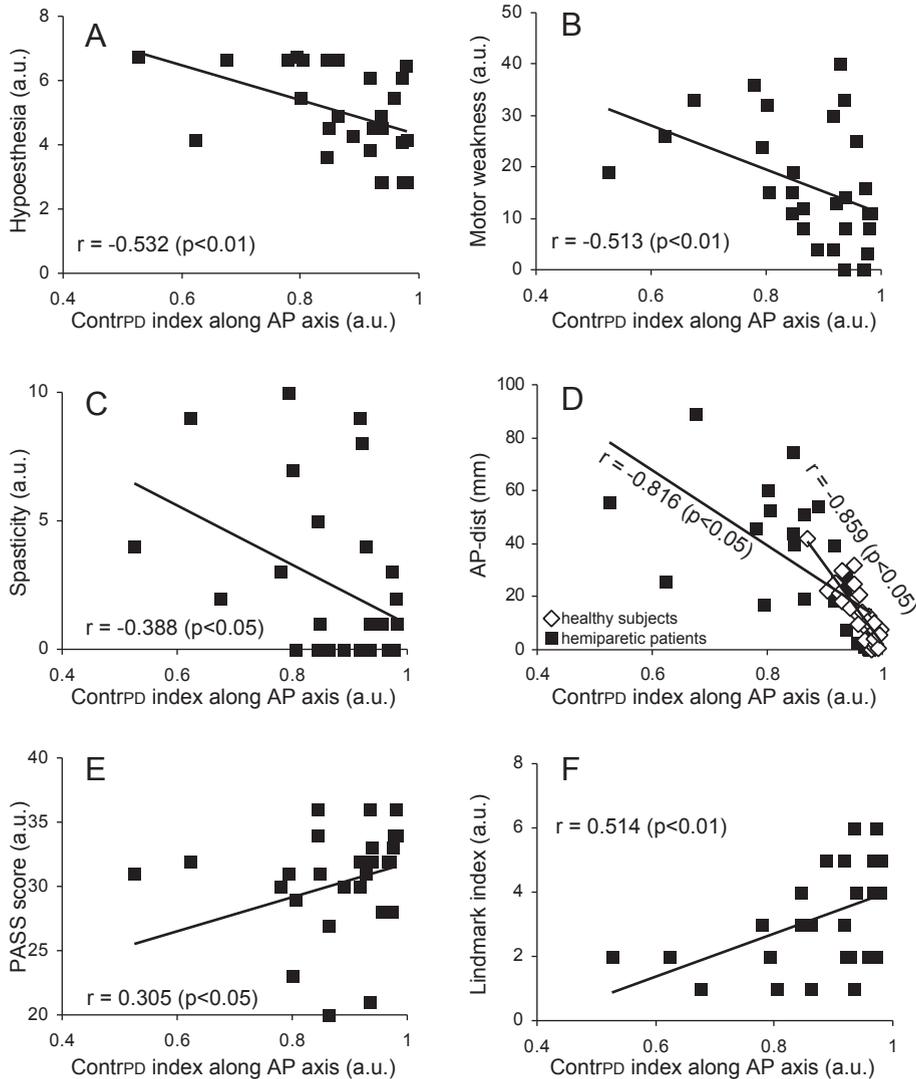


Fig. 4. Three typical trajectories obtained in a healthy subject and an hemiparetic patient. Note the predominant contribution of the LU and PD control mechanisms along the ML and AP axes, respectively, and the differences in scales between the two subjects.



**Fig. 5.** A–C and E–F: Scattergrams displaying linear correlation between clinical deficits and the ContrPD indices measured along the AP axis. D: Scattergrams displaying linear correlation between AP-dist values and the ContrPD indices measured along the AP axis for both groups.

more efficient PD mechanism was associated with better balance and gait capacities (Fig. 5E–F).

#### 4. Discussion

This study analyzed the relative role played by the two basic mechanisms, i.e. LU and PD, in maintaining upright quiet stance and the way they may be affected by sensorimotor impairments resulting from a stroke. As previously emphasized through a bio-mechanical approach (Winter et al., 1996), each joint plays a predominant role in controlling body movements along a specific axis for a given feet position. When feet are side-by-side, the hips along the ML axis and the ankles along the AP axis appears to be the main contributors. We hypothesized that, in hemiparetic patients, less impaired motor command of hip muscles might play a predominant role in controlling upright stance along both the ML and AP axes, with a relationship between this predominance and the severity of motor and somatosensory deficits. Surprisingly, the predominance of the PD mechanism was preserved in hemiparetic patients along the AP axis, even though it was less efficient in patients with severe distal sensorimotor deficits. In addition, the inability of the non-paretic lower limb to control upright stance (de Haart et al., 2004; Genthon et al., 2008; Mansfield et al., 2012; Singer et al., 2013; van Asseldonk et al., 2006) appears to affect both LU and PD basic mechanisms.

Along the ML axis, this study showed that the LU actions made up the predominant mechanism used to displace CP<sub>Res</sub> positions, in both controls (76%) and hemiparetic patients (80%) even though the latter were the more unstable, as demonstrated by the important CP<sub>Res</sub> standard deviations (Fig. 3). The lack of a significant difference between the two groups implied therefore that LU and PD mechanisms were both similarly altered in hemiparetic patients. Since no correlation was found between the LU contribution and

the clinical deficits, the altered LU mechanism might be due to the biases in the spatial references, frequent and possibly severe after stroke (Barra et al., 2009; Pérennou et al., 2008), raising the question of why the PD mechanism was non-negligible along the ML axis. The combination of three features might explain this finding. First, a covariation mechanism between ML and AP CP displacements may occur due to the 30° toe-out positioning. Second, a whole lateral body tilt (lateropulsion) around the ankle (and possibly the feet) due to a biased body orientation with respect to gravity (Pérennou et al., 2008) may induce lateral CP displacements going in the direction of the body tilt. Third, ankle and foot inversion/eversion controls without body tilt may also induce lateral CP displacements under each foot, with CP displacements under a given foot not compensated for by the CP displacements under the other foot. This segmental action implies precise control of distal muscles, which may be not the case in most hemiparetic patients. Further studies should clarify the respective contribution of these three possible features, in both controls and hemiparetic patients.

Along the AP axis, hemiparetic patients displayed a decreased contribution of the PD mechanisms (88 vs 96%) and consequently an increased contribution of the LU in controlling their balance. The interpretation is based on both biomechanical and clinical factors. In a subject standing with the heels together, a similar sagittal position of both CP (null AP-dist) under the feet makes the control of  $CP_{Res}$  displacements along the AP axis impossible through LU mechanisms. If the CP under each foot are differently positioned along the AP axis (AP-dist) these LU mechanisms along the ML axis induce AP CP displacements. This is usually the case in subjects standing with a weight-bearing asymmetry (Genthon et al., 2008; Geurts et al., 2005). This biomechanical approach explains that hemiparetic patients may partly control their CP displacements along the AP axes by LU mechanisms around the hip in the frontal plane. This novel finding of a greater involvement of the LU mechanisms along the AP axis in hemiparetic patients could be viewed as an adapted response to clinical deficits. Indeed, hypoesthesia, motor weakness, and spasticity correlated with this control strategy, and the lowest  $Contr_{PD}$  values were found in patients with the greatest deficits. This indicates that relying more on proximal hip muscles may be a compensatory strategy for hemiparetics to counterbalance more severely impaired balance control from the ankles. The greater involvement of the LU mechanisms along the AP axis in hemiparetic patients (12 vs 4%) is made possible by a relatively preserved motor command of the trunk muscles due to a bilateral innervation (Carr, Harisson, & Stephens, 1994; Lawrence & Kuypers, 1968a, 1968b).

A surprising finding of our study was the relatively high values of PD contribution indices, even in patients combining very poor distal motor command and deep sensory loss, indicating that the non-paretic foot alone is able to ensure a PD mechanism for controlling a static erect stance. This feature confirms the observation previously reported by Genthon et al. (2008) which highlighted the role of this non-paretic leg, despite evident impairments for the more severe patients, to control the AP postural sway. This strategy is mainly piloted by the non-paretic limb because of the weight-bearing asymmetry and is preferred to a predominant hip control through lateral trunk motion. Considering that ankles are the main joints involved in PD mechanisms, two interpretations may be proposed. First, there is an advantage to remaining with ankle control, even if one ankle is severely impaired. The plantar sole yields a precise mapping of the pressure distribution by the postural soleus muscles known to be specialized in controlling CP positions (Okada & Fujiwara, 1984). For these reasons, controlling AP postural sways by the ankle remains the reference strategy anchored in the human repertoire of postural strategies (Nardone, Giordano, Corra, & Schieppati, 1990; Nashner & McCollum, 1985). Shifting to a new LU mechanism would thus require specific relearning. Second, in this study the imposed foot positioning restrained the sagittal distance between the CP under each foot (AP-dist) and consequently limited the potential involvement of the LU mechanism along the AP axis. At this stage, it is worth noting that the AP-dist parameter is computed from the difference between the two mean positions of each plantar left and right CPs along that AP axis. In our mind, two factors contribute to explain the differences observed between the two groups. Undoubtedly, the first, their respective mean positions, constitutes the main factor. Nonetheless, the second, the respective displacements of the two CPs, might also play a significant role. As emphasized in previous works in both healthy subjects with weight-bearing asymmetry (Genthon & Rougier, 2005) or in hemiparetic patients (Genthon et al., 2008), differences in the amplitudes of the two CPs are noticeable, especially for the latter group.

Lastly, considering the clinical features of hemiparesis, it appears that there are several ways to magnify the AP-dist: either by positioning the paretic foot more forwardly (this option being even amplified by the use of a cane) or by positioning the non-paretic foot more backwardly.

*The negative correlations found between the PD contribution and postural abilities* in daily life and gait capacities offer novel perspectives for the rehabilitation of the upright balance after stroke. Indeed, by highlighting the link between sensorimotor deficits and the impaired PD mechanism for controlling the erect stance, the data from this study evidence the benefits that might be expected by reinforcing the control of the distal muscles involved in setting this mechanism, i.e. the ankles. Analytical rehabilitation protocols, based on voluntary command and force production in both paretic and non-paretic ankles should be regarded as a primary objective of the therapist.

*In conclusion*, although our study was an exploratory study with a limited number of patients, we complete previous findings demonstrating the key contribution of the non-paretic lower limb to compensate for the inability of the paretic limb to control the upright stance (Genthon et al., 2008) and overall show that AP postural sway remains predominantly controlled through PD mechanisms and therefore by the ankles, even in severely impaired patients. From a biomechanical point of view, an increased contribution of the LU mechanism in the AP postural sway control appears to depend on the capacity of the patients to set important AP-dist values. Presently, this AP-dist parameter is computed from the difference between the two mean positions of each plantar left and right CPs along the AP axis. Focusing on the dynamics of this distance, i.e. the capacity of the subjects at a given instant to position with a sagittal gap their two  $CP_{LF}$  and  $CP_{RF}$ , could enlighten this peculiar capacity. Lastly, the voluntary nature of this strategy remains to be determined.

From a clinical point of view, our results have emphasized the main contribution of PD mechanism to control AP sway, even in those combining poor distal motor command and deep sensory loss. This ankle control, piloted by the more-loaded non-paretic limb,

would therefore be preferred to a hip control through lateral trunk motion. This should be considered when defining the objectives of the postural rehabilitation after stroke.

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## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.humov.2019.01.004>.

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