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1 **Upright standing after stroke: how loading-unloading mechanism**  
2 **participates to the postural stabilization**

3

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25

## 1 **Abstract**

2 Postural strategies employed by hemiparetic stroke patients need to be better understood to  
3 guide rehabilitation. Of the two complementary mechanisms used to stabilize the standing  
4 posture, loading-unloading (LU) and pressure distribution (PD), it is hypothesized that the  
5 former would be predominantly used. To this aim, posturographic assessments, through a dual  
6 force-platform, were performed in 30 Hemiparetics tested 3 months after a unilateral stroke,  
7 and 30 matched healthy Controls. Original indices (from 0 to 1) were calculated to assess LU  
8 and PD contributions. The results show that along the mediolateral axis, the LU contribution  
9 was very high and similar in Hemiparetics and in Controls ( $0.80 \pm 0.07$  vs  $0.76 \pm 0.09$  a.u.;  
10  $p > 0.05$ ), indicating a predominant hip involvement. Along the anteroposterior axis, the PD  
11 contribution was very close to 1 in controls ( $0.96 \pm 0.03$  a.u.) indicating an exclusive ankle  
12 involvement. Despite a lower contribution in Hemiparetics ( $0.88 \pm 0.11$  a.u.;  $p < 0.01$ ), the  
13 indices were surprisingly always above 0.5, meaning that ankle movements remain  
14 predominant for controlling postural sways along the anteroposterior axis in all patients even  
15 those with severe clinical deficits. However the PD contribution appeared larger in patients  
16 with light or moderate deficits of the sensitivity ( $r = -0.532$ ;  $p < 0.01$ ) or the motor command ( $r$   
17  $= -0.513$ ;  $p < 0.01$ ). These results indicate that postural stabilization of hemiparetic persons  
18 remains controlled by a PD mechanism along the anteroposterior axis, even in those  
19 combining poor distal motor command and deep sensory loss. This ankle control, piloted by  
20 the more-loaded non-paretic limb, would therefore be preferred to a hip control through  
21 lateral trunk motion. This should be considered when defining the objectives of the postural  
22 rehabilitation after stroke.

23

24

25 **Key words:** stroke; hemiparesis; hip; ankle; postural control; force platform.

26

## 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 (Perennou, Mazibrada, Chauvineau, Greenwood, Rothwell, Gresty, & Bronstein, 2008; Barra Oujamaa, Chauvineau, Rougier, & Pérennou, 2009). 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; van Asseldonk, Buurke, Bloem, Renzenbrink, Nene, van der Helm, & van der Kooij, 2006; Genthon, Rougier, Gissot, Froger, Pelissier, & Perennou, 2008; Mansfield, Mochizuki, Inness, & McIlroy, 2012; Singer, Mansfield, Danells, McIlroy, & Mochizuki, 2013). Indeed, even though in the most impaired patients, the 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, & 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 & McCollum (1985) and extended by Winter et al. (1996) to describe the control of

1 the quiet stance along the AP axis has been widely accepted for several decades, it has so far  
2 aroused little clinical interest, limited to vestibular or peripheral somatosensory deficits  
3 (Horak, Nashner, & Diener, 1990; Lafond, Corriveau, & Prince, 2004). To our knowledge,  
4 this objective to differentiate the respective contribution of these two main joints in the  
5 postural control, never applied on individuals with a brain lesion, appears especially relevant  
6 for at least three reasons. First, the integration of vestibular or somaesthetic information may  
7 be altered depending on the brain areas encroached on by the lesion. This might influence the  
8 selection of the predominant mechanism involved in the stabilization of the erect stance and  
9 therefore the joint: either the hip or the ankle. Second, the motor deficit is generally more  
10 severe for distal muscles (ankle) than for proximal muscles (hip) (Adams, Gandevia, & Skuse,  
11 1990), which should favor a predominant role of the LU mechanisms in stabilizing the erect  
12 stance. Third, the weight-bearing asymmetry often observed in these patients induces opposite  
13 shifts of the plantar CPs, forward for the unloaded paretic limb and backward for the loaded  
14 nonparetic limb (Geurts, de Haart, van Nes, & Duysens, 2005; Genthon et al., 2008).  
15 Regardless of clinical deficits, this forward-backward gap creates the unique condition for  
16  $CP_{Res}$  displacements to covariate along both ML and AP axes through a simplified motor  
17 command involving in larger proportions the hip muscles. To be more precise, we  
18 hypothesized that, in hemiparetic patients, less impaired motor command of hip muscles  
19 might play a predominant role in controlling upright stance along both the ML and AP axes,  
20 with a relationship between this predominance and the severity of motor and somatosensory  
21 deficits. This type of balance control strategy would be in accordance with the hypothesis that  
22 patients suffering from disease-related deficiencies, at their feet and legs, may exaggeratedly  
23 solicit proximal control mechanisms involving their hip (Bonnet & Lepeut, 2011).

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

## 4 5 **2. Methods**

### 6 7 *2.1. Subjects*

8 Thirty hemiparetic patients, with a first hemispheric stroke and impaired balance control, and  
9 30 healthy subjects gave informed consent to participate in accordance with the guidelines of  
10 the local ethics committee. In order to be eligible for the study, patients had to have the  
11 inclusion criteria of a first and unique hemisphere stroke. Exclusion criteria included unstable  
12 status, neuropathy, psychiatric disorders, major comprehension problems due to aphasia,  
13 dementia, signs of vestibular disorders and significant orthopaedic disease. As proposed in  
14 our previous studies (Perennou, Leblond , Amblard , Micallef, Herisson, & Pelissier, 2001,  
15 Genthon et al., 2008), clinical assessments were performed as follows by a trained practitioner  
16 who examine patients at the time of posturography. Motor weakness of eight muscle groups  
17 of the lower limb was assessed manually at hip (flex- ion, extension, abduction, adduction),  
18 knee (flexion, exten- sion), and ankle (flexion, extension) levels through a five-point scale  
19 adapted to patients with central neurological disorders (Held, Pierrot-Desselligny, Bussel,  
20 Perrigot, & Mahler, 1975). Spasticity of 5 muscle groups of the lower limbs (psoas, hip  
21 adductors, quadriceps, hamstring, triceps surae) was assessed using the Ashworth scale  
22 (Ashworth, 1964). Hypoesthesia of the paretic side was assessed through pressure sensitivity  
23 at the pulp of the big toe using the Semmes-Weinstein aesthesiometer (Semmes, Weinstein,  
24 Ghent, & Teuber, 1960). The value of the force needed for a given patient to perceive the  
25 stimulus applied to the skin was then submitted to a log transform to obtain a 20-point linear

1 increasing scale. The severity of the spatial neglect was quantified by a standard scale of  
2 behavioral neglect assessing both body and nonbody spaces (Azouvi, Olivier, de Montety,  
3 Samuel, Louis-Dreyfus, & Tesio, 2003). Balance disorders and gait ability were assessed by  
4 the Postural Assessment Scale for Stroke (PASS, Benaim, Perennou, Villy, Rousseaux, &  
5 Pelissier, 1999) and a six-point walking score (Lindmark & Hamrin, 1988), respectively. All  
6 these clinical characteristics, displayed through table 1 with anthropometric information, are  
7 those usually found in series of patients with a recovery compatible with the maintenance of  
8 the standing posture without help for several minutes after a hemisphere stroke of various size  
9 and location.

10

11 \*\*\*\*\*insert table 1 here

12

### 13 *2.2. Procedure*

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

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### 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 (Winter et al., 1996; Rougier, 2007). 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).

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

1 
$$\text{Contr}_{\text{PD}} = \text{s.d. CP}_{\text{PD}} / (\text{s.d. CP}_{\text{LU}} + \text{s.d. CP}_{\text{PD}})$$

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

5

6 \*\*\*\*\* insert figure 1 here

7

#### 8 *2.4. Statistics*

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

14 
$$Z=1/2\text{Log}[\log(1 + \text{Contr}_{\text{LU}})/\log(1-\text{Contr}_{\text{LU}})]$$
 or 
$$Z=1/2\text{Log}[\log(1 + \text{Contr}_{\text{PD}})/\log(1-\text{Contr}_{\text{PD}})]$$

15

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

21

### 22 **3. Results**

23

1 3.1. *Mean Body-weight distribution.* Hemiparetic patients adopted a spontaneous distribution  
2 of  $63.0 \pm 11.3\%$  over the loaded nonparetic limb. This value was matched by controls who  
3 voluntarily stood asymmetrically ( $65.7 \pm 3.0\%$ ;  $U=319$ ;  $p>0.05$ ).

4  
5 3.2. *CP<sub>Res</sub> mean positions along the AP axis,* expressed relative to the foot length, were  
6 similar in both groups (hemiparetic patients:  $0.44 \pm 0.10$ ; controls:  $0.41 \pm 0.05$ ;  $U= 405$ ;  
7  $p>0.05$ ). Taken together, these two results demonstrate controls' ability to adopt a posture  
8 similar to that of hemiparetic patients with respect to the base of support (Fig. 2).

9  
10 \*\*\*\*\* insert figure 2 here

11  
12 3.3. *CP<sub>Res</sub> dispersion* differed between groups [ $F(1,58)=39.50$ ,  $p<0.01$ ] but not between the  
13 two axes [ $F(1,58)=0.23$ ,  $p>0.05$ ], with an interaction between the two factors [ $F(1,58)=9.38$ ,  
14  $p<0.01$ ]. Post-hoc analyses indicated greater dispersions, thus instability, in hemiparetic  
15 patients than in controls along the ML axis ( $p<0.01$ ) but not along the AP axis (Fig.3). *CP<sub>Res</sub>*  
16 dispersion along the ML and AP axes were correlated in controls ( $r = 0.366$ ;  $p<0.05$ ) but not  
17 in hemiparetic patients ( $r = 0.278$ ,  $p>0.05$ ).

18  
19 \*\*\*\*\* insert figure 3 here

20

1 3.4. *The AP-dist*, which is the distance between mean CP positions under each foot along the  
2 AP axis, was twice greater in hemiparetic patients ( $28.3 \pm 23.8$  mm) than in controls ( $13.7 \pm$   
3  $10.2$  mm,  $U=297$ ;  $p<0.05$ ). This difference was mainly due to a more forward position of the  
4 CP under the paretic limb (Fig. 2).

5

### 6 3.5. *Contribution indices*

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

12

13 \*\*\*\*\* Insert figure 4 here

14

15 Along the ML axis, the  $Contr_{LU}$  indices were high in both groups, indicating the  
16 predominance of LU mechanism. However, and contrary to what was noted along the AP  
17 axis, the values reported for the hemiparetic patients were close to those found in controls  
18 ( $0.80 \pm 0.07$  a.u.; range 0.59–0.89 vs  $0.76 \pm 0.09$  a.u.; range 0.49–0.88;  $p>0.05$ ). No  
19 correlation was found between  $Contr_{LU}$  indices and clinical deficits. These results indicate that  
20 the part devoted to hip and ankle strategies for controlling body stabilization in the frontal  
21 plane was unaffected by clinical deficits in hemiparetic patients.

22

23 \*\*\*\*\* Insert figure 5 here

24

25 Along the AP axis,  $Contr_{PD}$  indices were very close to 1 in controls ( $0.96 \pm 0.03$  a.u.; range,  
26 0.87–1.00), meaning that  $CP_{Res}$  displacements were almost exclusively produced by PD

1 mechanism. Although less PD involvement was found in hemiparetic patients ( $0.88 \pm 0.11$   
2 a.u.;  $p < 0.01$ ), the indices were surprisingly always above 0.5 (range, 0.53–0.98). This meant  
3 that the PD mechanism predominantly controls AP postural sways in all patients, even those  
4 with severe clinical deficits.  $\text{Contr}_{\text{PD}}$  indices were negatively correlated with hypoesthesia ( $r =$   
5  $-0.532$ ;  $p < 0.01$ ), motor weakness ( $r = -0.513$ ;  $p < 0.01$ ), and spasticity ( $r = -0.388$ ;  $p < 0.05$ ),  
6 indicating that the more severe the weakness and sensory loss, the less the PD mechanism  
7 contributed to body stabilization (Fig. 5A-C). Conversely, no significant linear correlation  
8 was found for behavioral neglect ( $r = -0.255$ ;  $p > 0.05$ ). In addition,  $\text{Contr}_{\text{PD}}$  was negatively  
9 correlated with the AP-dist parameter for both controls ( $r = -0.859$ ;  $p < 0.01$ ) and hemiparetic  
10 patients ( $r = -0.816$ ;  $p < 0.01$ ): the greater the AP-dist, the smaller the PD contribution (Fig.  
11 5D). This meant that the LU contribution to body stabilization increased with the distance  
12 between the average positions of the CP under each foot. Conversely, positive correlations  
13 were found with the PASS ( $r = 0.305$ ,  $p < 0.05$ ) and Lindmark scores ( $r = 0.514$ ,  $p < 0.01$ ),  
14 indicating that a more efficient PD mechanism was associated with better balance and gait  
15 capacities (Fig. 5E-F).

16

#### 17 4. Discussion

18

19 This study analyzed the relative role played by the two basic mechanisms, i.e. LU and PD, in  
20 maintaining upright quiet stance and the way they may be affected by sensorimotor  
21 impairments resulting from a stroke. As previously emphasized through a biomechanical  
22 approach (Winter et al., 1996), each joint plays a predominant role in controlling body  
23 movements along a specific axis for a given feet position. When feet are side-by-side, the hips  
24 along the ML axis and the ankles along the AP axis appears to be the main contributors. We  
25 hypothesized that, in hemiparetic patients, less impaired motor command of hip muscles

1 might play a predominant role in controlling upright stance along both the ML and AP axes,  
2 with a relationship between this predominance and the severity of motor and somatosensory  
3 deficits. Surprisingly, the predominance of the PD mechanism was preserved in hemiparetic  
4 patients along the AP axis, even though it was less efficient in patients with severe distal  
5 sensorimotor deficits. In addition, the inability of the non-paretic lower limb to control upright  
6 stance (de Haart et al., 2004; van Asseldonk et al., 2006; Genthon et al., 2008; Mansfield et  
7 al., 2012; Singer et al., 2013) appears to affect both LU and PD basic mechanisms.

8  
9 *Along the ML axis*, this study showed that the LU actions made up the predominant  
10 mechanism used to displace CP<sub>Res</sub> positions, in both controls (76%) and hemiparetic patients  
11 (80%) even though the latter were the more unstable, as demonstrated by the important CP<sub>Res</sub>  
12 standard deviations (Fig.3). The lack of a significant difference between the two groups  
13 implied therefore that LU and PD mechanisms were both similarly altered in hemiparetic  
14 patients. Since no correlation was found between the LU contribution and the clinical deficits,  
15 the altered LU mechanism might be due to the biases in the spatial references, frequent and  
16 possibly severe after stroke (Pérennou et al., 2008; Barra et al., 2009), raising the question of  
17 why the PD mechanism was non-negligible along the ML axis. The combination of three  
18 features might explain this finding. First, a covariation mechanism between ML and AP CP  
19 displacements may occur due to the 30° toe-out positioning. Second, a whole lateral body tilt  
20 (lateropulsion) around the ankle (and possibly the feet) due to a biased body orientation with  
21 respect to gravity (Pérennou et al., 2008) may induce lateral CP displacements going in the  
22 direction of the body tilt. Third, ankle and foot inversion/eversion controls without body tilt  
23 may also induce lateral CP displacements under each foot, with CP displacements under a  
24 given foot not compensated for by the CP displacements under the other foot. This segmental  
25 action implies precise control of distal muscles, which may be not the case in most

1 hemiparetic patients. Further studies should clarify the respective contribution of these three  
2 possible features, in both controls and hemiparetic patients.  
3  
4 *Along the AP axis*, hemiparetic patients displayed a decreased contribution of the PD  
5 mechanisms (88 vs 96%) and consequently an increased contribution of the LU in controlling  
6 their balance. The interpretation is based on both biomechanical and clinical factors. In a  
7 subject standing with the heels together, a similar sagittal position of both CP (null AP-dist)  
8 under the feet makes the control of CP<sub>Res</sub> displacements along the AP axis impossible through  
9 LU mechanisms. If the CP under each foot are differently positioned along the AP axis (AP-  
10 dist) these LU mechanisms along the ML axis induce AP CP displacements. This is usually  
11 the case in subjects standing with a weight-bearing asymmetry (Geurts et al., 2005; Genthon  
12 et al., 2008). This biomechanical approach explains that hemiparetic patients may partly  
13 control their CP displacements along the AP axes by LU mechanisms around the hip in the  
14 frontal plane. This novel finding of a greater involvement of the LU mechanisms along the  
15 AP axis in hemiparetic patients could be viewed as an adapted response to clinical deficits.  
16 Indeed, hypoesthesia, motor weakness, and spasticity correlated with this control strategy, and  
17 the lowest Contr<sub>PD</sub> values were found in patients with the greatest deficits. This indicates that  
18 relying more on proximal hip muscles may be a compensatory strategy for hemiparetics to  
19 counterbalance more severely impaired balance control from the ankles. The greater  
20 involvement of the LU mechanisms along the AP axis in hemiparetic patients (12 vs 4%) is  
21 made possible by a relatively preserved motor command of the trunk muscles due to a  
22 bilateral innervation (Lawrence & Kuypers, 1968a, 1968b; Carr, Harisson, & Stephens, 1994).  
23 A surprising finding of our study was the relatively high values of PD contribution indices,  
24 even in patients combining very poor distal motor command and deep sensory loss, indicating  
25 that the non-paretic foot alone is able to ensure a PD mechanism for controlling a static erect

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

24         Lastly, considering the clinical features of hemiparesis, it appears that there are several  
25 ways to magnify the AP-dist: either by positioning the paretic foot more forwardly (this

1 option being even amplified by the use of a cane) or by positioning the non-paretic foot more  
2 backwardly.

3

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

12

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

25

1 From a clinical point of view, our results have emphasized the main contribution of PD mechanism to  
2 control AP sway, even in those combining poor distal motor command and deep sensory loss. This  
3 ankle control, piloted by the more-loaded non-paretic limb, would therefore be preferred to a hip  
4 control through lateral trunk motion. This should be considered when defining the objectives of the  
5 postural rehabilitation after stroke.  
6

## 1 **References**

- 2 Adams RW, Gandevia SC, & Skuse NF. (1990). The distribution of muscle weakness in  
3 upper motoneuron lesions affecting the lower limb. *Brain*, 113, 1459-1476.
- 4 Ashworth B. (1964). Preliminary trial of carisoprodol in multiple sclerosis. *Practitioner*, 192,  
5 540-542.
- 6 Azouvi P, Olivier S, de Montety G, Samuel C, Louis-Dreyfus A, & Tesio L. (2003).  
7 Behavioral assessment of unilateral neglect: study of the psychometric properties of the  
8 Catherine Bergego Scale. *Archives of Physical Medicine and Rehabilitation*, 84, 51-57.
- 9 Barra J, Oujamaa L, Chauvineau V, Rougier P, & Pérennou D. (2009). Asymmetric standing  
10 posture after stroke is related to a biased egocentric coordinate system. *Neurology*, 72, 1582-  
11 1587.
- 12 Benaim C, Perennou DA, Villy J, Rousseaux M, & Pelissier JY. (1999). Validation of a  
13 standardized assessment of postural control in stroke patients: the Postural Assessment Scale  
14 for Stroke Patients (PASS). *Stroke*, 30, 1862-1868.
- 15 Bonnet CT, & Lepeut M. (2011). Proximal postural control mechanisms may be  
16 exaggeratedly adopted by individuals with peripheral deficiencies: a review. *Journal of Motor*  
17 *Behavior*, 43, 319-328.
- 18 Carr L, Harisson L, & Stephens JA. (1994). Evidence for bilateral innervation of certain  
19 homologous motoneurone pools in man. *Journal of Physiology*, 475, 217-227.
- 20 de Haart M, Geurts AC, Huidekoper SC, Fasotti L, & van Limbeek J. (2004). Recovery of  
21 standing balance in postacute stroke patients: a rehabilitation cohort study. *Archives of*  
22 *Physical Medicine and Rehabilitation*, 85, 886-895.
- 23 Genthon, N., & Rougier P.R. (2005). Influence of an asymmetrical body weight distribution  
24 on the control of undisturbed upright stance. *Journal of Biomechanics*, 38(10), 2037-2049.

1 Genthon N, Rougier P, Gissot AS, Froger J, Pelissier J, & Perennou D. (2008). Contribution  
2 of each lower limb to upright standing in stroke patients. *Stroke*, 39, 1793-1799.

3 Geurts AC, de Haart M, van Nes IJ, & Duysens J. (2005). A review of standing balance  
4 recovery from stroke. *Gait and Posture*, 22, 267-281.

5 Held J, Pierrot-Desselligny E, Bussel B, Perrigot M, & Mahler M. (1975). Devenir des  
6 hémipariés vasculaires vasculaire par atteinte sylvienne en fonction du côté de la lésion.  
7 *Annales de Réadaptation et de Médecine Physique*, 18, 592-604.

8 Horak FB, Nashner LM, & Diener HC. (1990). Postural strategies associated with  
9 somatosensory and vestibular loss. *Experimental Brain Research*, 82, 167-177.

10 Lafond D, Corriveau H, & Prince F. (2004). Postural control mechanisms during quiet  
11 standing in patients with diabetic sensory neuropathy. *Diabetes Care*, 27, 173-178.

12 Lawrence D, & Kuypers H. (1968a). The functional organization of the motor system in the  
13 monkey I. The effects of bilateral pyramidal lesions. *Brain*, 91, 1-14.

14 Lawrence D, & Kuypers H. (1968b). The functional organization of the motor system in the  
15 monkey II. The effects of lesions of the descending brainstem pathways. *Brain*, 91, 15-36.

16 Lindmark B, & Hamrin E. (1988). Evaluation of functional capacity after stroke as a basis for  
17 active intervention. Presentation of a modified chart for motor capacity assessment and its  
18 reliability. *Scandinavian Journal of Rehabilitation Medicine*, 20, 103-109.

19 Mansfield A, Mochizuki G, Inness EL, & McIlroy WE. (2012). Clinical correlates of  
20 between-limb synchronization of standing balance control and falls during inpatient Stroke  
21 rehabilitation. *Neurorehabilitation and Neural Repair*, 26, 627-635.

22 Nardone A, Giordano A, Corra T, & Schieppati M. (1990). Responses of leg muscles in  
23 humans displaced while standing. Effects of types of perturbation and of postural set. *Brain*,  
24 113, 65-84.

1 Nashner LM, & McCollum G. (1985). The organization of human postural movements: A  
2 formal basis and experimental synthesis. *Behavioral and Brain Sciences*, 8, 135-172.

3 Okada M, & Fujiwara K. (1984). Relation between sagittal distribution of the foot pressure in  
4 upright stance and relative EMG magnitude in some leg and foot muscles. *Journal of Human*  
5 *Ergology*,13, 97-105.

6 Pérennou DA, Leblond C, Amblard B, Micallef JP, Herisson C, & Pelissier JY. (2001).  
7 Transcutaneous electric nerve stimulation reduces neglect-related postural instability after  
8 stroke. *Archives of Physical Medicine and Rehabilitation*, 82, 440-448.

9 Pérennou DA, Mazibrada G, Chauvineau V, Greenwood R, Rothwell J, Gresty MA, &  
10 Bronstein AM. (2008). Lateropulsion, pushing and verticality perception in hemisphere  
11 stroke: a causal relationship? *Brain*, 131, 2401-2413.

12 Rougier PR. (2007). Relative contribution of the pressure variations under the feet and body  
13 weight distribution over both legs in the control of upright stance. *Journal of Biomechanics*,  
14 40, 2477-2482.

15 Semmes J, Weinstein S, Ghent L, & Teuber H. (1960). Somatosensory changes after  
16 penetrating brain wounds in man. Cambridge: Harvard University Press.

17 Singer JC, Mansfield A, Danells CJ, McIlroy WE, & Mochizuki G. (2013). The effect of post-  
18 stroke lower-limb spasticity on the control of standing balance: Inter-limb spatial and  
19 temporal synchronisation of centres of pressure. *Clinical Biomechanics*, 28, 921-926.

20 van Asseldonk EH, Buurke JH, Bloem BR, Renzenbrink GJ, Nene AV, van der Helm FC, &  
21 van der Kooij H. (2006). Disentangling the contribution of the paretic and non-paretic ankle  
22 to balance control in stroke patients. *Experimental Neurology*, 201, 441-451.

23 Winter DA, Prince F, Frank JS, Powell C, & Zabjek KF. (1996). Unified theory regarding  
24 A/P and M/L balance in quiet stance. *Journal of Neurophysiology*, 75, 2334-2343.

25

1 **Table and Figure captions**

2

3 Figure 1

4 Top: The two plantar CP displacements, associated with the body-weight distribution (not  
5 displayed on the graph) can be used for computing a resultant center-of-pressure ( $CP_{Res}$ )  
6 trajectory.

7 Bottom: In turn, a  $CP_{Res}$  trajectory can be split into two elementary  $CP_{PD}$  and  $CP_{LU}$   
8 trajectories, each expressing the main contribution of ankle and hip joints to this movement.

9

10 Figure 2

11 Mean positions (with standard deviations) of the plantar ( $CP_{LF}$  and  $CP_{RF}$ ) and  $CP_{Res}$   
12 displacements for the two groups. Note the asymmetric body-weight distribution for both  
13 groups (inferring a lateral shift of the  $CP_{Res}$ ) and the tendency in both groups to shift  
14 forwardly and backwardly on their two plantar CP positions (AP-dist).

15

16 Figure 3

17 Mean standard deviations of the  $CP_{Res}$  movements along both ML and AP axes for the two  
18 groups of subjects (\*\*:  $p > 0.01$ ).

19

20 Figure 4

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

24

25 Figure 5

1

2 A-C and E-F: Scattergrams displaying linear correlation between clinical deficits and the  $\text{Contr}_{\text{PD}}$

3 indices measured along the AP axis. D: Scattergrams displaying linear correlation between AP-dist

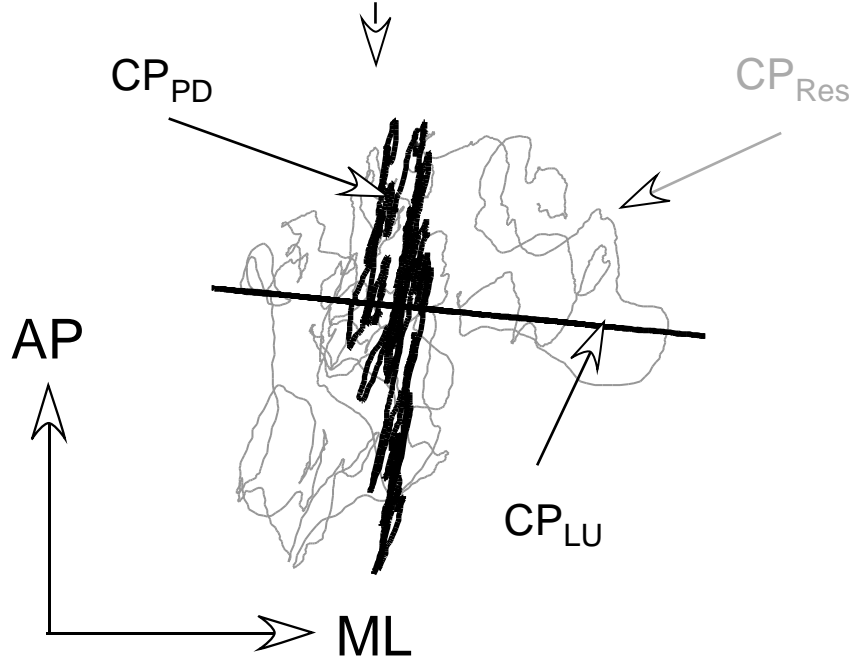
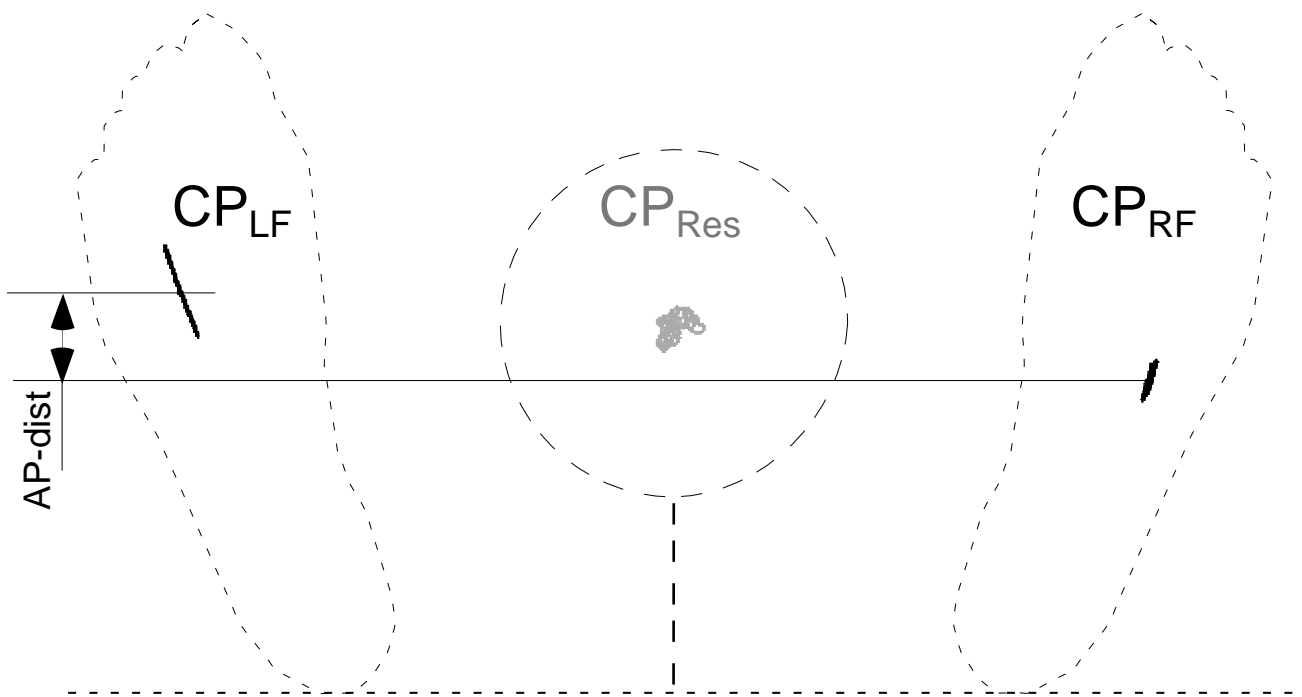
4 values and the  $\text{Contr}_{\text{PD}}$  indices measured along the AP axis for both groups.

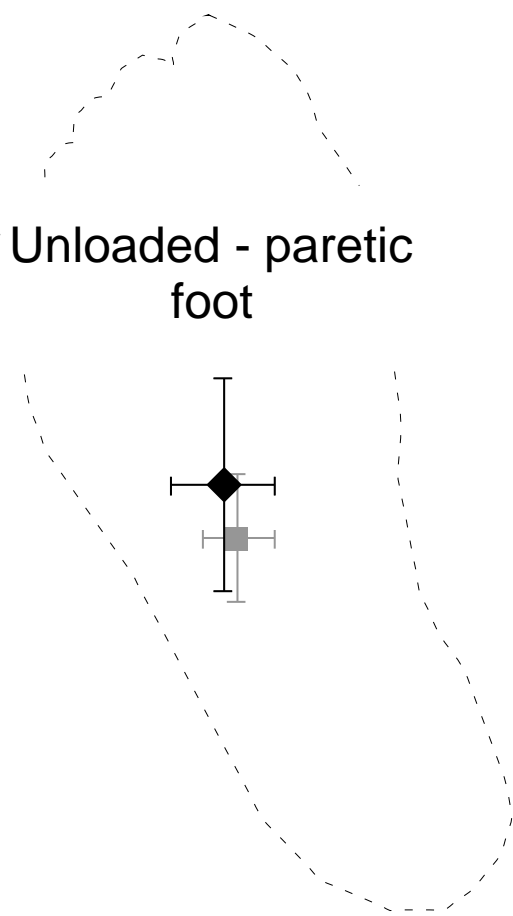
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1 Table 1: Features of participants (mean  $\pm$  s.d.)

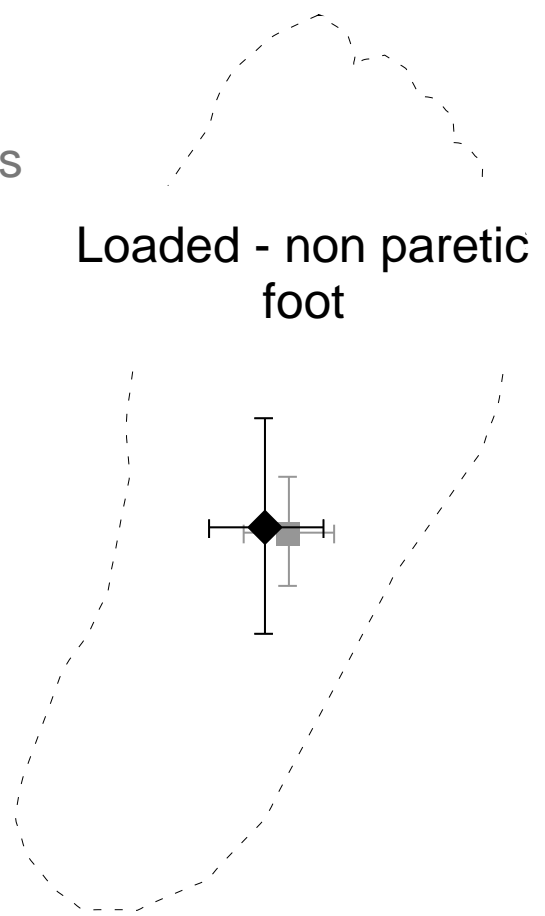
	<b>Healthy subjects</b>	<b>Hemiparetic patients</b>
<b>Sex</b>	20 M; 10 F	22 M; 8 F
<b>Age (years)</b>	59.7 $\pm$ 12.2	60.1 $\pm$ 14.7
<b>Height (m)</b>	171.9 $\pm$ 8.7	168.7 $\pm$ 8.9
<b>Weight (kg)</b>	68.8 $\pm$ 12.9	66.0 $\pm$ 13.1
<b>Time elapsed from stroke (days)</b>		92.0 $\pm$ 48
<b>Stroke side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

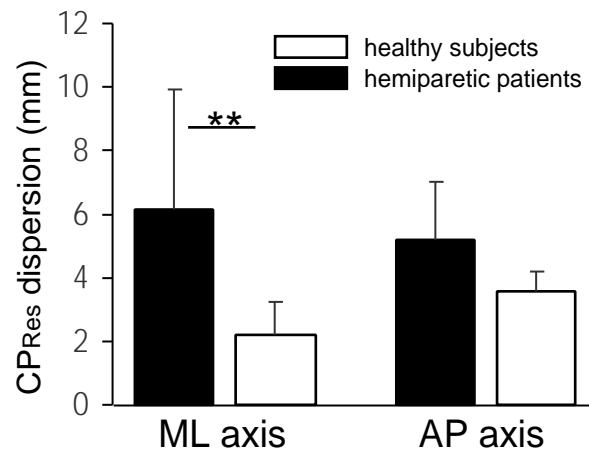
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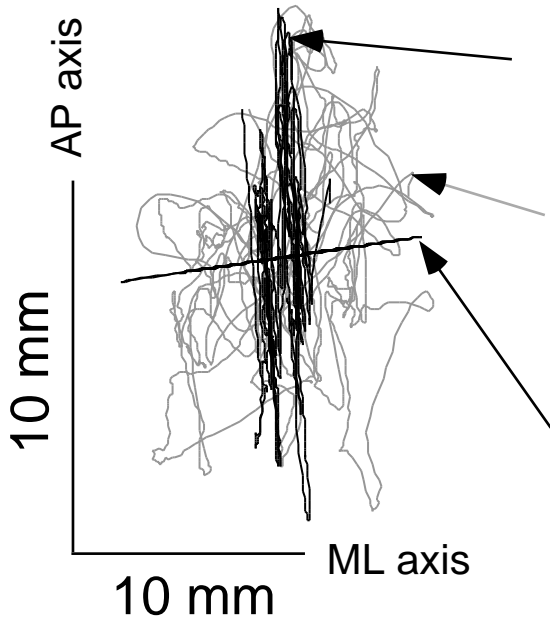


◆ Hemiparetic patients  
■ Healthy subjects





Healthy subject



Hemiparetic patient

