Contribution of Each Lower Limb to Upright Standing in Stroke Patients

Nicolas Genthon, PhD; Patrice Rougier, PhD; Anne-Sophie Gissot, PT, PhD; Jérôme Froger, MD; Jacques Pélissier, MD; Dominic Pérennou, MD, PhD

Background and Purpose—To analyze the postural behavior of standing stroke patients: (1) To differentiate between postural impairment attributable to the neurological condition (deficits attributable to the cerebral lesion) and postural impairment attributable to new mechanical constraints caused by body weight asymmetry; (2) To assess the involvement of each limb in the postural impairment; (3) To better understand which clinical deficits underlie the postural impairment.

Methods—The posturographic characteristics of each limb in 41 stroke patients (first hemispheric stroke: 16 left, 25 right cerebral lesions) required to stand in their preferred posture were compared to those in 40 matched healthy individuals required to stand asymmetrically.

Results—Compared to normal individuals in a similar asymmetrical posture, stroke patients were more unstable. The weight bearing asymmetry and the lateral postural instability were mainly related to spatial neglect. The paretic limb was unable to bring into play a normal longitudinal pattern of the center of pressure, which reflects an impaired stabilization control. Overall postural instability occurred when the strong limb was unable to compensate for the postural impairment of the paretic limb.

Conclusions—The weight bearing asymmetry of standing stroke patients is not the primary cause of their postural imbalance, which is rather the consequence of impaired control of postural stabilization involving both limbs. Weight bearing asymmetry may not be the principle target of rehabilitation programs aiming at restoring standing balance after stroke. Instead it is suggested that more account should be taken of the compensatory role of the strong limb. (Stroke. 2008;39:1793-1799.)

Key Words: posturography ■ spatial neglect ■ weight-bearing asymmetry ■ upright stance ■ postural control
patients known to stand asymmetrically with those of healthy controls standing symmetrically.

To differentiate between postural impairment attributable to the neurological condition (deficits attributable to the cerebral lesion) and postural impairment attributable to new mechanical constraints caused by body weight asymmetry, we compared stroke patients standing in their natural asymmetrical posture to healthy subjects instructed to adopt an asymmetrical weight bearing stance. Their first objective was to analyze the involvement of each limb in the postural imbalance of standing stroke patients. Our second objective was to analyze possible relationships between postural behavior and the clinical features of stroke patients.

Methods

Subjects

Forty-one hemiparetic patients (25 left; 16 right) with a first, recent hemispheric stroke (93.0 ± 46.2 days from onset) and 40 matched healthy subjects participated in the experiment (Table 1). They all gave informed consent in accordance with the guidelines of the local ethics committee. They were able to stand for 40 seconds without technical or human aid. Patients with psychiatric disorders, dementia, orthopaedic diseases, or any deficiency that could affect balance were excluded.

The principal clinical features of the patients were collected before the experiments (means and standard deviations are provided in this section). Eight brain locations (frontal, Rolando’s, parietal and temporal cortices, corona radiata, internal capsule, striatum, and thalamus) were examined using the atlas of Talairach and Tour out and noted as 0 if no lesion was present or 1 if there was a lesion. The mean lesion size, ranging from 0 to 8 was 4.0 ± 2.2. This corresponded to a lesion of intermediate size, affecting on average 4 brain areas. The motor weakness of eight muscle groups of the lower limb was assessed manually on a 5 point scale adapted to patients with central neurological disorders. The mean motor weakness of the weaker limbs, ranging from 0 (normal strength) to 40 (no contraction), was 15.5 ± 10.7. This corresponded to an average strength that was sufficient to mobilize segments of the paretic lower limb against gravity but not against resistance. The spasticity of 5 muscle groups of the lower limbs was assessed using the Ashworth scale. The mean score, ranging from 0 (no spasticity) to 20 (severe and diffused spasticity), was 2.4 ± 2.9. Hypoesthesia of the paretic side was assessed through pressure sensitivity at the pulp of the big toe using the Semmes-Weinstein aesthesiometer. It was on average 5.2 ± 1.4 log F-mg, which corresponded to 14 on a linear scale of hypoesthesia ranging from 1 to 20 (deep hypoesthesia). The severity of the spatial neglect was quantified by a standard scale of behavioral neglect (ranging from 0 to 30) assessing both body and nonbody spaces. The mean neglect score was 3.2 ± 3.7. This apparently low score was attributable to the fact that, as expected 3 months after stroke onset, many patients showed no or mild signs of spatial neglect (especially those with a left stroke), whereas about 1/5 showed moderate to severe spatial neglect. All of these clinical characteristics are those usually found in series of patients with a degree of recovery that is compatible with the maintenance of the standing posture for several minutes without help after a hemisphere stroke of various size and location.

Experimental Procedures

Postural sway was measured by 2 rectangular (21 × 32 cm) force platforms (FF02, Equi+, France) placed side by side. The subjects stood barefoot, each foot placed on one of the 2 platforms (heels separated by 9 cm, toe out at 30°), arms relaxed and hanging freely along the body without orthoses, eyes open looking straight ahead at a fixed target (diameter 30 mm) in an environment composed of vertical lines. They were instructed to sway as little as possible for 4 trials of 32 seconds separated by seated rest periods ranging from 1 to 3 minutes. Stroke patients had to adopt a spontaneous stance whereas controls had to stand asymmetrically with the mean body weight distribution adopted by the stroke patients. Prior to testing, the control subjects performed 1 practice trial to be trained to stand asymmetrically at a required body weight distribution (60% to 65% of body weight on the right lower limb). Auditory feedback was used to guide them to reach this required body weight distribution. Then, no feedback or information was given during the 4 recorded trials. As expected, the body weight distribution adopted by the healthy subjects was comparable to that spontaneously adopted by stroke patients [65 ± 7% versus 62 ± 12%; F(1,158) = 2.50, P = 0.12].

Signal Processing

The reaction forces under both lower limbs were simultaneously recorded on a personal computer from 4 vertical dynamometric load cells placed under each platform (range of measurements 0 to 150 daN). The signals generated by the 8 load cells were amplified and converted from analog to digital form through a 14 bit acquisition card and then recorded at a frequency of 64 Hz (without any filtering).

The CP trajectories under each foot were calculated from the vertical reaction forces measured by the 4 load cells of each platform. The CP uf, displacements were then computed from the CP displacements under each foot and from the body weight distribution between the supports:

\[
CP_{\text{uf}} = CP_{\text{uf}}/R_{\text{uf}} + CP_{\text{lf}}/R_{\text{lf}}
\]

where CP uf, CP lf, were the center of the unloaded (uf) and the loaded (lf) foot, respectively. The sum of R uf, R lf corresponded to the total body weight.

Data Analysis

The mean CP positions (CP uf, CP lf, CP b) were calculated with regard to a frame of reference defined by the axis passing behind the heels (ML axis) and the sagittal axis passing between the 2 feet (AP axis). Positive values indicate that the CP was situated forward of the ML axis and to the right of the AP axis.

The temporal and spatial characteristics of the different displacements were evaluated through a frequency analysis. The CP trajectories (CP uf, CP lf, CP b) were converted into the frequency domain through fast Fourier transforms to obtain the amplitude distribution as a function of the frequency. The frequency spectrums were then characterized by 2 parameters: the mean amplitude (MA), quantifying the range of the motions independently of the frequency, and the mean power frequency (MPF), representing the mean frequency and thus the mean time for these motions to return to an identical position. Both parameters were calculated in the 0 to 3Hz bandwidth. As each foot was at an angle of 15° with regard to the ML/AP plane of reference, the frequency analysis was performed independently of each axis by calculating the total CP trajectories under each foot on a planar basis. In contrast, CP b trajectories along the ML and AP
distributions were normal, parametric statistics were performed using 2 factor ANOVAs. Relationships between clinical and posturographic parameters were first analyzed using Pearson correlations then by multiple regression to classify significant relationships between clinical and experimental data, and to identify independent relationships. The first level of significance for all tests was set at \( P<0.05 \). All data are presented as mean±SD. Because of the \(-1\) to \(1\) range of the LR calculated for CPuf and CPlf trajectories, statistical tests were done on \( z \) transforms to normalize these distributions such that: 

\[
z = \frac{1}{2} \log \left( \frac{1+LR}{1-LR} \right)
\]

### Results

**Comparison Stroke/Control Subjects: CP\(_{\text{Res}}\) Characteristics**

#### Mean CP\(_{\text{Res}}\) Positions

The ANOVA showed no group effect (\( F(1,158)=0.65, P=0.42 \)), an expected axis effect (\( F(1,158)=134.59, P<0.001 \)), and no interaction between group and axes (\( F(1,158)=0.22, P=0.64 \)). The mean CP\(_{\text{Res}}\) positions were similar in control and stroke subjects whatever the axis (Figure 2).

#### CP\(_{\text{Res}}\) Mean Amplitude

The ANOVA revealed a group effect (\( F(1,158)=30.76, P<0.001 \)), no axis effect (\( F(1,158)=0.19, P=0.66 \)), and an interaction between group and axis factors (\( F(1,158)=4.24, P<0.05 \)). The mean amplitude of the CP\(_{\text{Res}}\) displacements was greater for stroke patients along both ML (\( P<0.001 \)) and AP (\( P<0.01 \)) axes. The interaction was attributable to the fact that displacements were greater along the ML than the AP axis in stroke patients, whereas the reverse was true in healthy subjects (Table 2, upper part and Figure 3).

#### CP\(_{\text{Res}}\) Mean Frequency

The ANOVA showed a group effect (\( F(1,158)=5.12, P=0.05 \)), an axis effect (\( F(1,158)=13.48, P<0.001 \)), and no interaction between group and axis factors (\( F(1,158)=2.21, P=0.14 \)). The mean frequency of the CP\(_{\text{Res}}\) trajectory was greater in patients than in controls and greater along the AP axis than along the ML axis.

### Characteristics of the CP Under Each Foot

#### Mean CP Positions Under Each Foot

Along the ML axis, the ANOVA showed a group effect (\( F(1,158)=3.89, P=0.05 \)), a foot effect (\( F(1,158)=7.54, P<0.01 \)), and no interaction between group and foot factors (\( F(1,158)=1.26, P=0.26 \)). In stroke patients, the CP positions under each foot were more laterally located than in control subjects. Moreover in absolute values, the CP of the unloaded foot was more laterally located than that of the loaded foot, in both groups. Along the AP axis, the ANOVA showed no group effect (\( F(1,158)=2.03, P=0.16 \)), a foot effect (\( F(1,158)=7.42, P<0.01 \)), and no interaction between group and foot (\( F(1,158)=0.39, P=0.53 \)). In both groups, the mean CP position of the unloaded foot (CP\(_u\)) was more forwardly located than that of the loaded foot (CP\(_l\)) (Figure 2).
Mean Amplitude of the CP Under Each Foot
The ANOVA showed a group effect (F(1,158)=20.94, P<0.001), a foot effect (F(1,158)=6.26, P<0.01), and an interaction between group and foot factors (F(1,158)=4.45, P<0.05). In stroke patients, the mean amplitude of the loaded CP trajectories was greater than the MA of the unloaded foot and than the MA of both limbs of control subjects (Table 2, lower part and Figure 3).

Mean Frequency of the CP Under Each Foot
The ANOVA showed a group effect (F(1,158)=9.66, P<0.01), no foot effect (F(1,158)=1.79, P=0.18), and no interaction between group and foot factors (F(1,158)=1.62, P=0.20). The mean frequencies of the CP trajectories under each foot were larger in stroke patients than in controls (Table 2, lower part).

Table 2. Parameters From Frequency Analysis Applied on Resultant (Upper Part) and on Plantar (Lower Part) CP Displacements Are Shown for the Control Group and the Stroke Patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>Stroke Patients</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPre trajectories</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean amplitude, mm</td>
<td>0.41±0.25</td>
<td>0.17±0.10</td>
</tr>
<tr>
<td>MPF, Hz</td>
<td>0.66±0.12</td>
<td>0.64±0.07</td>
</tr>
<tr>
<td>CP trajectories under each foot</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean amplitude, mm</td>
<td>0.51±0.32</td>
<td>0.26±0.16</td>
</tr>
<tr>
<td>MPF, Hz</td>
<td>0.76±0.13</td>
<td>0.67±0.09</td>
</tr>
</tbody>
</table>

Lengthening Ratio of the CP Under Each Foot
The ANOVA showed a group effect (F(1,158)=11.47, P<0.001), a foot effect (F(1,158)=14.78, P<0.001), and an interaction between group and foot factors (F(1,158)=6.43, P<0.01). The control group was characterized by an LR close to 0.8, revealing that the 2 CP trajectories were 8 times greater along the longitudinal axis of the foot than along the lateral axis. Stroke patients were characterized by a reduced LR under the unloaded foot (P<0.001), revealing that the CP trajectories under the paretic foot were less structured along the longitudinal foot axis (Figure 3).

Correlations Between Clinical and Posturographic Features in Stroke Patients
Data are shown in Table 3. The more unloaded the paretic limb, the greater the severity of behavioral neglect, hypoesthesia, motor weakness and spasticity. Thirty-two percent (r²=0.32) of the weight bearing asymmetry was explained by the model extracted from multiple regression analysis (r=0.56, F(4,36)=4.20, P<0.01). Spatial neglect was the best predictor of weight bearing asymmetry (β=-0.29, P<0.05). Coefficients of motor weakness, spasticity, and hypoesthesia were not significant (β=0.01, P=0.95; β=-0.32, P=0.07; β=-0.20, P=0.20, respectively) indicating that these variables did not independently contribute to the model.

Behavioral neglect, hypoesthesia, level of spasticity, and motor weakness also correlated with lateral instability (CPRes MA in ML axis), but not with anterior-posterior instability. The multiple regression model was significant (r=0.55, F(4,36)=3.9, P<0.01), with behavioral neglect as the best predictor of lateral instability (β=0.31, P<0.05). Coefficients of motor weakness (β=0.14, P=0.45), spasticity (β=0.21, P=0.21), and hypoesthesia (β=0.13, P=0.43) were not significant.
Discussion

To clarify the origin of the postural disorders of stroke patients, the reaction forces under each limb were measured separately and their postural behavior compared to that of a control group adopting similar body weight asymmetry. This approach revealed 3 important findings: (1) weight bearing asymmetry could not totally explain the postural impairment of stroke patients; (2) the pattern of CP trajectories under the paretic foot was perturbed and associated with increased displacements under the nonparetic foot. This may reflect the limited participation of the paretic lower limb in postural stabilization, and involvement of the nonparetic limb in postural instability; (3) spatial neglect was the clinical feature which correlated most strongly with lateral orientation and instability.

Beyond Weight Bearing Asymmetry

The difference between the 2 feet not only concerned the part of loaded body weight, but also the location and the orientation of CP trajectories.

In accordance with previous studies the CP was found more forwardly located (about 2 cm) under the unloaded paretic limb than under the loaded strong limb. The fact that this forward shift was also reported in asymmetrical healthy subjects clearly indicates that this feature is attributable to asymmetrical body weight distribution on the lower limbs, and not to neurological deficits, such as spasticity.

Ankle joints are principally involved in the sagittal stabilization of quiet upright standing. Planter CP displacements are mainly controlled along the longitudinal axis of the foot by using ankle flexor/extensor muscles. In the present study, a lengthening ratio (LR) was computed to give further insights into the way in which CP displacements are distributed along the sagittal and lateral foot axes. In patients, the LR measured under paretic and nonparetic limbs were extremely different, indicating different patterns for plantar CP displacements. Whereas the similarity between the LR measured under the nonparetic limb and that found in normal subjects indicates an appropriate development of a longitudinal pattern in the foot, the longitudinal excursion of the paretic CP was very restricted. This reflects the limited participation of the paretic limb in body stabilization.

The Nonparetic Limb Was Involved in Postural Instability

Plantar CP displacements in stroke patients were greater than those in normal subjects. This feature cannot be explained here by body weight distribution. Moreover, CP dispersion measured under the nonparetic limb was greater than that in normal subjects.

Table 3. Pearson Coefficients of Correlation Between the Percentage of Body wt on the Paretic Foot, the Mean Amplitude of the CPRes Trajectories Along ML and AP Axes With the Clinical Features of Stroke Patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>% Body wt on the Paretic Limb</th>
<th>MA for the Resultant CP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>ML</td>
</tr>
<tr>
<td>Age</td>
<td>0.14</td>
<td>0.09</td>
</tr>
<tr>
<td>Delay from stroke onset</td>
<td>-0.37*</td>
<td>0.11</td>
</tr>
<tr>
<td>Lesion size</td>
<td>-0.30</td>
<td>0.32</td>
</tr>
<tr>
<td>Motor weakness</td>
<td>-0.33*</td>
<td>0.39**</td>
</tr>
<tr>
<td>Spasticity</td>
<td>-0.40**</td>
<td>-0.40**</td>
</tr>
<tr>
<td>Hypoesthesia</td>
<td>-0.40**</td>
<td>0.35*</td>
</tr>
<tr>
<td>Behavioural neglect</td>
<td>-0.41**</td>
<td>0.45**</td>
</tr>
</tbody>
</table>

Statistically significant correlations are in bold with *P<0.05, **P<0.01.
measured under the paretic limb and was also greater than that found under the loaded limb in controls.

From a clinical point of view, this behavior could be considered as an adaptive process to compensate for the inability of the paretic limb to control upright stance. Engardt et al. were among the first to speculate that patients may favor the nonparetic limb for reasons of safety and speed, thus resulting in “disuse” of the paretic limb. These greater movements reveal stronger muscle contractions and a wider range of motion under the nonparetic limb in comparison to the paretic one. By increasing the somatosensory input and the muscular output intervening on the nonparetic limb, this adaptive postural strategy could thus be viewed as a way to facilitate maintenance of the upright stance.

In parallel, with regard to CP_res trajectories, the greater plantar CP displacements combined with the considerable body weight applied on the limb necessarily result in a huge increase in CP_res dispersion. Indeed, the computation of CP_res positions takes into account both the magnitude of the plantar reaction forces and the position of their application points (see methods). Our results indicate that the postural instability of standing stroke patients resulted partly from the inability of the nonparetic limb to completely stabilize the body.

The postural strategy adopted by hemiparetics could be viewed as a compromise between relieving their paretic limb, which seems to be unable to control upright stance, and loading their nonparetic limb as much as possible. However the nonparetic limb cannot adequately control this constraint. One may wonder whether an impaired ipsilesional motor function involving ipsilateral motor pathways or an altered body scheme often related to spatial neglect could alter stabilization capacities of the nonparetic limb.

In this foot positioning, ankle mechanisms play a major role in the generation of CP_res displacements along the antero-posterior axis, whereas hip mechanisms predominate in displacements along the ML axis. The increased CP_res displacements mainly occurring along the ML axis should thus be viewed as deterioration in hip mechanisms. On the other hand, the increased plantar CP displacements under the nonparetic limb are likely the main cause of the increased CP_res displacements along the AP axis. The constancy of the MPF measured for plantar CP displacements under both limbs indicate that the mean time for the plantar CP to return to its initial position is not influenced by the magnitudes of their displacements, hence suggesting a regulatory process at the level of the ankle to secure equally fast CP changes under both feet. Further understanding of these mechanisms could be provided by analyzing plantar CP trajectories along the ML and AP axes separately.

**Relationship Between Postural Impairment and Clinical Features**

Few studies have investigated the possible relationship between the clinical deficits of stroke patients and their postural characteristics. In the present study, it was found that spatial neglect was the best predictor of postural imbalance, accounting for both weight bearing asymmetry and lateral postural instability. These findings could be considered surprising given the low mean score of spatial neglect. In fact, data distribution was scattered with many patients showing no signs of spatial neglect, especially those with a left stroke, but about 1/5 showing mild to severe spatial neglect. The strong relationship between postural imbalance and spatial neglect found in our study on the standing posture confirms and builds on the findings of several previous studies that focused on the sitting posture or even postural control in daily life.

Spatial neglect may be interpreted as the existence of distortion in the coordinates used to distribute body weight over the 2 limbs while standing. Postural control is organized on the basis of an internal(s) model(s) which closely deals with the body scheme. Neglect may be associated with a disruption of, or failure to attend to, the body scheme. It has been suggested that the representation of the mid sagittal plane, used as a postural reference, could be shifted toward the lesion-side in some stroke patients, explaining the weight bearing asymmetry. A contralesional rotation in the representation of the long body axis (LBA), still in relation with spatial neglect, could also be involved in this weight bearing asymmetry. Indeed, a tilted LBA implies an equal distribution of body mass about this erroneous egocentric reference frame, thus an unequal distribution of body mass about a normal midsagittal plan. On the basis of previous studies and also on the basis of the present one, it can be stated that the pronounced postural disorders of some stroke patients constitute a basic manifestation of spatial neglect.

**Conclusion**

This study emphasizes the complexity of postural imbalance in stroke patients and confirms the key role of spatial neglect in their postural disorders. Two important findings are revealed: (1) the paretic limb seems to be unable to control upright stance, acting more passively than an articulated limb. Consequently, patients are constrained to build an adaptive strategy consisting in involvement of their nonparetic limb. (2) The nonparetic limb may be unable to completely compensate for the insufficiency of the paretic limb and may consequently be involved in the overall postural instability.

**Disclosures**

None.

**References**


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