Biomechanical assessment of the sitting posture maintenance in patients with stroke

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Abstract

Background and purpose. Regaining control of sitting posture is one of the first goals in the rehabilitation of patients with stroke. So, it requires a precise quantification of this postural behaviour. The purpose of the present investigation was thus to assess postural control during sitting in people with hemiparesis through a biomechanical analysis.

Methods. Centre-of-pressure displacements were recorded by means of a force platform on which 10 patients with stroke and 10 age-matched healthy subjects were sitting. Centre-of-pressure trajectories were processed through space-time and frequency analyses.

Results. These centre-of-pressure displacements of the patients with stroke were characterised by an increased control for maintaining sit position and by reduced postural performance, as enlightened by the larger surfaces covered by the centre-of-pressure displacements (P < .05) and increased velocities (P < .001), respectively. As shown from the frequency analysis, the impairment have affected predominantly the displacements occurring along the antero-posterior axis (P < .05).

Interpretations. The analysis of centre-of-pressure displacements during sitting posture indicates an increased postural disturbance in patients with stroke. A platform device, because of the non-invasive, easy and fast measures carried out, should thus be viewed as an attractive tool for assessing the postural dysfunctioning encountered in sat patients with stroke. This tool could also be used for evaluating, the rehabilitation process following stroke.

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1. Introduction

Trunk muscles are mostly involved in the construction of sitting posture and then in those of more complex abilities such as reaching, upright standing or walking. Following stroke, it is generally accepted that the control of trunk muscle is severely impaired (Bohannon et al., 1995; Davies, 1990; Olney and Martin, 1997). More precisely, even though the loss of muscular strength is larger for the parietic side in comparison to the non-parietic one, a lower isometric strength in bilateral trunk muscles is however observed (Bertrand and Bourbonnais, 2001; Bohannon et al., 1995; Gauthier et al., 1992). Also, patients are generally characterised by a contralateral somatosensory deficit, inducing a dysfunctioning in the construction of the body scheme (Perennou et al., 1998), an hemianopsia and some reorganisation on the sensory collection strategy (Bonan et al., 2004; Di Fabio and Badke, 1990).

As a consequence, independent sitting of patients with stroke is generally disturbed. For instance, a prospective study involving 93 patients (Mayo et al., 1991) has shown that 48% and 27% of the patients with stroke were unable to sit independently at the onset and the end of the rehabilitation process, respectively. These dysfunctions are suggested to be caused by some perceptual impairments (Mayo et al., 1991) and motor loss (Franchignoni et al.,...
1997; Hsieh et al., 2002). Due to the importance of trunk control in more complex abilities, restoration of sitting posture appears determinant for recovering independent functions (Franchignoni et al., 1997; Hsieh et al., 2002; Wade et al., 1983). In other words, the observation of decreased sitting postural control capacities at an earlier stage following stroke is generally associated with a poor subsequent functional recovery. Consequently, sitting postural control must be one of the main goals in the rehabilitation process following stroke and needs to be accurately investigated.

Following stroke, functional evaluation of the trunk function or of independent sitting is classically performed through clinical evaluations such as the capacity to maintain the sitting posture for a few seconds without falling (Bohannon et al., 1986) or through a clinical scale, such as the trunk control test (TCT) (Franchignoni et al., 1997; Hsieh et al., 2002), the postural assessment scale for stroke (PASS) (Benaim et al., 1999; Hsieh et al., 2002), or the Fugl-Meyer test (Fugl-Meyer et al., 1975; Hsieh et al., 2002). These procedures underline the major impairments characterising both static and dynamic sitting behaviours resulting from stroke. On the other hand, these methods can be operator dependent, and necessitate an apprenticeship. Another flaw of such methods lies in the impossibility to quantify precisely the postural behaviour during undisturbed sitting posture (Sandin and Smith, 1990), which is the necessary ability for building more complex tasks such as reaching, standing still upright or walking. The classical method used to assess the amount of postural sway encountered in undisturbed upright stance has, for many decades, consisted in measuring, using a force platform, the trajectories of the centre-of-pressure (CoP), i.e. the successive points of application of the resultant ground reaction forces. Interestingly, a force platform furnishes interesting insights for evaluating upright stance of both healthy subjects and people with stroke (Geurts et al., 2005; Mizrahi et al., 1989; Shumway-Cook et al., 1988).

Upright standing and sitting position maintenance are both closed tasks. Even though the biomechanical constraints applied to the human body structure are slightly different, these two tasks share many features such as countering gravity and preparing the body to move. These objectives could be achieved by using different sensory cues (somatosensory, visual, and labyrinthic), integrated at different central levels (spinal and cortical). Differences between upright and sitting postures concern principally: the localisation of cutaneous inputs (principally collected from the feet in upright standing and from buttock in sitting position), the number of segments and the type of segment to be controlled (lower and upper limb muscles in upright and sit positions, respectively).

The purpose of the present investigation was thus to quantify postural control during sitting in people with stroke through a biomechanical analysis generally used to assess upright undisturbed stance. Accordingly, CoP displacements were recorded during quiet sitting with a force platform. In parallel, a group of matched young healthy subjects was also tested. Due to the major sensory and motor impairments characterising the patients with stroke, it was thus hypothesised that hemiparetics would exhibit a decreased postural control during sitting as compared to the control group. This impairment should be characterised by increased displacements and velocities of the CoP. Furthermore, given the importance of muscular force loss in the trunk flexor/extensor muscles following stroke (Bohannon et al., 1995), discrepancies in the induced effects according to the frontal and sagittal planes could be expected consisting in a decreased postural control occurring mainly along the AP axis.

2. Methods

2.1. Subjects

Patients and healthy subjects enrolled in the study gave informed consent prior the study. Ten patients with hemiparesis who had suffered a first stroke and 10 healthy subjects, of similar age, took part in the study. The patients were admitted to the hospital for rehabilitation and met the following criteria: (1) adults of more 55, (2) stroke of less than 3 months, (3) medical stability, (4) ability to understand instructions and to execute them adequately. Patients with (1) psychiatric disorders or dementia, (2) orthopaedic diseases that could affect balance were excluded. The present study was conducted with a mean time delay of 19.8 (SD: 9.9) days after stroke. Summary of clinical data are given through Table 1.

2.2. Task and procedure

CoP displacements were measured by an equilateral triangular (each side = 80 cm) force platform (PF01, Equi+, Aix les Bains, France) on which the subjects were sitting. The platform was positioned on a rigid table border one meter above the floor. The subjects sat directly on the platform, back unsupported, feet not touching the ground, popliteus hollows on the platform border, shanks and feet hanging, arms crossed on the abdomen, and facing a white wall 1.5 m in front of them (see Genthon and Rougier

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Summary of the characteristics for the control groups and patients with stroke (mean (SD))</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>71.1 (11.17)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>70.9 (7.7)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.70 (0.05)</td>
</tr>
<tr>
<td>Sex (f/m)</td>
<td>5f, 5 m</td>
</tr>
<tr>
<td>Lesion location</td>
<td>l/r; r/l</td>
</tr>
</tbody>
</table>

The two groups are paired for the age, weight and height.
(2006) for an illustration of the positioning). Patients were evaluated during five successive trials each lasting 32 s, during which they were required to move as little as possible. The rest periods between the trials lasted more than 1 min.

2.3. Signal processing

The ground reaction forces, issued from three vertical mono-axial dynamometric load cells (range: 0–50 daN), were simultaneously recorded with a 64 Hz frequency on a personal computer during the tests (without any filtering). The signals were then amplified and converted from analogue to digital form through a 12 bits acquisition card, CoP displacements along medio-lateral (ML) and antero-posterior (AP) axes were calculated from ground reaction forces.

At a first step, the planar CoP movements were analysed through a classical analysis including computations of the surface area (Tagaki et al., 1985) and mean velocity. Temporal and spatial characteristics of the CoP movements along the ML and AP axes were evaluated through a frequency analysis. This methodology offers different interests: (i) an independence of the evaluation modalities and more precisely of the duration of the acquisition. (ii) A characterisation of both CoP spatial and temporal characteristics along both ML and AP axes. This methodology consists, at a first step, to convert CoP movements in the frequency domain through a fast Fourier transform for obtaining the amplitude distribution as a function of the frequency. The frequency spectra were then characterised, on a 0–3 Hz bandwidth, by some parameters such as the root mean square (RMS) and the mean power frequency (MPF). The former quantifies the range of the movements independently of the frequency whilst the latter represents its mean frequency, i.e. the mean time for these movements to return to an identical position

\[
\text{RMS} = \sqrt{\frac{\sum_{i=k}^{k} A_i^2}{k}} \\
\text{MPF} = \frac{\sum_{i=j}^{i-k} (S_i \times A_i)}{\sum_{i=j}^{i-k} A_i}
\]

where \( i \) represents the frequency class, \( j \) and \( k \) the range of the frequency bandwidth, \( A_i \) the range of the class and \( S_i \) its median frequency.

2.4. Statistical analysis

For each dependant variable and each group, the mean and standard deviation were computed. Data from both control subjects and patients with stroke were compared through a non-parametric statistical analysis based on the ranks: the non-paired Mann–Whitney \( U \)-test. On the other hand, the results for each subject along ML and AP axes were assessed through a paired Wilcoxon \( T \)-test. In both cases, the first level of significance was set at \( P < .05 \).

3. Results

3.1. Space-time domain analysis

Representative CoP displacements obtained from a typical control subject and a patients with stroke are illustrated through Fig. 1a. Patients with stroke exhibited larger surface area covered by the CoP displacements (\( U = 21, P > .05 \); Fig. 1b) and larger mean velocities (\( U = 6, P < .001 \); Fig. 1c) than the control subjects.

3.2. Frequency domain

A representative frequency decomposition of the CoP displacements obtained from a typical control subject and a patients with stroke is illustrated through Fig. 2a. In comparison to the control group, the RMS from the patients with stroke remained unchanged along the ML axis (\( U = 36, P > .05 \); Fig. 2b), whereas larger values were observed along the AP one (\( U = 23, P < .05 \); Fig. 2b). On the other hand, RMS measured from ML and AP axes were not different, this fact being noticed for both healthy subjects and patients with stroke (\( T = 19, P > .05 \); \( T = 11, P > .05 \); respectively).

No difference was observed between healthy subjects and patients with stroke for the MPF for both ML and AP axes (\( U = 47, P > .05 \); \( U = 25, P > .05 \), respectively) (Fig. 2c).

Fig. 1. (a) Representative CoP displacements obtained from a typical control subjects (black line) and patient with stroke (grey line). Note the dispersion of the CoP movements of the patients with stroke in comparison to those of the control subject. Means and standard deviations of the surface area (b) and CoP velocity (c) characterising healthy and patients with stroke CoP displacements. The statistically significant values between patients with stroke and control group are reported (* \( P < .05 \); ** * \( P < .001 \)).
4. Discussion

The purpose of the present study was to investigate the sitting posture by using a system classically used for analysing upright undisturbed stance: a force platform. In particular, this study was aimed to analyse biomechanically the sitting posture following stroke through the CoP displacements.

Until now, such a biomechanical analysis was mostly used to assess undisturbed upright stance maintenance. Upright stance is regulated by multiple sensory cues including vision, vestibular, proprioceptive and tactile plantar inputs. In undisturbed sitting position some sensory and mechanical changes have to be emphasised. At a sensory level, it is easy to see that both tactile plantar and leg somatosensorial thresholds are ineffective in the sitting position regulation whereas tactile buttock inputs are used. At a motor level, centre of gravity height is reduced, the base of support is larger and the number of joints to be controlled is reduced. Sitting position is indeed principally controlled by the trunk muscles mobilising the pelvic joints. Despite of these differences, the control involved in sitting position presents the same characteristics to those observed in upright standing, suggesting that both sitting and upright postures have similar mechanical characteristics and/or are controlled by a same central behaviour (Genthon and Rougier, 2006). Nevertheless, due to the reduced biomechanical constraints, CoP magnitudes are smaller in sitting position than in upright standing (Genthon and Rougier, 2006). This feature could be interpreted as the result of reduced co-contractions involving agonistic and antagonistic muscles in sitting position stabilisation.

Concerning patients with stroke sitting behaviour, a larger disturbance, characterised by an increase of both CoP areas and mean velocity, was observed in our patients tested early after stroke (20 days), as compared to the healthy ones. These results are in accordance with our hypothesis and with previous studies on patients with stroke sitting posture based on clinical scales. Whatever the test used, TCT (Franchignoni et al., 1997; Hsieh et al., 2002), PASS (Hsieh et al., 2002) or Fugl-Meyer (Hsieh et al., 2002), patients with stroke exhibit some minor scores, hence revealing some disability in trunk function (flexion/extension; adduction/abduction, transfer) and in maintaining sitting position (dynamic and undisturbed). The body scheme misrepresentation (Perennou et al., 1998) and the loss of force in the trunk musculature (Bohannon et al., 1995; Davies, 1990; Olney and Martin, 1997) generally observed following stroke might easily explain these impairments.

Interestingly, a biomechanical evaluation of undisturbed sitting posture gives more insights than does the clinical evaluation. In previous studies, the area covered by the CoP excursion was considered as an indicator of postural performance, defined by some reduced body oscillations. Rigorously, this conception is not totally correct. CoP trajectory is the net product of the muscular action developed in order to controlled body stability. Postural stability could be better quantified by the centre of gravity (CG) movements, which express body sways. It is well known
that CG movements are directly controlled by the CoP displacements. If CoP frequency displacements remain unchanged, enhanced CoP trajectories are already associated with enhanced CG movements (Breniere, 1996). In our case, because the CoP frequency bandwidths of patients with stroke and healthy subjects are similar, enhanced CoP trajectories observed following stroke could be interpreted as a reduced postural performance.

The mean velocity of CoP displacements for the sampled period is generally considered as a postural activity index (Geurts et al., 1993; Maki et al., 1990). It can be viewed as representing the amount of activity required to maintain a given posture. Along these lines, our tested patients with stroke, which are characterised by enhanced CoP velocity, would involve an increased activity to maintain finally a more unstable sitting posture. Furthermore, it is admitted that the stability limits of patients with stroke, which are defined as the maximal CoP positioning that patients are able to maintain without falling, are reduced during sitting posture (Nichols et al., 1996). Moving the CoP faster near these stability limits can thus be considered as an important indicator of risk of falling (Pai and Patton, 1997).

More precisely, our frequency analysis reveals enhanced CoP displacements along the AP axis 20 days following stroke as compared to those along the ML one. These results suggest that the sagittal equilibrium would be more impaired than the frontal one. It could be interpreted as a larger impairment in the bilateral antagonistic recruitment of the rectus abdominis and latissimus dorsi muscles, principally involved in the control of the flexion and extension of the trunk, respectively, than in the bilateral antagonistic recruitment of the left and right obliques abdominis externi and obliques abdominis interni, which are principally involved in the control of lateral trunk motions (Zedka et al., 1998). This feature is in accordance with a previous work of Bohannon et al. (1995) which had shown a multidirectional loss of trunk musculature force following stroke, even though this loss was more marked in the trunk flexion.

Thus, a force platform device should be viewed as an interesting tool for a better understanding of sitting posture impairment following stroke.

By using pertinent parameters, it allows us to understand specific components of postural control: the performance (sway area) and the activity (mean velocity) used to maintain sitting position. What is more, by dissociating the movements occurring along ML and AP axes, the deficiency intervening in the anterior and posterior trunk muscles can be dissociated from those involving the lateral trunk muscular groups. In order to evaluate objectively a patient or a rehabilitation protocol, some precise parameters aimed at characterising the sitting posture behaviour can be extracted. These parameters permits to better understand the origins of the postural trouble of patients with stroke and are largely more reproducible and objective than standard clinical evaluation scales. On the other hand, since it has been reported that 48% of the patients with stroke are not able to sit independently at the time of admission in a rehabilitation centre (Mayo et al., 1991), an important limit of this methodology is the time (40 s) the patients have to maintain their sitting posture alone. Consequently, this methodology would be inappropriate for evaluating the more severely impaired patients or at the earlier period following stroke. Recourse in parallel of both a clinical evaluation, for the more impaired patients, and biomechanical measures, for the patients able to sit independently, appears thus necessary.

5. Conclusion

This study has clearly assessed one postural impairments characterising patients with stroke when maintaining undisturbed sitting. Theses impairments include both velocity and magnitude of postural regulations. These facts have been interpreted by an enhancement of the amount activity associated by a reduction of the postural performance of patients with stroke during sit maintenance. In addition, some major impairments are noted on the sagittal plane, likely due to the major loss of muscular control intervening on the trunk flexor/extensor muscles. Since interesting and precise parameters can be given early (as soon as 20 days for our patients), a force platform system appears pertinent for evaluating the rehabilitation process following stroke. For instance, the short and long terms effects on such posture of rehabilitation techniques based on feedback techniques (mirror or CoP feedback from a computer) could be easily evaluated with this device.

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References


