UNIVERSITY OF TWENTE.

Faculty of Electrical Engineering, Mathematics & Computer Science

2D Random walk analysis of center of pressure trajectories in low back pain patients

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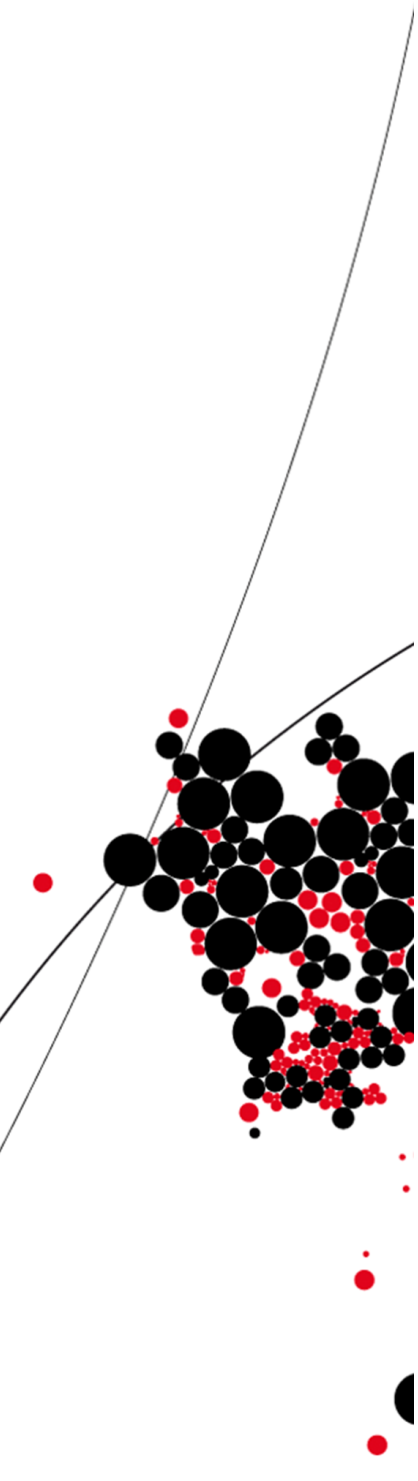

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Contents

Chapter 1

Background

Over a decade, several studies have focused on the neuromuscular control of trunk muscles and postural control in relation to this, to understand changing neural mechanisms in recurrent LBP patients [1],Ruhe2011,Silfies2009 . These attempts including several sophisticated methods such as detecting the distribution of lumbar erector spinae muscle activity using the high-density modality of EMG recording [2] detecting cortical activity to understand the contribution of feedforward control mechanism to deficit postural control [3] as well as using mechanical perturbation to investigate the change in sensory input as feedback information to control trunk muscles during standing in LBP patients [4]. Although these studies provided valuable insides about the variation in individual feedforward and feedback control mechanisms underlying deficit postural control in LBP patients, there is no study yet investigating how a proportional change of these mechanisms related to the severity of the LBP during quiet standing.

1.0.1 Postural control and Low back Pain

Postural control is an essential mechanism in our daily life to maintain balance and upright stance. Two mechanisms regulate postural control that is feedforward and feedback control. Feedforward control is used to anticipate expected postural perturbations involving the activation of trunk and leg muscles [5], Whereas; feedback control is a compensatory system using sensory feedback after perturbation such as gravity and external forces, which requires an integration of the three most critical sensory inputs: visual, vestibular and somatosensory. Findings suggest that multisensory integration of proprioception may be negatively affected by LBP [6]. Moreover, studies have shown that chronic pain produces reorganization in the primary somatosensory cortex.

Postural control is an essential mechanism in our daily life to maintain balance and upright stance. Two mechanisms regulate postural control that is feedforward

and feedback control. Feedforward control is used to anticipate expected postural perturbations involving the activation of trunk and leg muscles (Mohapatra et al., 2015), Whereas; feedback control is a compensatory system using sensory feedback after perturbation such as gravity and external forces, which requires an integration of the three most critical sensory inputs: visual, vestibular and somatosensory. Findings suggest that multisensory integration of proprioception may be negatively affected by LBP(Pedersen Sjo, 1997). Moreover, studies have shown that chronic pain produces reorganization in the primary somatosensory cortex. Additionally, LBP reduces trunk repositioning accuracy, seemingly due to a modified paraspinal afference in the muscle spindle and its processing(Brumagne et al., 2000). Thus, the postural control strategy adapts from hip to ankle strategy to reduce the reliance on the proprioceptive input from lumbar muscle spindles. Furthermore, increased coactivation in LBP subjects may suggest a compensatory joint stiffening strategy to cope with reduced proprioception(Dieen et al., 2003). Additionally, previous studies have shown abnormal motor control of the deep trunk muscles in individuals with chronic LBP(Caffaro et al., 2014). These abnormalities manifested with delays in neuromuscular recruitment and variations in the lumbosacral proprioception.

1.0.2 Postural sway and measuring its variability

The postural sway is the measurement that is commonly utilized to investigate changes in the postural control mechanisms. It indicates the center of mass (CoM) movement with respect to the ground support (Schiffman et al., 2006). The COP results from the body CoM on the ground support, resulting from the postural control outcome combination (Duarte Zatsiorsky, 2001). Specifically, force platforms are used to measure the center of pressure (COP). Summary statistics, i.e., traditional parameters, have been used to assess different pathologies, abnormalities, altered postural strategies and have been widely used to study LBP's effects. Based on the COP that is commonly used to measure balance performance in patients with LBP, the parameters are the mean velocity, the mean distance, root-mean-square (RMS), and the sway area(A. Ruhe et al., 2011). These parameters were described in detail by Prieto et al. (Prieto et al., 1996). By using the parameters mentioned above, some studies have shown that individuals with non-specific LBP have an increased postural sway (Mok et al., 2004), an essential diminution in sway velocity, and decreased stability in the sagittal plane due to the use of alternative strategies to compensate the affected proprioceptive system in the low back (Mok et al., 2004)and pelvic stabilization muscles (Norris, 1995). However, inconsistent results have been founded (Mazaheri et al., 2013), showing contradictory results across methods measuring

postural sway between LBP and healthy subjects. Therefore, traditional parameters are limited to the conclusion that body sway and body sway velocity might change for certain conditions, consequently not understanding the dynamic performance of the balance control and the underlying mechanisms involving feedforward and feedback control.

1.0.3 Stabilogram Diffusion Analysis

The Stabilogram diffusion analysis (SDA) method has been used to analyze the dynamic features of COP trajectories based on the assumption that COP can be modeled as a Fractional Brownian motion. Mandelbrot and van Ness introduced this phenomenon, an extension of the classical Brownian motion to have a more generalized Gaussian stochastic processes [7]. According to Collins and De Luca [7], the COP's displacements during quiet upright standing can be described as coupled and correlated random walks. Namely, the COP movements can be considered a combination of stochastic and deterministic mechanisms. It has been suggested that stochastic mechanisms are immersing in all levels of the neuromuscular system [8], [9]. Specifically, a study [10] showed that random errors are present in the motor unit recruitment and the neurotransmitters' release. These errors create stochastic variations in the muscle characteristics, namely force generation and timing [**?**]. Whereas fully deterministic processes are adjustments to the postural sway that are intentional and result in predictable and specific postural corrections. In terms of implementation, a straightforward method is used to calculate the stabilogram's scaling component, calculating the square of the displacements between all pairs of points separated in time by a specified time interval (fig.1).

Then, the square displacements are averaged to calculate the COP series.

$$\langle \Delta x^2 \rangle_{\Delta t} = \frac{1}{(N-l)} \sum_{i=1}^{N-l} (x_{i+l} - x_i)^2 \tag{1.1}$$

This relationship can be expressed using the mean square displacement, calculated from a one-dimensional random walk, which is correlated to the intervals of time expressed by the equation:

$$\langle \Delta x^2 \rangle = 2D\Delta t \tag{1.2}$$

Where D is the diffusion coefficient and can be seen as a measure of the average of a random walk's stochastic mechanism. For fractional Brownian motion, the equation can be generalized to the scaling law:

$$\langle \Delta x^2 \rangle \sim \Delta t^{2H} \tag{1.3}$$

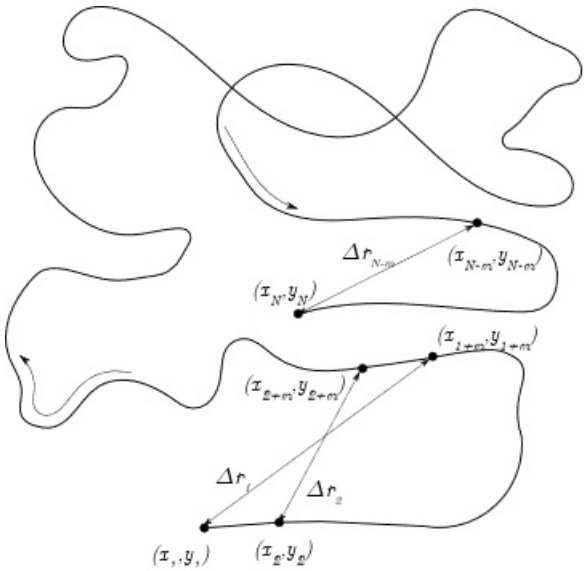


Figure 1.1: Diagram of the calculation of the mean square planar displacement $\langle \Delta x^2 \rangle$ as a function of time intervals Δt from a trajectory taken from COP with N data points.

Where H is the scaling exponent and will determine the nature of the stochastic process. This parameter is also known as Hurst exponent (Hurst, 1951) and express the correlation between past and future displacements in COP; It can be any real value in the range from 0 to 1. A Hurst exponent equal to 0.5 means an uncorrelated time-series (Classical Brownian motion)(fig.2). When $H > 0.5$, the COP shows a persistent behavior meaning that the COP moves away from a relative equilibrium point, indicating an open-loop control mechanism, in this context a higher H signifies a more remarkable persistence, having a deterministic boundary at $H = 1$ (fig.2). Whereas, when $H < 0.5$ the COP shows an anti-persistent behavior, meaning that the COP moves towards a relative equilibrium point indicating an anti-persistent behavior indicating a closed-loop control mechanism. In this context, lower H means a higher anti-persistence level with a deterministic boundary at $H = 0$ [11]. In the closed-loop mechanism, the visual, vestibular, and proprioceptive systems function as feedback in the postural control system. From the time of the important work from Collins and De Luca, following studies, specially Delignieres et al [12], suggested that the results of Collins and De Luca were not adapted to biological bound imposed by physiological limits, in this case, the support of the feet, and therefore having statistical artifacts. These artifacts may lead to underestimating diffusion processes in long-term intervals. Another study [9] suggested, that there are several different stochastic models that can explain COP motion and more parsimonious than the SDA method proposed by Collins and De Luca. Thus, proposing a random walk process involving a single continuous operation, instead of the two proposed by

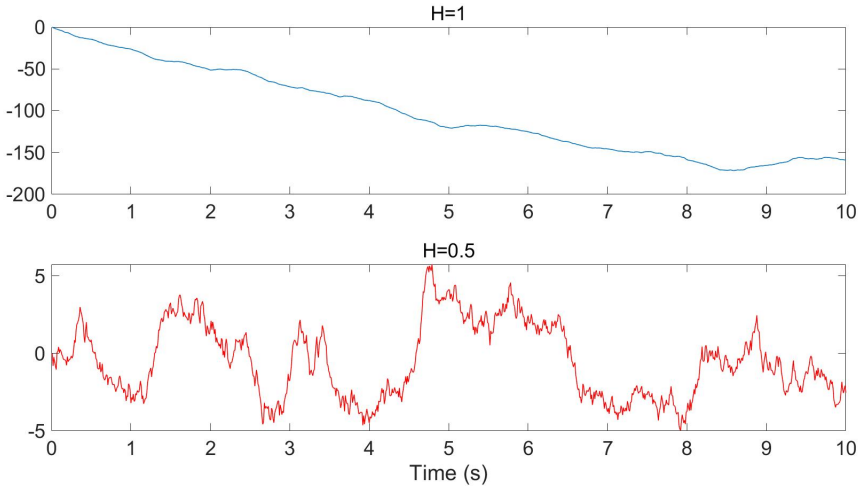


Figure 1.2: Hurst exponents for fully deterministic (H=1) and fully stochastic (H=0.5) systems.

Collins and de Luca.

Chapter 2

Introduction

Low Back Pain (LBP) has been considered one of the most frequent health issues in society, leading to significant disability, health service usage like hospitalization, physician visits, and work absenteeism [13]. Previous studies show that this condition will affect 50-80% of the general population at some point in their lives [14]. However, still, more than 75% of the patients will get a non-specific LBP diagnosis [15], [16]. By the virtue of the non-specific treatment, the majority of LBP patients have persistent or recurrent pain episodes within 12 months [13]. For this reason, it is of utmost importance to determine the underlying neural changes for specific treatment.

Postural control is an essential mechanism in our daily life to maintain balance and upright stance. Studies have been used the center of pressure (COP) summary statistics, i.e, traditional parameters to assess different pathologies, abnormalities, altered postural strategies, and also have been widely used to study the effects of LBP. However, inconsistent results have been founded [17], showing contradictory results across methods measuring postural sway between LBP and healthy subjects. Therefore, traditional parameters are limited to the conclusion that body sway and body sway velocity might change for certain conditions. Thus, not allowing the understanding of the dynamic performance of balance control and the underlying mechanisms, involving feedforward and feedback control. However, the Stabilogram diffusion analysis (SDA) method can be used to analyze these mechanisms. The SDA method has been adopted by several research groups who have shown that SDA parameters are sensitive to the effects of age [**?**], vision [18], [19], Parkinson's Disease [20], adults with stroke [21], reduced plantar cutaneous sensation [22], and to older adults who retrospectively reported falls [22].

This study aims to gain a better insight into subjects with chronic LBP and its effects on postural control mechanism and sway behavior using statistical mechanical tools like SDA in upright standing for adults between 19 and 65 years old. It is to be considered that SDA parameters may eventually provide useful biomarkers for LBP, with

the possibility to be used as indicators of the chronicity of the pain, the efficacy of treatments, and to monitor progress.

Chapter 3

Body of the thesis

3.1 Methods

3.1.1 Patients and study protocol

The study was approved by the Ethical Committee of the University Hospital of Göttingen (No 13/12/15) Germany, and was conducted according to the recommendations of the Declaration of Helsinki. A total of 19 severe chronic nonspecific LBP patients and 23 healthy control participants, 48.512.1 and 34.513.1 years old, respectively, including relevant functional limitations due to chronic pain, were recruited in the Pain Clinic of the University Hospital Göttingen, Germany. The COP and ground reaction force were collected using Kistler Multicomponent Force Plate for Biomechanics Type 9281E, sampled at a frequency of 2048 Hz for each foot and low-pass filtered at 5Hz using a fourth-order Butterworth digital filter to eliminate noise. Subjects stood on a force platform with their feet separated approximately 10cm in the mediolateral direction and were instructed to stand still for 60 seconds with their eyes open and immediately 60 seconds with their eyes closed (fig.1). The first 10 seconds were removed from every data set due to accommodation of the subject in the platform.

The COP data were measured independently for each foot and was required to merge the two measurements in one unified COP [23] using the vertical reaction forces using standard biomechanics analyses.

$$WhereCOP_{net}(t) = COP_l(t)\frac{R_{vl}(t)}{R_{vl}(t)+R_{vr}(t)} + COP_r(t)\frac{R_{vr}(t)}{R_{vl}(t)+R_{vr}(t)} \tag{3.1}$$

Where $COP_{net}(t)$ is the center of pressure for a single force platform, $COP_l(t)$ and $COP_r(t)$ and the center of pressure of left and right foot respectively. $R_{vl}(t)$ and $R_{vr}(t)$ are the forces in Z direction (reaction forces) under the left and right respectively.

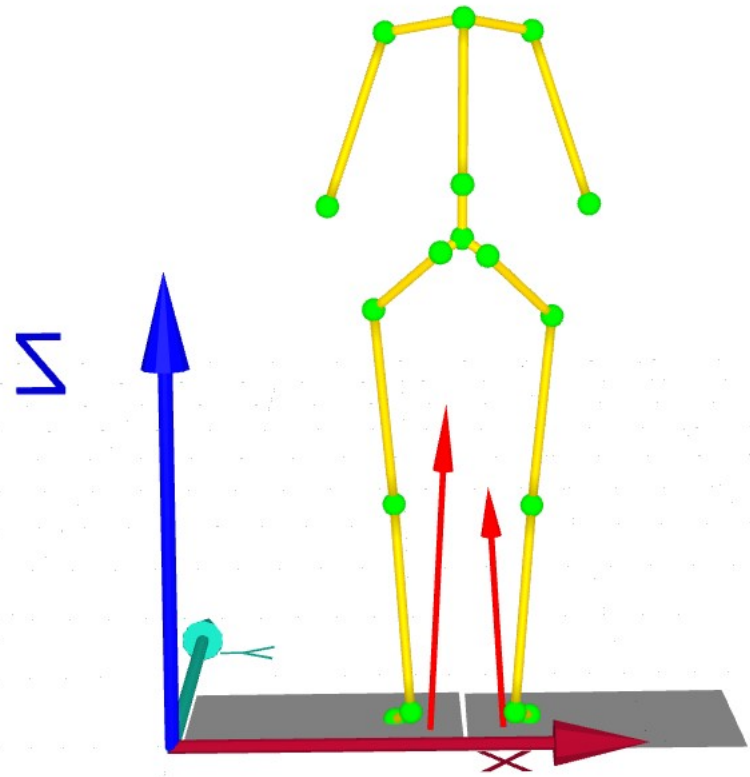


Figure 3.1: Force platform configuration, representing the individual platforms for right and left foot and the reference system.

Measurements of pressure pain sensitivity before and after the task and an assessment of pain intensity, functional and psychological characteristics were also obtained each time. Pressure pain sensitivity and patients functional assessments and reports. Reports of the subjects' pain and disability were also obtained.

3.1.2 In- and exclusions

Patients with radicular low back pain or back pain related to trauma, fractures, and spinal stenoses were not included. Further exclusion criteria were opioid consumption of $>$ 30 mg of morphine equivalents per day, intake of anticonvulsives and antidepressant medication and participation in other studies conducted at the same time. Age- and gender-matched healthy individuals were recruited via local advertisement to act as a control group. Pain-free participants were included if they had no relevant history over the last three years of back or lower limb pain or injury that limited their function and/or required treatment from a health professional. Patients and control subjects had to have the capacity to give consent at his/her own will. Concurrent systemic, rheumatic or neuro-musculoskeletal disorders that may confound testing or current pregnancy were general exclusion criteria.

3.1.3 Stabilogram Diffusion Analysis

As Collins and De Luca [18] proposed, the movement of the center of pressure in upright quite standing in humans can be seen as a correlated, coupled random walk. The trajectories of the center of pressure were studied in 1D in Medio Lateral and Anteroposterior directions. The squared displacements were computed between all paired points separated by a fixed interval of time. An increasing number of time intervals is used to obtain the whole time series, which is then averaged over all the time intervals.

$$\langle \Delta x^2 \rangle_{\Delta t} = \frac{1}{(N-l)} \sum_{i=1}^{N-l} (x_{i+l} - x_i)^2 \tag{3.2}$$

Where m is the time interval between two values indicating the number of data. N is the total number of points in the COP data set. Two regions (Short term and Long term) are identified, using to parametrize the stabilogram diffusion plot and are divided by a critical point that is used to identify the transition point that differentiates the linear relation in both regions. The critical point is obtained by calculating the local minimum (between 0 and 2.5s) of the second derivative of the stabilogram diffusion plot to determine the time in which the change in the slope is maximum and be able to split the stabilogram diffusion plot into two regions.

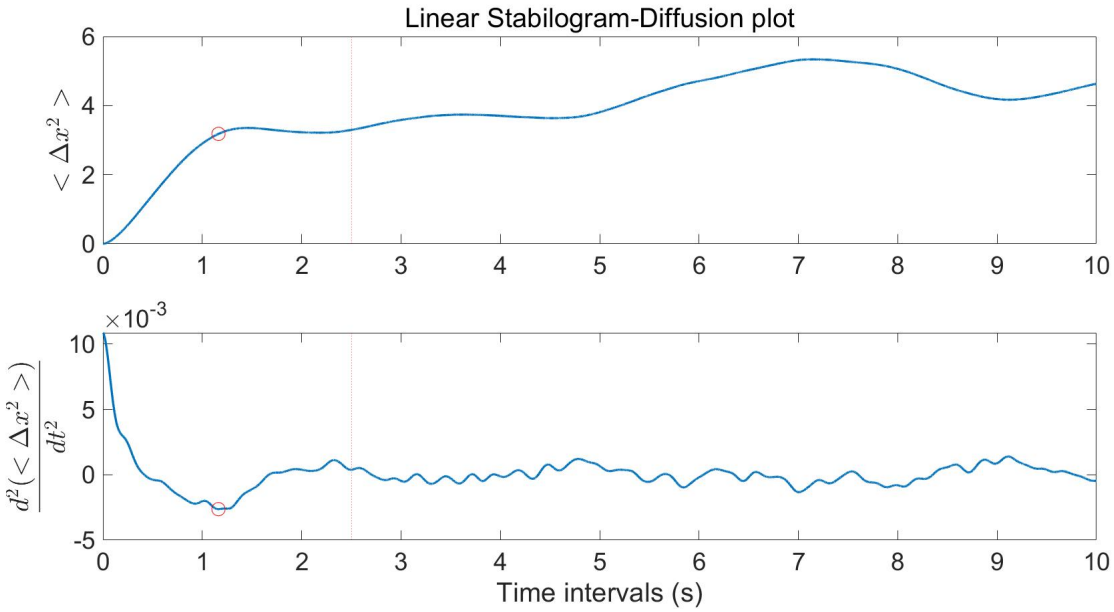


Figure 3.2: Calculation of the critical point using the local minimum (0 to 2.5s) of the second derivative (bottom) of the mean squared displacement (top).

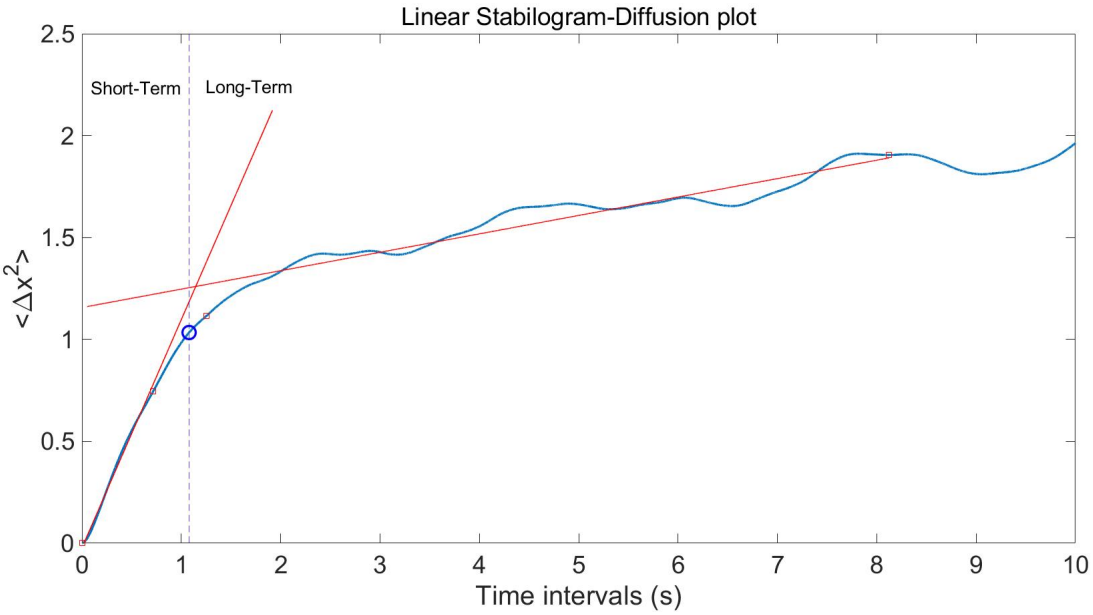


Figure 3.3: Stabilogram diffusion function showing the mean square displacement vs the time intervals, the slopes calculated using least squares are evident. The critical point is evidenced in division of the two regions.

The diffusion coefficients are calculated from the slopes of the stabilogram diffusion plot using the least squares method to find the best fit possible for the two regions. In the same way the Hurst scaling components in the short and long term regions are calculated from the slope in the log scale of the stabilogram diffusion plot. To accurately calculate the slopes in the stabilogram diffusion for the diffusion coefficient and the Hurst component for the log scale four times are obtained from the second derivative of the $\langle \Delta x^2 \rangle_{\Delta t}$ T1 is always the initial time, T2 is the maximum before the first second. fig2.2 Calculation of the critical point using the local minimum (0 to 2.5s) the second derivative of the mean squared displacement. Moreover, T3 is the second local maximum and it is measured from the critical point onwards using windows in order to account for noisy derivatives and slight fluctuations and T4 is calculated using firstly the first minimum going from 9s backward, after this minimum is obtained, the first maximum to the left is T4. If there is no maximum before 7 s, 9s is taken as T4. Consequently, the short-term components are measured between T1 and T2 and the long term between T3 and T4.

3.1.4 Pressure pain thresholds

Pressure pain thresholds (PPT) were measured with an electronic algometer (fig.2.4, Somedic Production, Stockholm, Sweden) over 8 locations distributed across the lumbar region, on the side of greatest pain for the people with LBP, and on the right side for the control group. The distance between the locations was 2.5 cm each starting from L5 (detected via palpation) in the cranial direction, and 2.5 cm in the lateral direction starting from the spine. The algometer probe tip (1 cm2) was applied to the skin at a rate of 30 kPa/second and the participant was instructed to depress a handheld switch at their first perception of pain, at which point the application of pressure ceased (0 to 1000). An explanation of the PPT measurement procedure, followed by a demonstration on the patient's forearm or thigh, was performed prior to 2 consecutive PPT measures at each location in a randomized order. The mean of the 2 PPT measures at each location was used for further analysis. The same researcher performed the PPT measurements in all subjects before and after the repetitive lifting task.

3.1.5 Pain Scale

Before performing the task, participants were asked to rate their pain intensity on an 11-point numerical rating scale (NRS, 0 = no pain, 10 = worst pain).

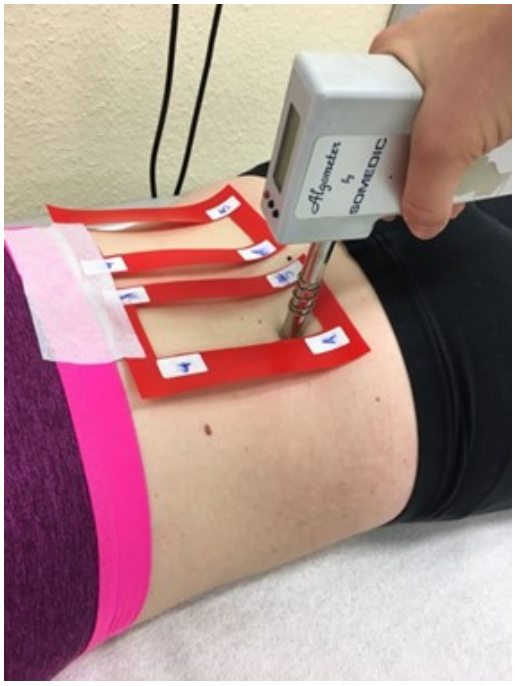


Figure 3.4: Electronic algometer Somedic Production to measure PPT over 8 locations distributed across the lumbar region.

3.1.6 Statistical analysis

For all the statistical analyses IBM SPSS software was used. Shapiro-Wilk test was applied to all the dependent variables for the assumption of normality, accordingly a mixed two-way ANOVA was performed for the parameters that satisfied the assumptions, the independent variables were group (LBP vs. Control) with a condition of eyes closed and eyes open. For the parameters that did not satisfied the assumptions, namely these parameters did not show any evidence to be normally distributed, a non-parametric test (Mann-Whitney U) was used and likewise Wilcoxon W used to measure the effect of the eye condition. However, some parameters did not fulfilled the symmetry condition, so the Sing test was performed. The dependent variables were Long-Term Scaling Exponent (Hl) Short-Term Scaling Exponent (Hs), Short-Term 'Effective' Diffusion. Coefficient (Ds), Long-Term 'Effective' Diffusion. Coefficient(Dl), critical time (CT) and critical displacement (CD) for anteroposterior, mediolateral and planar directions A Pearson Correlation was used to quantify the correlation between PPT(Pressure Pain Threshold), the pain scale and the normally distributed parameters Kendall's Tau b was used was used to quantify the correlation between PPT, scale pain and the non-normally distributed parameters. Traditional parameters were analyzed using Mann Whitney U test for differences in median between LBP subjects and Healthy controls. For the eye condition, Wilcoxon W test was used.

Characteristic, mean ± SD	Healthy	LBP
Age (years)	34.6±2.4	47.9±2.6
Height (cm)	175.4±1.6	171.3±1.7
Weight (Kg)	71.7±3.1	79.4±3.3

Table 3.1: Subject anthropomorphic characteristics. Values are the Mean ± SEM

3.2 Results

The SDA parameters that were normally distributed (Table A1), namely Hs_{AP} was significantly larger for LBP subjects compared with healthy subjects (p=0.037). In contrast, Hl_{AP} and Hl_R were significantly lower in LBP subjects compared with the healthy subjects (p=0.043 and p=0.041, respectively).(Table A2). The non-normally distributed parameters, Dl_{MD}, Dl_R, $Dl_{AP}(EC)$ (p=0.003,p=0.001,p=0.042)were significantly lower for LBP subjects. (Table 2). The parameters Ds, CT and CD did not show any significantly difference between groups.

Pearson's r correlation between PPT and the normally distributed parameters extracted from SDA was used. Showing that the short-term and long-term scaling exponent in mediolateral was correlated with PPT. Where the short-term scaling exponent in ML showed a negative correlation, H_sML (r=-0.376, p=0.024) and the long-term scaling in ML exponent showed a positive correlation, H_l ML (r=0.360, p=0.031)(Table A6). Kendall's Tau correlation for ranks between PPT and the non-normally distributed parameters extracted from SDA was used. Showing that the long-term effective diffusion coefficient for mediolateral DIML (tau=0.313, p=0.007) and PPT were found to be positively correlated. In addition, the critical displacement in the anteroposterior direction within the condition of eyes closed was positive correlated with the PPT (tau=0.256, p=0.028) (Table A7) Traditional measurements did not show any difference between LBP subjects and healthy controls measured by Mann Whitney U on ranks due to the lack of evidence of being normally distributed (Table A4). The interaction between the eyes and LBP condition did not show any statistically significant results for the normally distributed parameters (Table A5). The parameters CDAP and DsAP were significantly higher for LBP subjects and healthy subjects in closed eyes condition. Moreover, DIML was significantly higher for LBP subjects in eyes-closed condition. (Table A3.) Path area, Path RMS, Mean displacement and Path velocity presented significantly higher the medians using Wilcoxon W test for ranks in LBP subjects (Table A8).

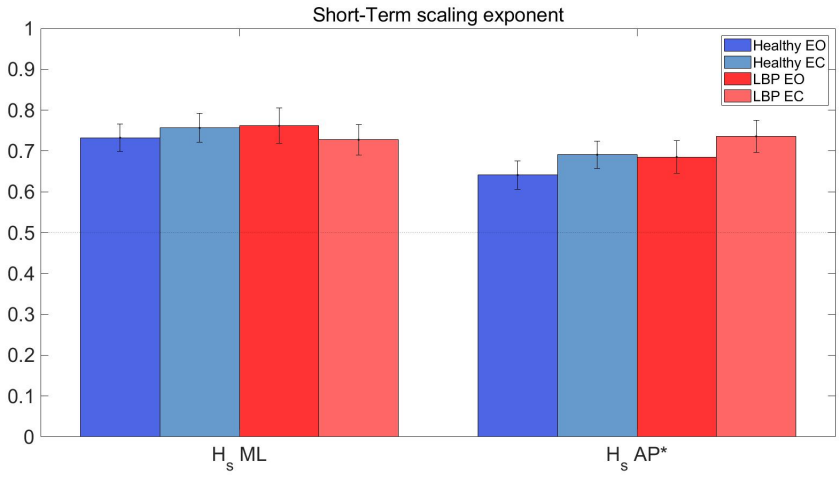


Figure 3.5: Group means and confidence interval for the Short-term scaling exponent under the condition of eyes-open (EO) and eyes-closed (EC). The symbol * denotes $P<0.05$.

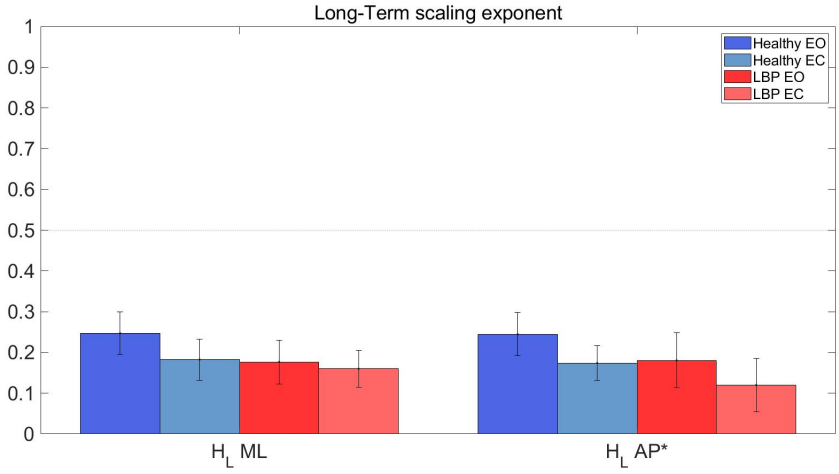


Figure 3.6: Group means and standard deviation for the Long-term scaling exponent under the condition of eyes-open (EO) and eyes-closed (EC). The symbol * denotes $P<0.05$.

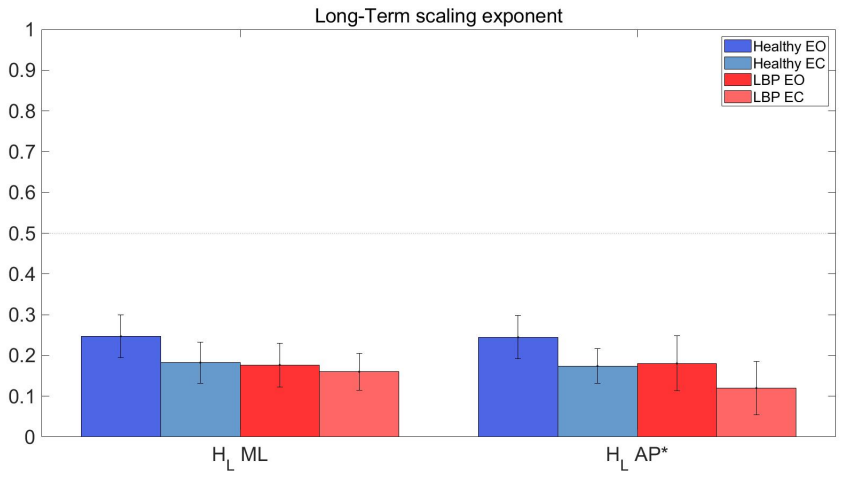


Figure 3.7: Group means and standard deviation for the Long-term effective diffusion coefficient under the condition of eyes-open (EO) and eyes-closed (EC). The symbol * denotes $P<0.05$.

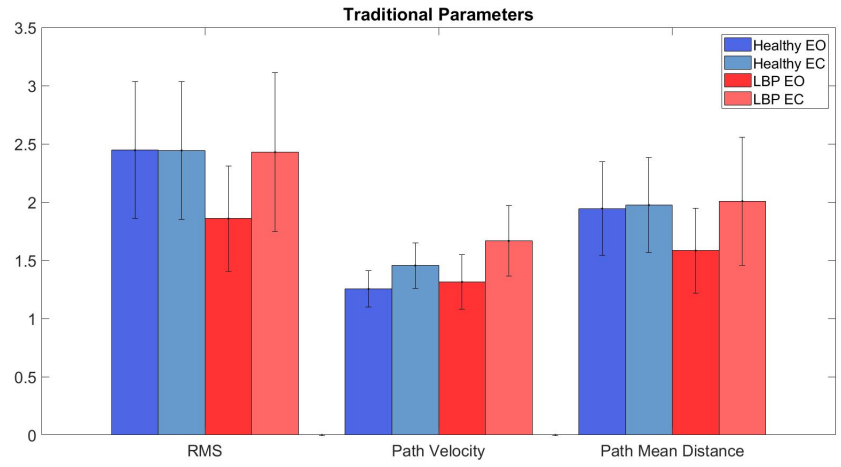
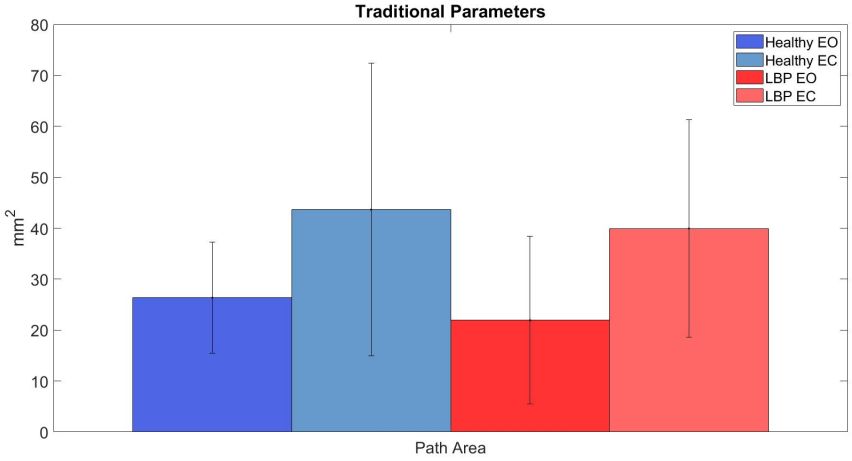


Figure 3.8: Group means and confidence interval of the traditional parameters (RMS, Path Velocity and Path Mean Distance)



Area.jpg

Figure 3.9: Group means and confidence interval of the traditional parameters Path Area.

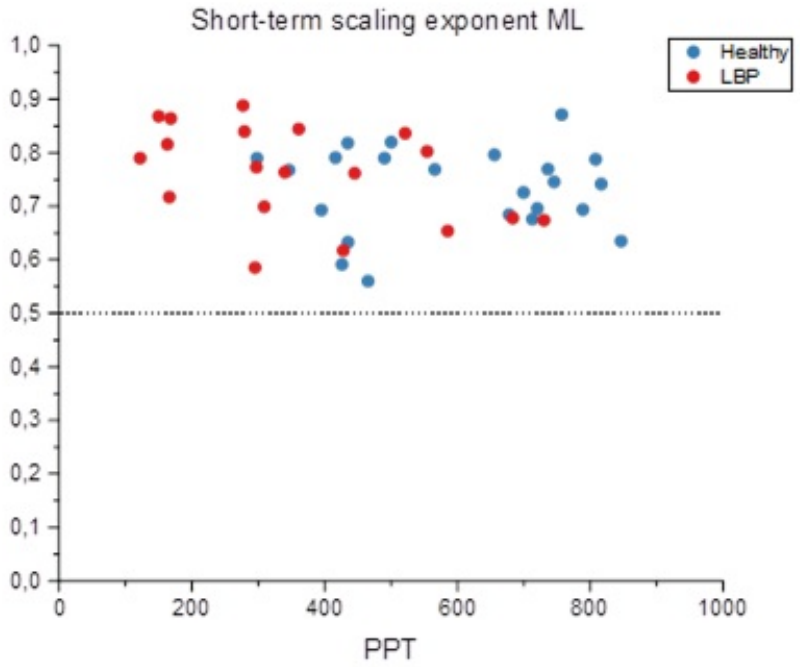


Figure 3.10: Scatter plot representing Pressure Pain Threshold vs. Short-term scaling exponent in the mediolateral direction. Pearson's R=-0.376, p=0.024.

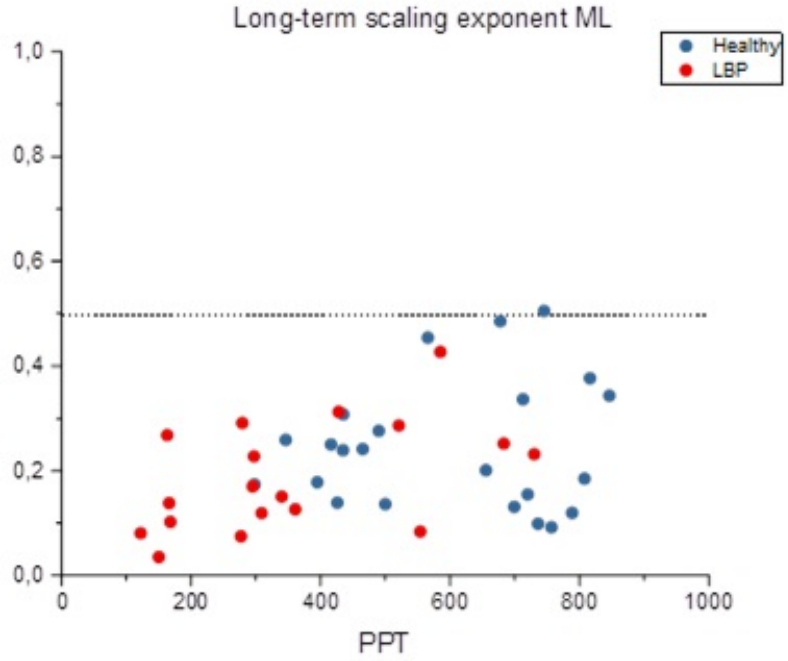


Figure 3.11: Scatter plot representing Pressure Pain Threshold vs. Long-term scaling exponent in the mediolateral direction. Pearson's R=.323, p=0.010.

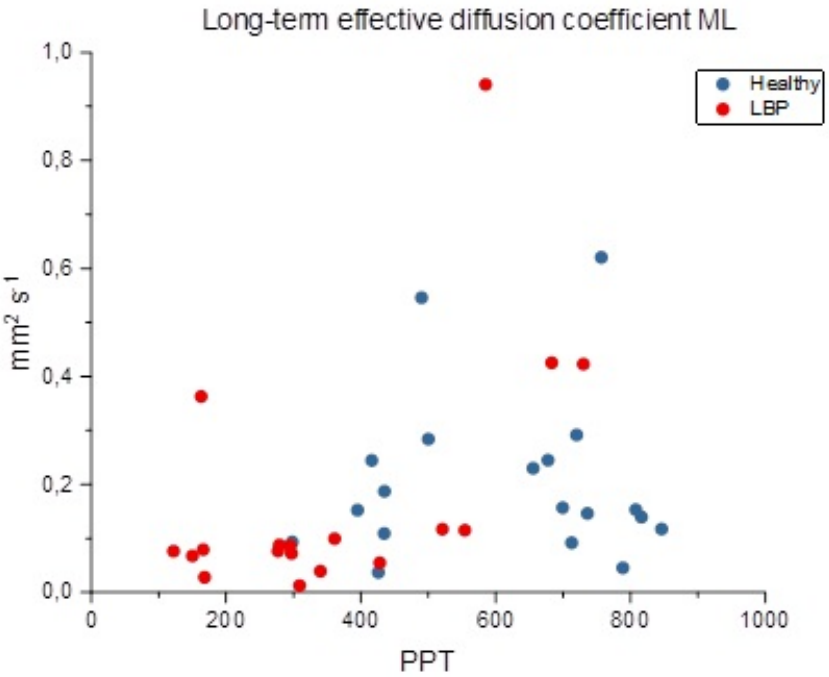


Figure 3.12: Scatter plot representing Pressure Pain Threshold vs. Long-term effective diffusion coefficient in the mediolateral direction. Kendall's tau=0.313, p=0.007.

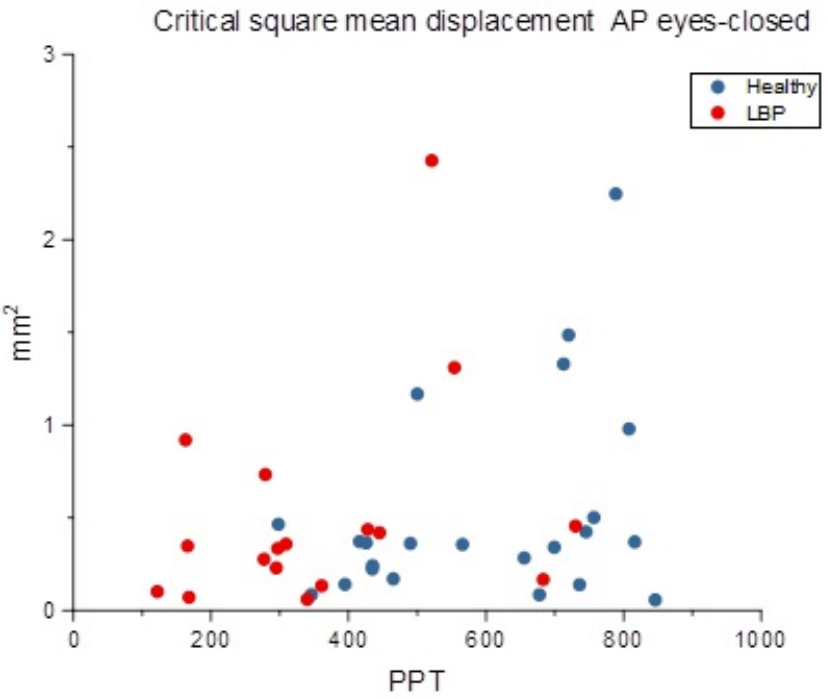


Figure 3.13: Scatter plot representing Pressure Pain Threshold vs. Critical square mean displacement in the anterioposterior direction with eyes closed. Kendalls's Tau=0.256, p=0.028.

3.3 Discussion

3.3.1 Difference between patients and control

The results indicate that three parameters from the SDA showed significant differences between healthy and LBP subjects. The short-term Hurst scalar exponent (HS) in the AP direction presented higher values in LBP subjects, indicating that the open-loop postural control mechanism in the LBP subjects is more positively correlated in and thus more unstable [18], it means that there is a greater tendency in the whole system to drift away from an equilibrium point in the short term intervals. Collins and De Luca [18] attributed this behaviour to a specific control strategy adopted in their case by elderly individuals increasing the net stiffness in their musculoskeletal system using a higher level of muscular activity over their lower limb's joints. Stiffer systems have some advantages, like being better at making corrections and resisting temporary disturbances. Previous studies showed that non-specific LBP patients use a trunk stiffening strategy reached by using the trunk muscles' co-activation, assisted by an improved distal response, kept during the short-term phase of the postural response to be able to counteract unpredictable surface disturbances [24]. Furthermore, [25], [26]proposed that during standing, the capacity of

maintaining stability could be affected by the above-mentioned muscle co-activation. In contrast, the long-term Hurst scalar exponent (Hl) in the mediolateral direction is lower in LBP subjects than healthy subjects. Showing that in the steady-state of the closed-loop mechanism, LBP subjects have a more negatively correlated, consequently, in the long-term intervals, there is a higher corrective adjustment in the equilibrium direction, namely an increase in gain of feedback mechanisms, which allow LBP subjects to compensate for the short-term unstable behaviour. This increased gain might be due to a reweighting of the sensory inputs, a consequence of the impaired proprioceptive systems, specifically reduction in the lumbosacral proprioception in LBP subjects [27]. Therefore, due to the defective information about the position, LBP subjects might stiffen the trunk to decrease degrees of freedom. The long-term diffusion coefficients were lower for LBP subjects in the mediolateral direction when their eyes were open and in the anteroposterior direction when their eyes were closed. This decrease might be due to the higher gain in the feedback mechanisms represented by a higher anti-persistent behaviour, that for a specified level of effective stochastic activity, there would be a decrease in the probability of more significant displacements of the COP away from a relative equilibrium point [18] On the contrary, both directions' short-term diffusion coefficient was the same for LBP subjects and healthy subjects. Besides, the critical time and the critical displacement did not change for LBP subjects, expressing any modification in the time interval when the subjects started using the feedback control (a closed-loop mechanism)

3.3.2 The effect of vision

As expected, vision played a significant role in the motor control mechanism. Previous studies showed that SDA parameters are used to analyse how vision input change affects the open-loop and closed-loop postural control mechanisms [18]. The HS in the AP direction increased when the vision is restricted in both healthy and LBP subjects, showing that the visual input could reduce the stiffness, increasing the levels of muscular activity on the lower limbs [18], [19] and therefore significantly modifying the persistent behaviour of the open-loop mechanism. Furthermore, the HL in the AP direction decreased when the vision was restricted in healthy and LBP subjects. The result suggests vision input significantly modifies the closed-loop mechanism's characteristics that cause increased uncorrelated behaviour. This decrease may be due to reducing the stiffness of the musculoskeletal system caused a more visual strategy. Therefore, reducing the feedback gain making the system less strongly correlated [18]. However, there was no interaction between (HS, HL), and LBP. The two groups presented higher short-term diffusion coefficient values in both directions when their eyes were closed. It supports the results of the first group

on Collins and De Luca [18] in which is suggested that this group decreased their musculoskeletal system's stiffness through the lowering of the muscular activity over their lower limbs, especially the joints resulting in a more unstable short-term mechanism when there was no visual input. Moreover, critical displacement was reported to increase with eyes closed due to the privation of vision, which is directly affected by the higher short-term diffusion coefficient values' increment and no significant critical time changes. As Collins and De Luca proposed, change in mechanism from open to closed-loop is lead by displacement and not by time [18]. Interestingly, two parameters were sensitive to for the changes in visual input in the LBP subjects, CD and DL in the ML direction, showing higher values and therefore less stability, suggesting that LBP subjects have a higher reliance on the visual input to control balance in the ML direction than healthy subjects.

3.3.3 Correlation between pain and SDA parameters

PPT is an efficient and reliable measurement in the investigation of physio-pathological mechanisms involving syndromes in muscle pain like LBP [28], yet its subjective character cannot be avoided. PPT was assessed in correlation with SDA and traditional parameters. The parameter HS showed a moderate negative correlation with the PPT in the ML direction when the subjects had their eyes open, indicating that subjects with more severe LBP (lower PPT) had a higher short term scaling exponent and therefore a higher tendency of the system to drift away in the short-term. This correlation can be associated with the fact that pain might provoke an increment in presynaptic inhibition of trunk muscle afferents, in the same way altering the muscles' central modulation of the proprioceptive system, which may cause a decreased muscle control and consequently affecting and therefore, increasing the postural sway [29]. The parameter HL showed a moderate positive correlation with the PPT in the ML direction when the subjects had their eyes open, indicating that subjects with more severe LBP had a lower HL. Therefore a more negatively correlated postural control in the closed-loop behaviour, accordingly, a higher probability that larger displacements will be adjusted back into the relative equilibrium position [18]. This might be due to LBP subjects experiencing fear of injury or movements that can induce pain that might affect the postural control strategy. For instance, elderly subjects also showed a lower long-term Hurst component in the ML direction [18], which has been shown that fear in elderly subjects could increase trunk muscle activity and axial stiffness. DL in ML was positively correlated with PPT; thus, DL was lower for subjects with lower pain thresholds. As mentioned before, this might be due to higher anti-persistent behaviour. For a specified level of effective stochastic activity, there would be a decrease in the probability of more

significant displacements of the COP away from a relative equilibrium point [18]. Another parameter that showed a positive correlation with the PPT was the critical displacement in the anteroposterior when the subjects had their eyes closed. This result suggests that with occlusion of vision, subjects with more severe LBP had an anticipated activation of the closed-loop, in contradiction with what was expected, because it has been shown that subjects with LBP have a delayed muscle response that can contribute to impairing postural control [30]. Lastly, traditional parameters were not correlated with PPT.

3.3.4 Traditional parameters

The traditional parameters, RMS, path mean velocity, path mean distance, and path area, only presented statistically significant differences for changes in vision, showing higher values when the vision was occluded for LBP subjects. However, the traditional parameters were not sensitive enough to LBP, showing that, in general, SDA is a more sensitive method. As expressed before, traditional parameters do not reflect the dynamic features of the COP; therefore, this method is less convenient to understand the mechanisms to control posture that the central nervous system uses. On the contrary, SDA parameters can give insights about the short-term and long-term behaviour of the postural control, in which motor control theory can explain these behaviours [31]. Pain scale The pain scale did not show any correlation with the traditional nor the SDA parameters. This result contradicts Ruhe et al. results [29]that, despite the subjective nature of pain perception, could express a linear relationship between NRS scores and traditional postural sway parameters like postural sway velocity and sway area.

3.3.5 Limitations

One of the limitations is the number of trials per subject. It is suggested at least five trials per subject and trial lengths of 60s to produce reliable measurements for critical point coordinates, diffusion coefficients, and scaling components [32]. Another limitation is that there was no data recorded on how long the subjects have suffered the LBP.

Chapter 4

Conclusion

4.1 Conclusions

This research aimed providing a quantitative method to distinguishing the individual contribution of feedforward and feedback mechanisms to postural control in subjects with non-specific chronic LBP and to understand how they change in painful situation.. Overall, the results suggested that the outcomes of SDA analysis may provide indexes to identify changes in control strategy in correlation with the severity of the pain. The results indicated that the ratio between short- and long-term relations in COP signal is different in LBP patients with respect to healthy subjects. This result may be interpreted as a change in feedback and feedforward control strategies due to the effect of pain. For the short-term intervals, SDA exposed a more unstable behaviour in LBP subjects, resulting in greater displacements before any feedback can come into action. However, LBP subjects were able to adapt and correct these larger sway distances. Moreover, some specific SDA parameters were moderately correlated with pain, whereas traditional parameters were not. In addition, SDA parameters were more congruent and sensitive than traditional parameters and were able to identify the postural mechanisms affected by LBP in contrast with traditional parameters that were only significant when there was occlusion of vision. Equally important, SDA parameters were moderately correlated with PPT, that it has been found efficient and reliable, yet its subjective character cannot be avoided. In order to be able future use of SDA parameters as indicators of chronicity, the efficacy of treatments or even prevention, validation of the method is needed, assessing different characteristics of the pain, such as duration, site, frequency, severity, disability and psychosocial flags. The research's main objective is to better insight into subjects with chronic LBP and its effects on postural control mechanism and sway behavior using statistical mechanical tools like SDA in upright standing for adults between 19 and 65 years old. It is to be considered that SDA parameters may eventually provide useful biomarkers for LBP, with the possibility to be used as indicators of the

chronicity of the pain, the efficacy of treatments, and to monitor progress.

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Appendix A

Appendix

	Healthy	LBP	Healthy	LBP
Shapiro-Wilk test	Eyes Open		Eyes Closed	
	Sig.			
CTMD	0,089*	0,024	0,004	0,403*
CTAP	0,254*	0,104*	0,003	0,027
HsMD	0,425*	0,364*	0,640*	0,418*
HsAP	0,629*	0,234*	0,127*	0,214*
HlMD	0,067*	0,831*	0,524*	0,442*
HlAP	0,844*	0,564*	0,090*	0,337*
CDMD	0,000	0,000	0,000	0,000
CDAP	0,015	0,000	0,000	0,000
DsMD	0,000	0,000	0,000	0,000
DsAP	0,000	0,000	0,000	0,001
DlMD	0,000	0,000	0,000	0,000
DlAP	0,000	0,731*	0,035	0,003

Table A.1: Shapiro-Wilk test for all the parameters for LBP and Healthy subjects in Eyes-Open and Eyes-Closed conditions.

	Healthy	LBP	Healthy	LBP	ANOVA-Group	
	Eyes Open		Eyes Closed		F	Sig.
CTMD	0,93±0,35	1,00±0,48	0,89±0,41	1,19±0,45	3,782	0,059
CTAP	1,03±0,40	1,14±0,51	1,02±0,48	0,97±0,45	0,051	0,822
CTR	0,93±0,36	0,93±0,45	0,92±0,41	1,15±0,53	1,30	0,261
HsMD	0,73±0,08	0,76±0,09	0,76±0,08	0,73±0,08	0	0,993
HsAP	0,64±0,08	0,69±0,08	0,69±0,08	0,74±0,08	4,636	**0,037***
HsR	0,72±0,08	0,75±0,08	0,75±0,08	0,73±0,07	0,240	0,627
HlMD	0,25±0,12	0,18±0,11	0,18±0,12	0,16±0,09	2,749	0,105
HlAP	0,24±0,12	0,18±0,14	0,17±0,10	0,12±0,14	4,264	**0,045***
HlR	0,26±0,11	0,18±0,11	0,18±0,11	0,15±0,09	4,483	**0,041***

Table A.2: Stabilogram Diffusion Analysis for People with Low Back Pain (LBP) and Healthy Subjects in Eyes-Closed and Eyes-Open Conditions for normally distributed parameters

	Healthy	LBP	Sig.	Healthy	LBP	Sig.
	Eyes Open			Eyes Closed		
CDMD	4,10±5,80	3,86±6,49	0,850	6,69±16,08	8,21±14,24	0,553
CDAP	0,25±0,19	0,50±0,89	0,658	0,53±0,55	1,01±1,59	0,771
CDR	4,36±5,92	4,27±7,34	0,909	7,18±16,30	9,34±15,95	0,570
DsMD	2,09±2,44	2,09±3,34	0,771	4,81±12,09	3,05±3,55	0,850
DsAP	0,15±0,15	0,24±0,40	0,306	0,31±0,32	0,43±0,48	0,587
DsR	2,25±2,50	2,34±3,78	0,830	5,10±12,31	3,50±3,92	0,622
DlMD	0,49±0,69	0,16±0,24	**0,003***	0,34±0,48	0,32±0,37	0,850
DlAP	0,04±0,06	0,02±0,02	0,519	0,040±0,03	0,02±0,06	**0,042***
DlR	0,52±0,67	0,17±0,24	**0,001***	0,38±0,49	0,35±0,40	0,950

Table A.3: Stabilogram Diffusion Analysis for people with Low Back Pain (LBP) and Healthy Subjects in Eyes-Closed and Eyes-Open Conditions for non-normally distributed parameters using Mann-Whitney U test for ranks.

	Healthy	LBP	Mann-Whitney U Test	Healthy	LBP	Mann-Whitney U Test
	Eyes Open		Sig.	Eyes Closed		Sig.
RMS (mm)	2.45±0.28	1.86±0.21	0.132	2.44± 0.28	2.43± 0.33	0.969
Velocity (mms^{-1})	1.25±0.07	1.31±0.11	0.790	1.45± 0.94	1.66± 0.14	0.909
Mean Distance (mm)	1.94± 0.19	1.58±0.17	0.201	1.97± 0.20	2.01± 0.26	0.306
Area (mm^2)	26.36± 5.27	21.94± 7.83	0.153	43.65± 13.85	39.94±10.17	0.989

Table A.4: Traditional Postural Sway parameters for people with LBP and Healthy subjects for the condition of eyes-open and eyes closed for non-normally distributed parameters using Mann-Whitney U test for ranks.

	F	Sig.
HsMD	3,523	0,068
HsAP	0,001	0,982
HlMD	1,336	0,255
HlAP	0,042	0,839

Table A.5: Within subjects for the interaction between the condition of LBP and eyes open and eyes closed.

	Eyes Open		Eyes Closed	
	Pearson Correlation	Sig. (2-tailed)	Pearson Correlation	Sig. (2-tailed)
HsMD	-.376*	0,024	-0,017	0,922
HlMD	.360*	0,031	-0,164	0,341
HsAP	-0,077	0,654	0,042	0,806
HlAP	0,244	0,151	-0,098	0,568
HsR	-.425**	0,010	0,018	0,917
HlR	.384*	0,021	-0,203	0,236

Table A.6: Correlation between pain and normally distributed parameters using Pearson correlation.

	Eyes Open		Eyes Closed	
	Correlation Coeff.	Sig. (2-tailed)	Correlation Coeff.	Sig. (2-tailed)
CTMD	-0,078	0,511	-0,016	0,892
CTAP	0,214	0,066	0,214	0,066
CDMD	0,005	0,967	0,094	0,422
CDAP	0,151	0,196	.256*	0,028
DsMD	0,052	0,653	0,030	0,796
DsAP	0,141	0,225	0,151	0,196
DlMD	.313**	0,007	-0,052	0,653
DlAP	0,167	0,153	0,138	0,236

Table A.7: Correlation between pain and non-normally distributed parameters using Kendall's Tau correlation.

	Healthy		LBP	
	Z	Asymp. Sig. (2-tailed)	Z	Asymp. Sig. (2-tailed)
CTML	-.156b	0,876	-1.207b	0,227
CTAP	-.365b	0,715	-1.046c	0,295
CDML	-.852c	0,394	-3.099b	0,002*
CDAP	-3.041c	0,002*	-2.334b	0,020*
DsML	-2.159c	0,031*	-1.173b	0,030*
DsAP	-3.619c	0,000*	-2.817b	0,005*
DIML	-.547b	0,584	-2.857b	0,004*
DIAP	-.487c	0,627	-.563c	0,573
Path RMS	-.335c	0,738	-2.495b	0,013*
Path Velocity	-2.950c	0,003*	-3.058b	0,002*
Path Mean Distance	-.487c	0,627	-2.334b	0,020*
Path Area	-1.794c	0,073	-2.978b	0,003*

Table A.8: Wilcoxon W test for ranks for the condition of eyes-open and eyes-closed for non-normally distributed parameters.

	Z	Asymp. Sig. (2-tailed)
CDMD	-3,549	0,000*
DsMD	-3,549	0,000*
DIMD	-0,772	0,440
CDR	-2,932	0,003*
DsR	-3,858	0,000*
DIR	-1,389	0,165

Table A.9: Sign test for ranks for the condition of eyes-open and eyes-closed for non-normally distributed parameters that did not passed the symmetric test.

	Eyes Open		Eyes Closed	
	Correlation Coefficient	Sig. (2-tailed)	Correlation Coefficient	Sig. (2-tailed)
HsMD	0,255	0,293	-0,164	0,503
HIMD	0,114	0,641	0,150	0,539
HsAP	-0,155	0,527	0,029	0,907
HIAP	0,023	0,926	0,245	0,312

Table A.10: Correlation between normally distributed parameters and Pain Scale

	Eyes Open		Eyes Closed	
Kendall's tau_b	Correlation Coeff.	Sig. (2-tailed)	Correlation Coeff.	Sig. (2-tailed)
CTMD	0,202	0,253	0,037	0,831
CDMD	0,087	0,618	0,050	0,776
DsAP	-0,012	0,943	-0,012	0,943
DlMD	0,162	0,354	0,237	0,176
DsMD	0,000	1,000	0,187	0,285
DlAP	-0,025	0,887	0,000	1,000
CTAP	-0,025	0,887	-0,012	0,943
CDAP	-0,050	0,776	0,187	0,285
RMS	-0,175	0,318	0,125	0,476
Velocity	-0,075	0,669	0,162	0,354
Mean Displacement	-0,200	0,254	0,175	0,318
Area	-0,137	0,433	0,050	0,776

Table A.11: Correlation between normally distributed parameters and Pain Scale

Figure A.1: Group means and standard deviation for the critical time under the condition of eyes-open (EO) and eyes-closed (EC). The symbol * denotes $P<0.05$.

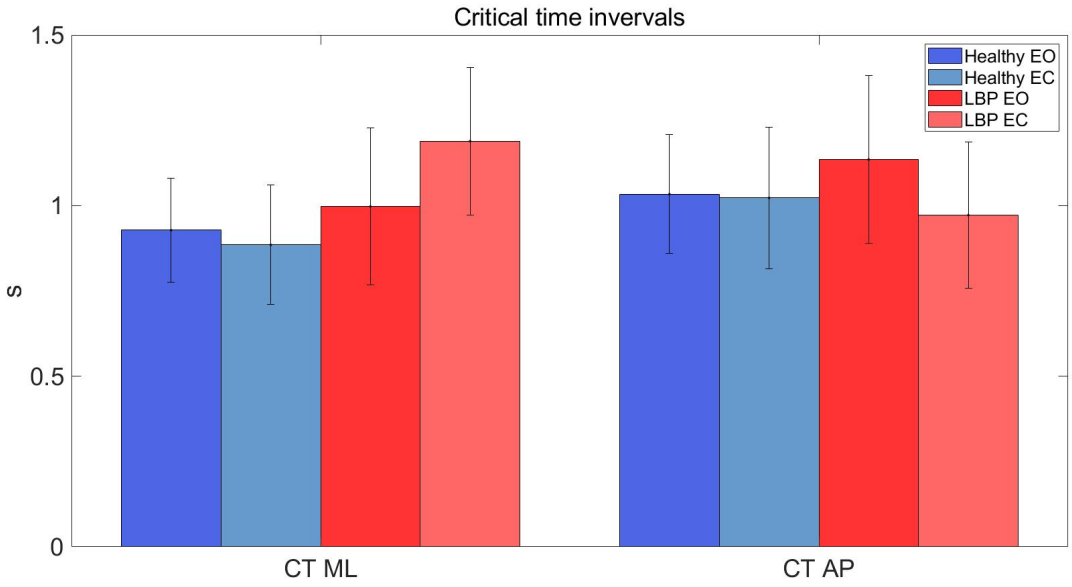

Figure A.2: Group means and standard deviation for the critical displacement exponent under the condition of eyes-open (EO) and eyes-closed (EC). The symbol * denotes P$<$0.05.

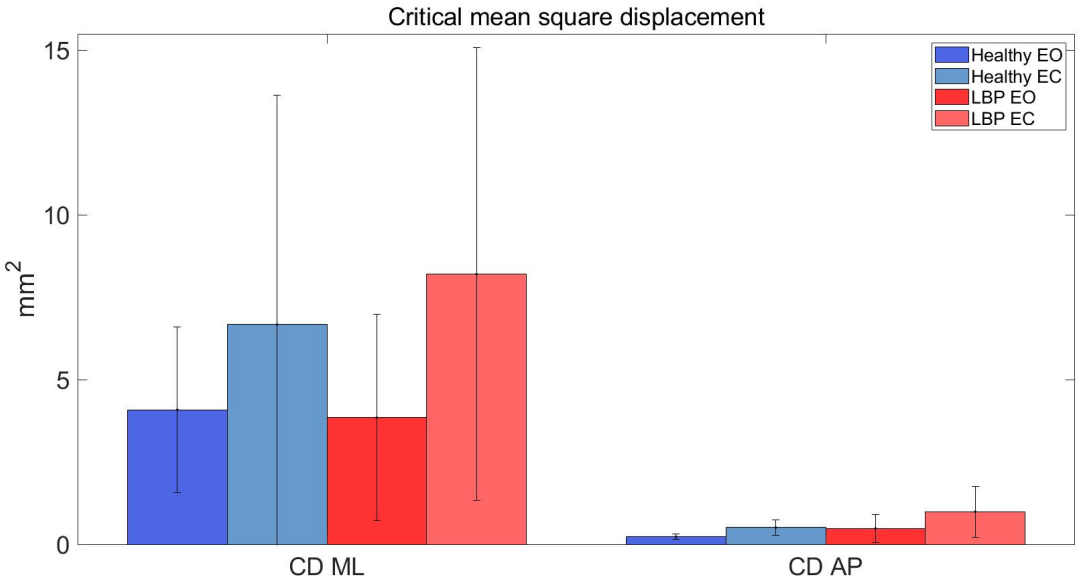

Figure A.3: Group means and standard deviation for the Short-term diffusion coefficient under the condition of eyes-open (EO) and eyes-closed (EC). The symbol * denotes P$<$0.05