Non-specific chronic low back pain elicits kinematic and neuromuscular changes in walking and gait termination

Lorenzo Rum[a*], Paolo Brasiliano[a], Giuseppe Vannozzi[a], Luca Laudani[b], Andrea Macaluso[a]

[a] University of Rome "Foro Italico", Piazza Lauro De Bosis 6, 00135 Rome, Italy

[b] Cardiff Metropolitan University, Cyncoed Campus, Cyncoed Road, CF23 6XD Cardiff, UK

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**Corresponding author*
Lorenzo Rum, PhD
Department of Movement, Human and Health Sciences, University of Rome "Foro Italico"
Piazza Lauro De Bosis 6, 00135 Rome – ITALY
Tel. 0039 06 36733522 - Fax. 0039 06 36733517
E-mail address: lorenzo.rum@uniroma4.it

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Abstract

Background: Chronic low back pain (CLBP) is associated with an increased trunk stiffness and muscle coactivation during walking. However, it is still unclear whether CLBP individuals are unable to control neuromechanically their upper body motion during a sudden termination of gait (GT), which involves a challenging balance transition from walking to standing.

Research question: Does CLBP elicit neuromuscular and kinematic changes which are specific to walking and GT?

Methods: Eleven individuals with non-specific CLBP and 11 healthy controls performed walking and sudden GT in response to an external visual cue. 3D kinematic characteristics of thorax, lumbar and pelvis were obtained, with measures of range of motion (ROM) and intra-subject variability of segmental movement being calculated. Electromyographic activity of lumbar and abdominal muscles was recorded to calculate bilateral as well as dorsoventral muscle coactivation.

Results: CLBP group reported greater transverse ROM of the lumbar segment during walking and GT compared to healthy controls. Thorax sagittal ROM was higher in CLBP than healthy participants during GT. Greater overall movement variability in the transverse plane was observed in the CLBP group while walking, whereas GT produced greater variability of lumbar frontal motion. CLBP participants showed higher bilateral lumbar coactivation compared to healthy participants after the stopping stimulus delivery during GT.

Significance: These results suggest that CLBP can elicit a wider and more variable movement of the upper body during walking and GT, especially in the transverse plane and at lumbar level. Alterations in upper body motor control appeared to depend on task, plane of motion and segmental level. Therefore, these findings should be considered by practitioners when screening before planning specific training interventions for recovery of motor control patterns in CLBP population.

Keywords Walking, gait termination, chronic low back pain, trunk muscle coordination, upper body kinematics.

Introduction

Chronic low back pain (CLBP) is a major leading cause of years lived with disability worldwide, most commonly arising in a non-specific form (e.g. unknown pathoanatomical cause of the pain) [1]. CLBP can affect the execution of many activities of daily living, resulting in changes in biomechanics and neuromuscular activity [2,3]. Previous studies on movement analysis of functional tasks, such as lifting or sit-to-stand, have commonly reported altered ranges of motion (ROM), angular velocity and acceleration of thoracic and lumbar spinal segments in CLBP patients [3]. Although research has shown ambiguous results due to lack of details and standardisation across studies, it has highlighted that CLBP patients are particularly challenged when shifting from a given posture to another (i.e. sit-to-stand). Thus, transitory motor tasks are deemed of interest in the evaluation of postural control in this population.

Neuromuscular and biomechanical alterations in upper body motor control associated with CLBP has also been observed during walking [4–7]. In particular, CLBP patients show an altered spinal movement and coordination as well as anticipated and higher trunk muscle activation compared to asymptomatic individuals. In a study by Lamoth et al. [8] on the effects of sudden changes in walking velocity on trunk-pelvis coordination and lumbar muscle activity, CLBP individuals demonstrated a reduced ability to react to such changes compared to healthy controls while attempting to stabilise the spine. However, it is still unclear whether this impaired reactive motor control of the upper body in CLBP patients could be exacerbated in transitory tasks that are featured by rapid decline in walking velocity, as in gait termination.

The need to slow down and smoothly arrest walking in response to external stimuli is quite common in daily life (i.e. when traffic light turns red). From a neuromechanical point of view, gait termination (GT) requires the exertion of braking forces that are necessary to arrest forward locomotion, with the interaction between biomechanical and neuromuscular components of movement being more complex when it is performed suddenly and without prior planning [9,10]. Depending on whether the

final stopping position is either known or unknown by the individual, the GT task can be defined as "planned" or "unplanned", respectively [10,11]. In planned GT, anticipation is possible, leading to increased stopping time and decreased rate of force development and peak posterior braking force compared to unplanned GT [10,11]. The probability of stopping stimulus occurrence also affects GT execution, with low probability level being associated with increased stopping distance and slower stopping response [9]. During planned GT, the backward momentum required to arrest forward progression is provided by the activation of posterior muscles (soleus and hamstring) in the weight-bearing limb, followed by the activation of knee and ankle extensors (soleus and quadriceps) in the swing limb [12]. A synergistic activation of lumbar erector spinae and gluteus medius accompanies the muscle activation pattern in lower limb muscles to prevent hip flexion and forward trunk movement due to body deceleration [12–14]. Previous work has shown the active role of upper body in arresting forward locomotion, highlighting the need for its control and stabilisation while stopping [15]. Therefore, it could be reasonable to hypothesise that the reduced motor control and altered coordination of upper body segments in CLBP individuals could lead to an altered ability to terminate gait, thus revealing different spinal motion and/or muscle activation patterns that could undermine balance maintenance or stress spinal structures.

This study aimed to compare upper body kinematics and trunk muscle activation between asymptomatic and CLBP individuals during walking and during sudden GT. We hypothesised that the execution of GT may emphasise the alterations associated with CLBP that have been previously observed during walking and show additional features that are specific to the task.

Methods

Participants

Eleven participants with non-specific chronic low back pain (CLBP; 7 females, 4 males) and 11 healthy controls (Healthy; 7 females, 4 males) participated in the study. Non-specific CLBP was defined as a condition of lumbar pain occurring at least four days per week during the three months previous the recruitment and which was not related to any known pathoanatomical cause [1]. Chronic pain intensity was evaluated at recruitment stage through a Visual Analog Scale (VAS) from 0 (no pain) to 10 (extreme pain) and all CLBP participants reported mild-to-moderate level of pain (min-max: 4-7) [16]. Exclusion criterion for both groups was the presence of any musculoskeletal and neurological pathologies (other than lumbar pain in CLBP group) and/or pharmacological therapy that can influence the balance and walking capacity. Healthy participants were excluded if they had any previous episodes of low back pain that needed medical treatment or caused any disability or limitation in the activity of daily living. The study protocol was approved by the institutional review board and all participants gave informed consent before testing.

Experimental protocol and set-up

The following pain-related measures were evaluated for characterization of CLBP group. Intensity of perceived current pain was assessed through a VAS at the beginning of the experimental session and no participant reported worsening in perceived pain to the experimenter over the testing session. The Italian version of Oswestry Disability Index (ODI) and the Tampa Scale of Kinesiophobia (TSK) were administered to provide information about the influence of CLBP on the activities of daily living and participant's fear of self-damaging during movement, respectively [17,18].

A seven-infrared camera motion capture system (Vicon MX3, Oxford, UK) was used to reconstruct the 3D position of 35 retro-reflective markers placed on the participant body landmarks according to the 15 body segments Plug-in Gait model (Vicon, Oxford, UK) with a sampling rate of 100 Hz. From this model, the whole-body CoM was obtained as well as the Cardan angles of the thorax and pelvis

relative to the global reference frame of the laboratory. In particular, the thorax and pelvis segments were defined by four markers each that were positioned on C7, T10, jugular notch and sternum (xiphoid process), and the anterior and posterior right and left superior iliac spines, respectively. Lumbar kinematics was also evaluated from the relative angle between thorax and pelvis segment axes. A dynamometric platform (Bertec Corp, Worthington, OH) embedded into the floor was located in the middle of a 10-meter walkway to measure the ground reaction force (GRF) with a sampling rate of 1000 Hz. A surface electromyography (EMG) device (PocketEMG, BTS Bioengineering, Italy) recorded muscular activity of lumbar erector spinae (LES) and the external obliques (EO) bilaterally via bipolar disposable electrodes (Ag/AgCl, 1 cm disc-electrodes, 2 cm inter-electrode distance) at a sampling rate of 1000 Hz. Electrodes for LES recording were placed parallel to the spine 2 cm laterally to the L3 spinal process, while EO electrodes location was parallel to muscle fibres and halfway between the anterior superior iliac spine and the lower crest of the last rib [19]. To improve signal quality, the skin was previously shaved and cleaned with a scrub solution, and the electrode wires were secured with tapes on the participant's skin. Signals from these three systems (motion capture, force plate and EMG device) were electronically synchronized through a trigger box (Trigger Box, BTS Bioengineering, Italy).

Participants were asked to walk straight at their comfortable pace while fixing the gaze on a black visual target that was displayed on a screen at the end of the 10-meters walkway. Four familiarization trials were performed to adjust the participant's starting position, thus enhancing the probability that the whole foot sole of the dominant leg always hits a force plate correctly (Fig.1); all participants in the study were right-leg dominant. First, each participant performed ten unconstrained walking trials at their most comfortable speed (UW condition). Then, participants performed a block of 60 walking trials during which a stopping signal was randomly delivered with a 20% occurrence requiring the participants to suddenly terminate gait as soon as they perceived the stopping signal (12 GT trials). Real-time data from the force plate were streamed through Vicon DataStream SDK (Vicon, Oxford,

UK) to Matlab R2016a software (Mathworks, Natick, MA) and used to deliver the stopping signal, which consisted in the black visual target turning red when the vertical GRF raised above 50 N. Particularly, when the stopping signal occurred, participants were asked to terminate gait within the next two steps and to arrest with both feet parallel (Fig.1). In case of no stopping signal, the participant was asked to continue walking as in the first 10 trials (Fig.1, white footprints). Trials were discarded from analysis if the right heel did not hit the force plate or multiple steps were performed to arrest the gait, resulting in at least 9 trials per participant being analyzed.

Three sub-maximal voluntary isometric contractions (SubMVIC) were performed at the beginning of testing session to obtain reference values for EMG normalisation of LES and EO muscle activity [20]. During LES SubMVIC, the participants were lying in a prone position with both knees at 90° and were asked to raise the legs 5 cm for 5 seconds. During EO SubMVIC, participants were asked to raise both legs 1 cm for 5 seconds from a supine lying position with the hips flexed at 45° and knees bent to 90°.

FIGURE 1 HERE

Data analysis

The stride after the heel contact with the force plate during the UW trials was selected for analysis. As shown in Fig. 1, the stride was defined based on the following events: first (RHC1) and second right heel contact (RHC2) as well as the left heel contact in-between (LHC). RHC1 was identified as to when vertical GRF raised above 50 N, while RHC2 and LHC were identified as the following lowest point of right and left heel marker vertical component, respectively. First step and stride length were calculated as the anteroposterior (AP) distance between right heel marker and left heel marker at LHC and the displacement of right heel marker in the AP direction from RHC1 to RHC2, respectively, and then normalised by participant's leg length. First step and stride time were defined

as the time interval from RHC1 to LHC and from RHC1 to RHC2, respectively. Mean walking speed was estimated from the AP CoM speed over the entire stride.

During GT trials, RHC1, LHC and RHC2 events were used to divide the task into two phases: *i*) Approaching Phase (Ph1), lasting from RHC1 to LHC, and *ii*) Braking Phase (Ph2), lasting from LHC to RHC2 (Fig.1). For these trials, the instant of complete stop was also identified as when AP CoM speed fell below 0.05 m/s [21]; instantaneous walking speed at RHC1, LHC and RHC2 was estimated from AP CoM speed to evaluate the rate of deceleration over the GT task. Length and time of the left step following the stopping signal were calculated as in the UW condition. Stopping distance and stopping time were defined as the AP displacement of right heel marker from RHC1 to the full stop and the time interval between the two events, respectively.

Kinematics data were low-pass filtered (fourth-order Butterworth filter, 5 Hz) and, then, segmented according to the previously defined gait events. ROM of thorax, lumbar and pelvis in the sagittal, frontal and transverse planes were calculated as the difference between maximum and minimum angular position over the complete stride in UW trials and during Ph1 and Ph2 in GT trials. Intra-subject movement variability of thorax, lumbar and pelvis segments was assessed through average standard deviation (AvgSD) of angular displacements of each body segment during the stride (RHC1-RHC2) in the UW trials and during Ph1 (RHC1-LHC) and Ph2 (LHC-RHC2) in the GT trials [22].

Raw EMG signals were band-pass filtered (third-order Butterworth filter, 20-450 Hz), high-pass filtered to remove electrocardiogram contamination (fourth-order Butterworth filter, 30 Hz) [23], demeaned and then full-wave rectified. The onset of EMG burst during SubMVIC test was obtained through the integrated protocol method [24] and the root mean square of EMG signal during the following 3 seconds was computed. Then, the mean value across the three SubMVIC trials for both LES and EO was selected for EMG normalisation during Walk and GT trials. After normalisation, EMG signal was furtherly low-pass filtered (fourth-order Butterworth filter, 20Hz) and integrated during RHC1-LHC and LHC-RHC2 intervals in Walk and GT trials to calculate coactivation indexes

for evaluation of bilateral (CI_{LES} and CI_{EO}) as well as dorsoventral co-contraction (CI_{dv}) [25]. CI_{LES} and CI_{EO} index values greater than 50% indicate a greater contribution of right-side musculature to total bilateral muscle activity, while CI_{dv} value above 50% corresponds to greater activation of dorsal muscles compared to ventral muscles. To evaluate the muscle response to the stopping signal during GT, the onset of muscle activation with respect to signal delivery was obtained from the filtered and normalised EMG through the integrated protocol method and visually checked by an experienced experimenter [24] (Fig.2). The onset of muscle activation was not computed for EO muscles as no clear bursts were identified, therefore only LES results are presented. The peak of the EMG burst, as well as its latency with respect to the stopping signal delivery (RHC1), were obtained from both LES and EO (Fig.2).

FIGURE 2 HERE

Statistical analysis

SPSS 23.0 software (Chicago, IL, USA) was used to perform all statistical analyses. Normal distribution of data was assessed through the Shapiro-Wilk test, and Tukey's power transformation was used on non-normally distributed data. To evaluate the effect of adaptation to the stopping stimulus during the GT condition on both spatio-temporal, kinematic and EMG parameters, a mixed ANOVA with the trial sequence as within-subject factor and group as between-subject factor was performed. No significant effect of trial sequence was found and, consequently, individual mean values were calculated and used for further analysis. Independent t-tests were used to evaluate differences between groups in demographic and anthropometric data, pain-related measures and spatio-temporal, kinematic and EMG parameters. Significance α level was set at 0.05 and r was adopted as a measure of effect size, with small, medium and large effect sizes corresponding to 0.1, 0.3 and 0.5, respectively [26].

Results

Demographic and anthropometric data from both groups, and pain-related characteristics from the CLBP group are shown in Table 1. There was no difference in anthropometric characteristics between groups ($p > 0.05$). CLBP group was characterised by mild pain (VAS lower than 5), minimal disability (0-20% range in ODI) and a moderate level of kinesiophobia (22-32 range in TSK).

TABLE 1 HERE

There was no difference in spatio-temporal parameters between groups during either UW or GT (Table 2). Similar walking speed values were found at each heel contact event during GT between Healthy and CLBP groups.

TABLE 2 HERE

Table 3 reports that, in UW, lumbar ROM in the transverse plane was approximately 36% greater in CLBP than Healthy ($t(20) = -3.27$, $p < 0.01$, $r = 0.58$). During Ph1 of GT, thorax sagittal ROM and lumbar transverse ROM were respectively 25% and 32% greater in CLBP than Healthy ($t(20) = -2.41$, $p < 0.05$, $r = 0.47$; and $t(20) = -2.41$, $p < 0.05$, $r = 0.47$, respectively). There was no difference in ROM between groups during Ph2 of GT.

TABLE 3 HERE

Figure 3 shows AvgSD of thorax, lumbar and pelvis angular displacements in sagittal, frontal and transverse planes. During UW, CLBP participants showed greater AvgSD of pelvis angular displacement in the frontal plane ($t(20) = 3.24$, $p < 0.01$, $r = 0.58$) and greater AvgSD of thorax,

lumbar and pelvis angular displacements in the transverse plane compared to Healthy participants ($t(20) = -3.11$, $p < 0.01$, $r = 0.57$; $t(20) = -2.61$, $p < 0.05$, $r = 0.50$; and $t(20) = -2.98$, $p < 0.05$, $r = 0.55$, respectively). During Ph1 of GT, greater AvgSD of pelvis frontal and transverse angular displacements and lumbar transverse angular displacement were observed in CLBP compared to Healthy ($t(20) = -2.31$, $p < 0.05$, $r = 0.45$; $t(20) = -2.25$, $p < 0.05$, $r = 0.45$; and $t(20) = -2.22$, $p < 0.05$, $r = 0.44$, respectively). During Ph2 of GT, greater AvgSD of pelvis transverse angular displacements and lumbar frontal angular displacement were observed in CLBP compared to Healthy ($t(20) = -2.34$, $p < 0.05$, $r = 0.46$; and $t(20) = -2.54$, $p < 0.05$, $r = 0.49$, respectively).

FIGURE 3 HERE

CI analysis showed that CLBP and Healthy had similar CI_{LES}, CI_{EO} and CI_{dv} values during UW condition (Fig.4). In the GT condition, a statistically significant difference was found in CI_{LES} at Ph1 (Fig.4), with CLBP showing greater levels of bilateral co-contraction and lower activation of right-side LES musculature compared to Healthy ($t(20) = 3.14$, $p < 0.001$, $r = 0.57$).

FIGURE 4 HERE

As shown by the group ensemble average EMG profiles in Figure 2 and mean group parameters in Table 4, Healthy and CLBP showed similar latency of bilateral LES muscle activation with respect to the timing of stopping stimulus delivery during GT ($p > 0.05$). No difference in EMG activation peak and peak latency of both bilateral LES and EO muscles was found between groups ($p > 0.05$). Noteworthy, the removal of an outlier in the Healthy group highlighted a significant difference in the activation peak latency of right EO ($t(19) = -3.05$, $p < 0.01$, $r = 0.57$), with CLBP showing greater latency with respect to the stopping stimulus compared to Healthy.

TABLE 4 HERE

Discussion

The main finding of the present study is that CLBP participants showed an increased transverse motion at lumbar level and upper body movement variability during both level walking and GT, with an altered bilateral coactivation in the LES specific to GT but not level walking. The changes in neuromuscular activation elicited by CLBP appeared to be tuned to the mechanical requirements of GT, likely attempting to increase spinal stiffness before the braking action.

Current literature on CLBP has reported poor consistency among results about the ROM of upper body segments during walking [3]. While some authors have reported no difference at distinct upper body levels [5,27,28], others have highlighted a reduction in ROM of the pelvis and lumbar segments altogether with alteration in intersegmental coordination in comparison to healthy controls [4,6,7,29]. Interestingly, to the authors' knowledge, none of these previous studies required the participants to fix the gaze on a visual target while walking. In contrast with previous research, CLBP participants from this study showed greater transverse ROM of the lumbar segment and, specifically during gait termination, greater thorax flexion-extension range than healthy controls. Moreover, this greater range of motion was accompanied by an increase in angular movement variability, especially in the transverse plane. In comparing the present experimental circumstances to those in previous research, one could speculate that these conflicting results may be ascribed to the visual constraint of gaze fixation. In healthy participants, staring the gaze on a fixed target while walking results in an alteration of balance control when compared to free gaze, with a subsequent increase in trunk movement [30]. In CLBP individuals, the impaired motor control of trunk could amplify this postural alteration to visual constraints with the consequence of greater and more variable upper body movements. However, to the extent of author's knowledge, the effect of different gazing conditions on upper body stabilisation mechanisms in the CLBP population has not been investigated yet, thereby posing new directions for future research. Noteworthy, in line with previous studies, the main kinematic

differences were found in the transverse plane that, therefore, appears to better discriminate between CLBP and healthy individuals compared to other planes of motion [29,31].

The greater trunk flexion-extension motion during gait termination suggests an altered motor control in CLBP individuals that is critical for spinal stability while arresting forward locomotion [15], especially because it was observed in response to a stopping stimulus and before the braking phase. This hypothesis is also supported by the analysis of trunk muscle coactivation which showed greater bilateral co-contraction of lumbar muscles in CLBP than in Healthy participants. From a neuromuscular standpoint, the increase in right lumbar activation during the approaching phase should anticipate the postural perturbation that occurs at ground contact of the contralateral leg (LHC) at the beginning of braking phase. Interestingly, previous work has mentioned that CLBP patients might prepare for an upcoming perturbation by co-contracting muscles, as it usually happens in healthy individuals when appropriate anticipation is not possible [32]. Although in this study the stopping stimulus was delivered with a low-probability occurrence rate, the final stopping position of feet was known and the participants may have stored a stopping motor programme to be released at the time of stimulus delivery. In consideration of this, the hypothesis of a co-contraction strategy in anticipation of an upcoming perturbation in CLBP individuals would be in line with the present results, thereby indicating an impaired neuromuscular control of trunk. Furthermore, the increase in stiffness due to a lumbar co-contraction strategy could explain the greater reduction in lumbar transverse ROM from the approaching phase to the braking phase that was observed in CLBP compared to Healthy participants, as indicated by the greater angle values found in the first but not in the second phase. These neuromuscular and biomechanical changes appear adjusted to the mechanical requirements of the task, implying that the alterations in trunk motor control associated with CLBP present a task-dependent feature that should be taken into account by practitioners for effective treatment interventions and evaluations.

The analysis of the muscle response to the stopping stimulus indicated that CLBP participants had similar onset of LES muscle activation as well as peak amplitude and latency of both LES and EO EMG bursts compared to Healthy participants. Previous findings indicated that CLBP affects lumbar muscle activity during sudden and unexpected changes in walking velocity, with phase shifts, amplitude modifications and additional burst activities [8]. On the contrary, no difference between CLBP and Healthy participants was reported in time and amplitude EMG parameters in the present study. This could be ascribed to the different experimental paradigm across the studies. The sudden gait termination with known final stopping position of the feet represents a behavioural motor pattern that can be pre-planned, stored and released at the stopping stimulus delivery to arrest forward locomotion [33]. Moreover, the type of the stopping stimulus (visual) adopted in this study did not determine any additional mechanical effect on the body of the participant. On the other hand, sudden and unpredictable changes in velocity imposed at ground level on a treadmill require an adaptation of the muscle activity to cope with the balance perturbation while preserving a stable walking pattern. Therefore, the absence of an additional mechanical perturbation as well as the chance of storing and releasing a pre-planned gait termination motor programme may have allowed CLBP participants to overcome previously reported poor muscle control. Nevertheless, it is worth noting that, although time and amplitude EMG parameters were not different between groups, the evaluation of co-contraction indexes indicated a CLBP-related modification at lumbar level. This result suggests that particular attention should be given to the intermuscular coordination as it may be capable of unveiling more subtle changes than time and amplitude indicators of muscle performance.

This study presents some limitations that should be considered. The small sample size makes difficult to generalise the present results, although the effect size of significant findings was medium to large. Within the field of LBP research, the issue of low number of participants has been previously reported and, altogether with the heterogeneity of LBP population, it may be one reason of the ambiguous results across studies [3]. Moreover, a greater sample size in this study could have disclosed nearly

significant results, such as the delay in the latency of EO activation peak, thus providing a better understanding of the neuromuscular adaptation related to CLBP. Another limitation of this study may be the low level of disability observed in LBP participants. Although the VAS showed pain level similar or even greater than in other studies [4–6,29], the low disability level might represent a confounding factor as it is correlated to the motor adaptations that have been reported in LBP participants [34].

In conclusion, the present results suggest that CLBP can elicit a wider and more variable movement of the upper body during walking and GT, especially in the transverse plane and at lumbar level. The alterations in upper body motor control appeared to be dependent on task and, from a neuromuscular point of view, to be exacerbated when a sudden termination of gait is required in response to an external visual stimulus. Furthermore, the constraint of fixing the gaze on a visual target and the subsequent need for increased head stabilisation may represent a greater postural challenge for the impaired motor control of upper body in CLBP compared to healthy people, although this aspect still requires further investigation. Therefore, these findings should be considered by practitioners when planning effective screening protocols to identify the neuromuscular and kinematic changes associated with CLBP since different motor tasks (i.e. walking or sudden gait termination) with specific postural constraints (i.e. with/without visual target) could influence the observed results.

Conflict of interest

The authors declare no conflict of interest.

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TABLES

Table 1. Mean (SD) of anthropometric data and pain-related measures.

	Healthy	**CLBP**
Age (years)	28 (6)	26 (8)
Body mass (kg)	59.4 (6.7)	62.8 (7.0)
Body height (m)	1.68 (0.06)	1.67 (0.09)
VAS (0-10)		4.6 (2.4)
ODI (0-100%)		12.5 (3.4)
TSK (1-68)		30.0 (5.6)

Table 2. Mean (SD) of spatio-temporal parameters from UW and GT trials in both groups. *P* values of between group comparisons are provided. WS: walking speed. LL: leg length.

	Healthy	**CLBP**	***p* value**
Unconstrained Walking			
Mean WS (m/s)	1.42 (0.16)	1.41 (0.17)	0.832
Normalised stride length (m/LL)	1.78 (0.09)	1.80 (0.13)	0.626
Stride time (s)	1.10 (0.07)	1.12 (0.12)	0.511
Normalised step length (m)	0.90 (0.05)	0.88 (0.07)	0.524
Step time (s)	0.55 (0.04)	0.55 (0.05)	0.950
Gait termination			
WS at RHC1 (m/s)	1.42 (0.16)	1.39 (0.14)	0.642
WS at LHC (m/s)	1.33 (0.14)	1.29 (0.12)	0.443
WS at RHC2 (m/s)	0.33 (0.07)	0.31 (0.07)	0.682
Stopping distance (m)	0.44 (0.04)	0.43 (0.06)	0.592
Stopping time (s)	1.39 (0.10)	1.40 (0.12)	0.907
Normalised step length (m/LL)	0.52 (0.03)	0.51 (0.06)	0.631
Stepping time (s)	0.53 (0.04)	0.54 (0.04)	0.572

Table 3. Mean (SD) of thorax, lumbar and pelvis ROM in the sagittal, frontal and transverse planes during UW and the two phases of Gait termination from Healthy and CLBP groups. * = Significantly different compared to Healthy group ($p < 0.05$).

	UW		GT Ph1		GT Ph2	
	Healthy	**CLBP**	**Healthy**	**CLBP**	**Healthy**	**CLBP**
Thorax ROM (°)						
Sagittal	2.8 (1.0)	3.4 (0.7)	2.8 (0.5)	3.5 (0.7) *	3.3 (1.1)	3.1 (0.9)
Frontal	3.0 (1.4)	2.3 (1.1)	2.7 (1.1)	2.5 (0.4)	1.9 (0.3)	2.0 (0.6)
Transverse	7.4 (2.1)	8.1 (2.7)	6.1 (1.7)	6.3 (2.2)	6.4 (2.0)	6.5 (1.9)
Lumbar ROM (°)						
Sagittal	3.4 (2.3)	3.2 (1.9	3.4 (1.6)	3.3 (1.5)	3.4 (0.8)	3.7 (1.3)
Frontal	10.0 (2.2)	10.4 (5.1)	9.8 (1.6)	11.0 (3.5)	6.6 (1.6)	7.8 (2.7)
Transverse	9.4 (1.9)	12.8 (2.9) *	10.0 (2.0)	13.2 (3.9) *	6.0 (0.9)	7.1 (2.9)
Pelvis ROM (°)						
Sagittal	2.5 (1.3)	2.5 (1.4)	2.6 (1.1)	2.7 (1.3)	3.4 (0.9)	3.8 (1.9)
Frontal	7.3 (2.4)	8.1 (4.5)	7.3 (2.4)	8.5 (3.3)	5.8 (1.9)	6.3 (2.1)
Transverse	9.1 (3.5)	10.6 (3.9)	9.6 (4.5)	10.1 (3.7)	6.4 (1.8)	6.2 (1.8)

Table 4. Mean (SD) of EMG temporal and amplitude parameters with respect to the stopping stimulus during gait termination from Healthy and CLBP groups. *P* values of between group comparisons are provided. LES: lumbar erector spinae; EO: external obliquous.

	Healthy	**CLBP**	***p* value**
Muscle activation latency (ms)			
Right LES	404 (52)	426 (50)	0.192
Left LES	409 (56)	438 (39)	0.102
EMG peak (% SubMVC)			
Right LES	79 (26)	69 (27)	0.396
Left LES	57 (23)	64 (26)	0.503
Right EO	56 (26)	52 (32)	0.636
Left EO	82 (52)	70 (56)	0.433
EMG peak latency (ms)			
Right LES	536 (63)	587 (74)	0.101
Left LES	559 (67)	602 (103)	0.264
Right EO	465 (110)	540 (83)	0.070
Left EO	478 (85)	465 (82)	0.713

FIGURE CAPTIONS

Fig. 1. Stepping events during unconstrained walking (1, 2 and 3a) and gait termination (1, 2, 3b) as well as positioning of the screen that was used to display the visual target and to deliver the stopping stimulus along the 10-meters walkway. 1: right heel contact with the force platform (RHC1); 2: left heel contact (LHC); 3a: second right heel contact during unconstrained walking condition (RHC2); 3b: second right heel contact during gait termination condition (RHC2).

Fig. 2. Ensemble average of bilateral LES (top) and EO (bottom) muscle activity profiles from Healthy (grey) and CLBP group (black) during gait termination. EMG signals are aligned to the instant of stopping stimulus delivery (vertical line, STOP), which corresponds to RHC1. The parameters of muscle recruitment onset for LES muscles and muscle activation peak for both LES and EO muscles are displayed on the left side only for representative purpose.

Fig. 3. Mean and standard deviation of AvgSD of thorax, lumbar and pelvis angular displacements in the sagittal (top), frontal (middle) and transverse planes (bottom) during unconstrained walking and the two phases of gait termination for both groups. * = Significantly different between groups ($p < 0.05$).

Fig. 4. Mean and standard deviation of CI_{LES}, CI_{EO} and CI_{dv} computed during the RHC1-LHC (left) and LHC-RHC2 time intervals (right) during unconstrained walking (UW) and gait termination (GT) for Healthy and CLBP groups. Horizontal dotted line indicates the value of 50% that refers to an equal contribution of sides (right-left in CI_{LES} and CI_{EO}, dorso-ventral in CI_{dv}) to total muscle activation. * = Significantly different between groups ($p < 0.005$).

Figure 1

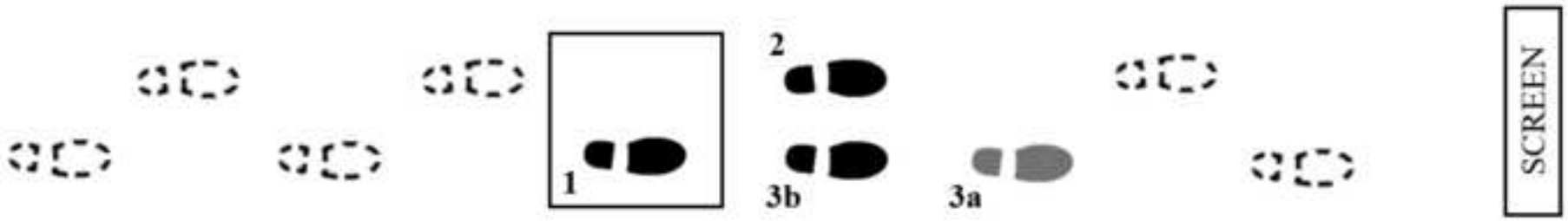


Figure 2

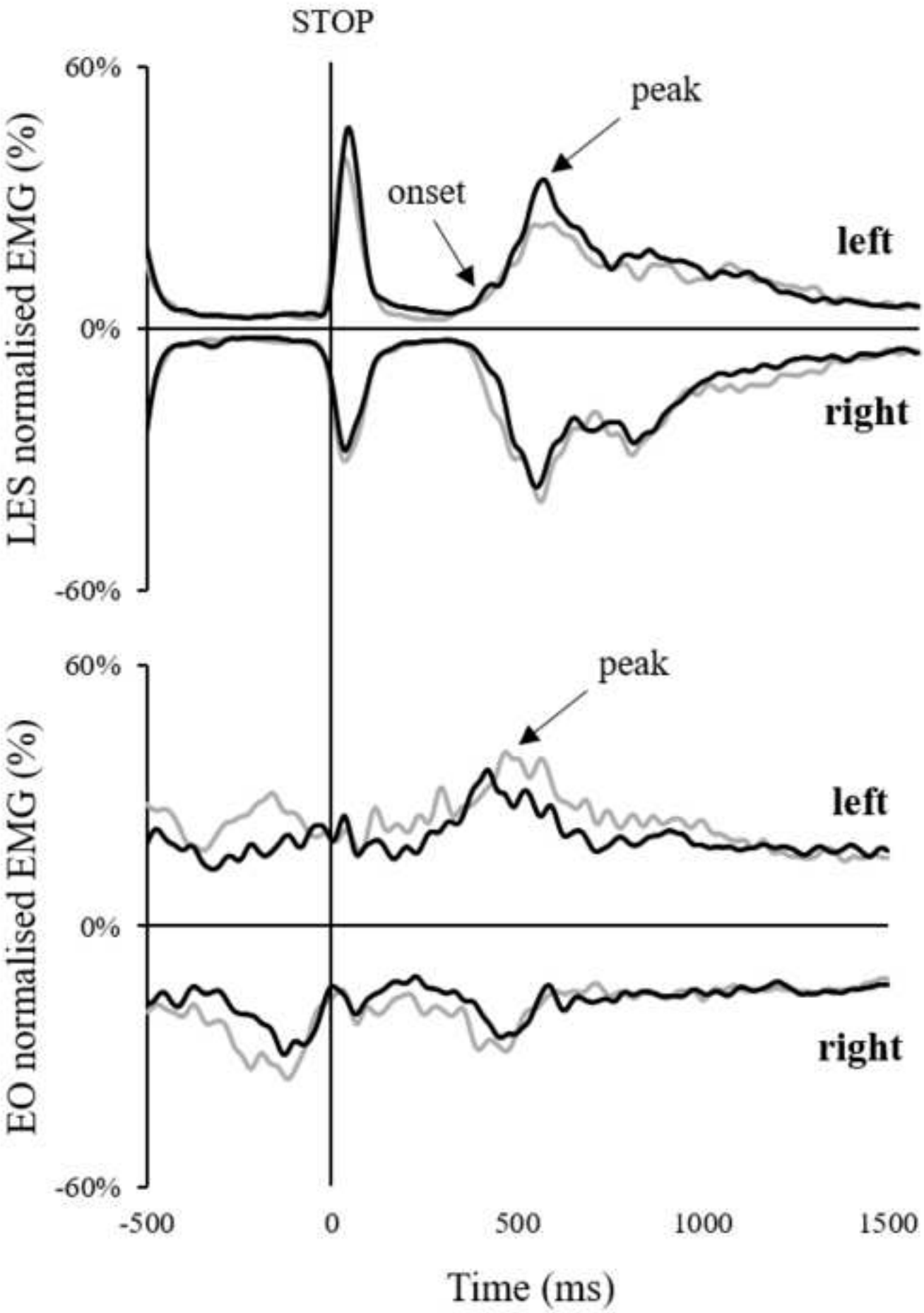

STOP
60%
peak
onset
left
0%
right
-60%
LES normalised EMG (%)
60%
peak
left
0%
right
-60%
EO normalised EMG (%)
-500
0
500
1000
1500
Time (ms)

Figure 3

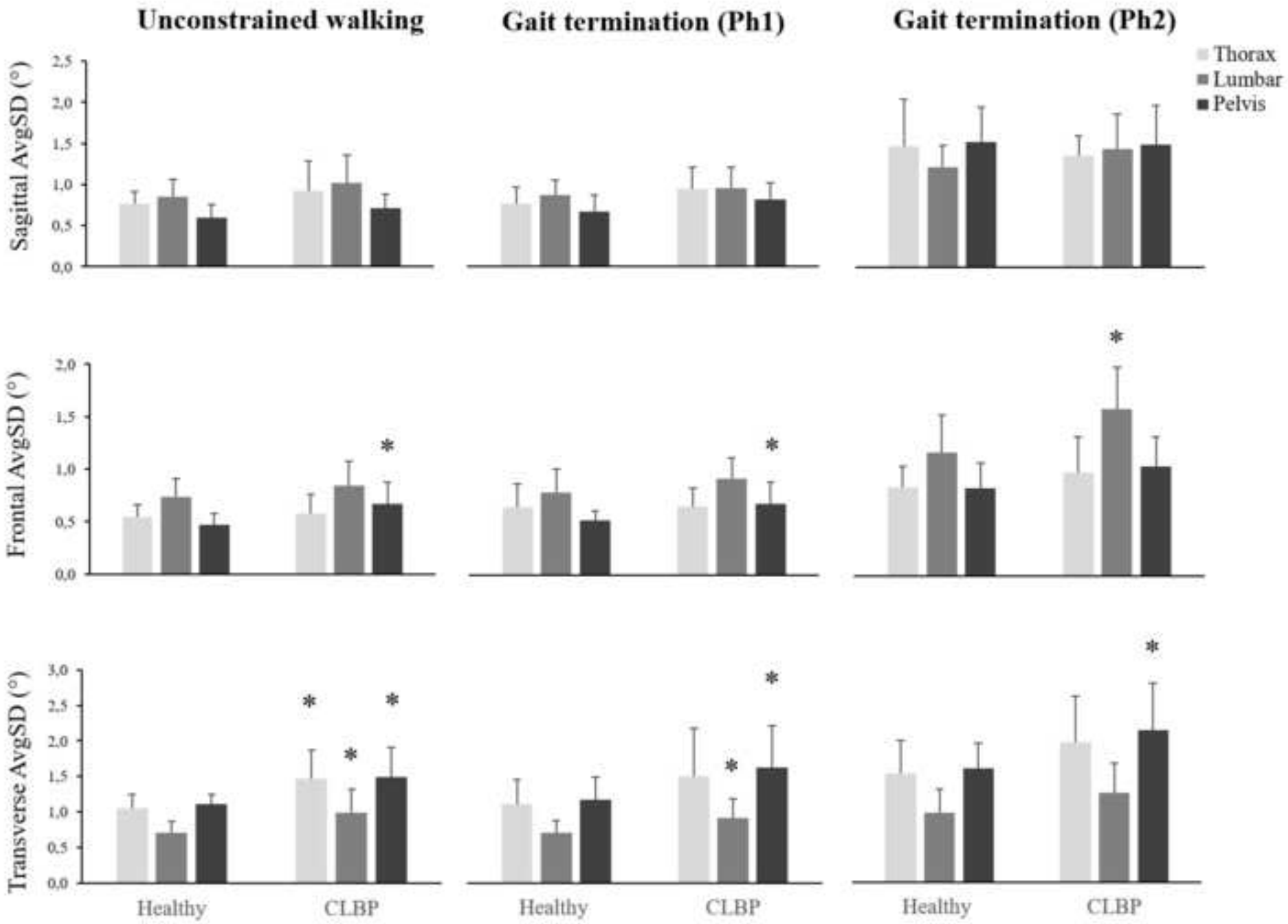
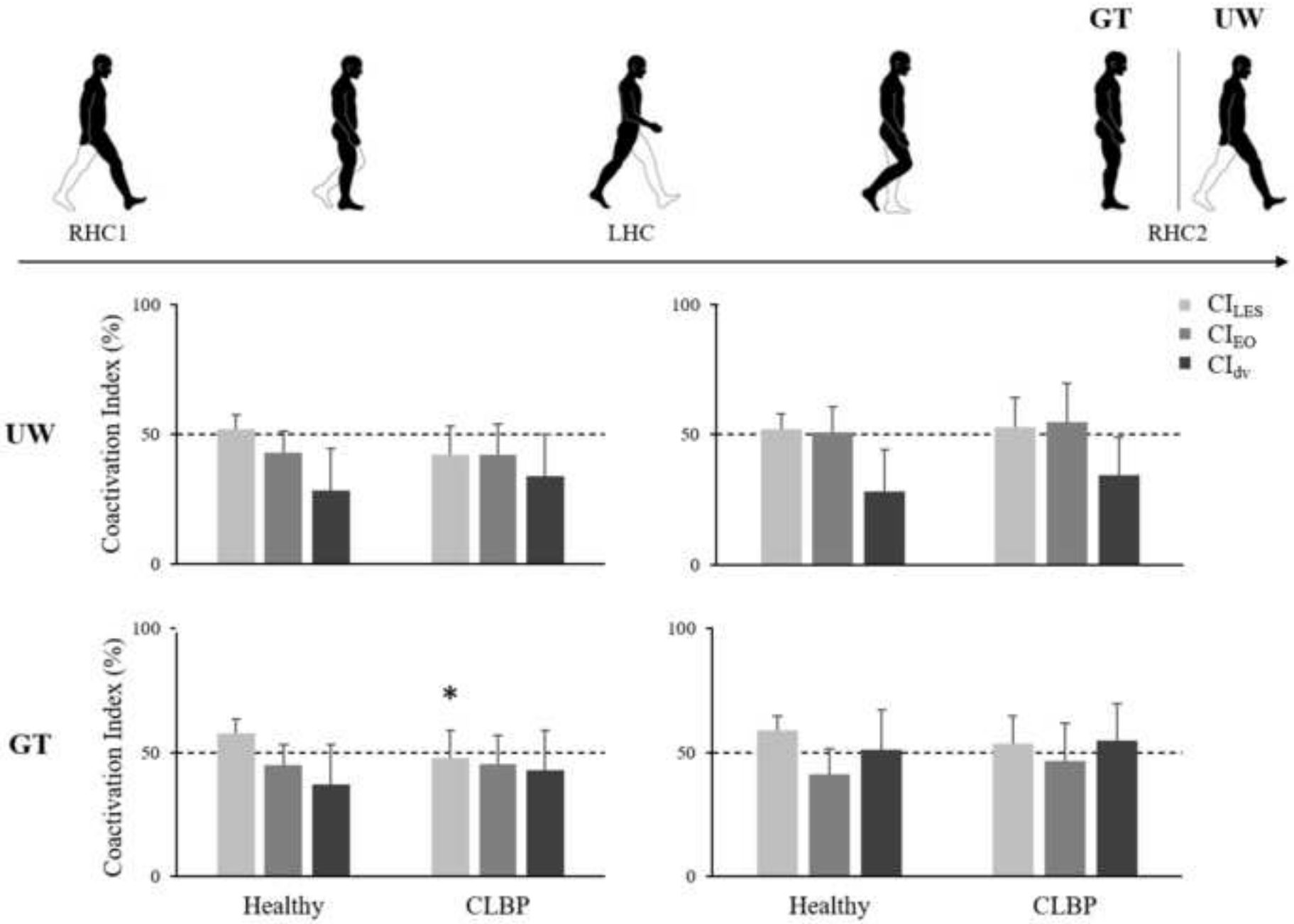

Unconstrained walking
Gait termination (Ph1)
Gait termination (Ph2)
Thorax
Lumbar
Pelvis
Sagittal AvgSD (°)
Frontal AvgSD (°)
Transverse AvgSD (°)
Healthy
CLBP

Figure 4


GT
UW
RHC1
LHC
RHC2
UW
GT
Coactivation Index (%)
100
50
0
CI_{LES}
CI_{EO}
CI_{dv}
*
Healthy
CLBP