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Original Article

# Restoration of the Broken Lumbopelvic-hip Neuromuscular Chain and Coordinated Synergistic Activation in Low Back Pain

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**Key Words**
Abdominal muscles
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**Background:** The presence of the lumbopelvic-hip neuromuscular chain is essential for dynamic spinal stabilization; its therapeutic effects on dynamic movements of the distal extremity segment and underpinning motor mechanism remain unknown and warrant further study on participants with low back pain (LBP).

**Objects:** We aim to compare the effects of the broken chain exercise (BCE) and connected chain exercise (CCE) on electromyography (EMG) amplitude and onset time in participants with and without LBP.

**Methods:** Randomized controlled clinical trial. A convenience sample of 40 nonathletic participants (mean age: 24.78 ± 1.70) with and without LBP participated in this study. All participants underwent CCE for 30 minutes, 30-minute daily. We measured EMG amplitude and onset times on bilateral erector spinae (ES), gluteus maximus (GM), hamstring (HAM), transverse abdominis (TrA), internal oblique (IO), and external oblique (EO) during the prone hip extension (PHE) test before and after the BCE and CCE. We used multivariate analysis of variance (MANOVA) to analyze the amplitude and onset time difference between exercises (BCE and CCE) and Pearson's correlations to determine any synergistic relationship among the HAM, GM, bilateral TrA/IO, and ES muscles. The statistical analyses were used at $p < 0.05$.

**Results:** MANOVA showed that CCE was more decreased on EMG amplitude in HAM and bilateral ES, while increased GM and contralateral TrA/IO than BCE ($p < 0.05$). MANOVA EMG onset time data analyses revealed that the main effect of the conditions was significant for all HAM, GM, and bilateral ES muscles, whereas the main effect for the group was significant only for GM and contralateral ES in healthy and LBP groups. Pearson's correlation coefficient was computed to assess the relationship between BCE and CCE on dependent variables. In most of the muscles, there was a strong, positive correlation between the two variables, and there was a significant relationship ($p < 0.001$).

**Conclusion:** CCE produced more effective and coordinated core stabilization and motor control mechanism in the lumbopelvic-hip muscles in participants with and without LBP during PHE than BCE.

## INTRODUCTION

The motor control dysfunction in the lumbopelvic-hip neuromuscular chain (LNC) has been identified as an important biomarker for low back pain (LBP) [1,2]. LNC is anatomically composed of 29 muscles connected between the core-lower extremity as the lumbopelvic-hip complex where our center of gravity is located and where all movements superimposed on the core stability embark [3,4]. Coordinated orchestration of the 29 lumbopelvic-hip muscle activation, length-tension, and force-couple relationships generates optimal arthrokinematics and osteokinematic movements in the lumbopelvic-hip complex during functional kinetic LNC movements. This provides a neuromuscular efficiency in the entire kinetic chain, producing acceleration, deceleration, and dynamic stabilization of the entire kinetic chain during functional extremity movements while maintaining proximal stability [5].

The electromyography (EMG) measurement during prone hip extension (PHE) has been examined in terms of EMG activation onset time sequence and amplitude in the participants

with and without LBP to objectively quantify motor control pattern in the LNC [6,7]. Normally, the EMG muscle activation sequence in the LNC involves the gluteus maximus (GM) and hamstring (HAM), followed by the contralateral erector spinae (ES) and the ipsilateral ES sequence PHE movement test. Similarly, during PHE, GM activation was the largest (52.78 %maximum voluntary isometric contraction [%MVIC]), followed by the contralateral ES (46.86 %MVIC) and HAM (29.81 %MVIC) in normal controls [6]. However, altered EMG muscle activation patterns, including delayed onset time and underactivity in the GM of individuals with LBP, were observed when compared with those of normal controls during the PHE movement test [8-10]. Specifically, earlier onset of HAM activation (30 ms) has been noted in patients with LBP as compensation for the delayed firing of the GM (50 ms) and contralateral ES [11-13]. Similarly, the EMG amplitude in the contralateral ES was the largest (72.11 %MVIC), followed by ipsilateral ES (70.74 %MVIC), the semitendinosus, and GM (42.32 %MVIC) in LBP [13]. Such altered EMG activation patterns justified the fact that coordinated activation of the lumbopelvic stabilizers (ES, external oblique [EO], internal oblique [IO], and transverse abdominis [TrA]) is a prerequisite to stabilizing the lumbopelvic muscles to control the hip during dynamic PHE. Thus, chain breakage can alter the motor recruitment firing sequence and amount of activation and associated interactive joint moments in the lumbar spine as a function of the compensatory mechanism, particularly in LBP, which is clinically manifested in compressive force-induced discogenic LBP, commonly in L4-5/lumbosacral [14,15]. Therefore, reconnecting or restoring an LNC chain breakage is crucial when intervening with the underlying cause of the LBP associated with motor control dysfunction, targeting to "normalize" the altered LNC muscle activation sequence and amplitude patterns.

It has been proposed that proximal segment (core) stabilization needs to be established and connected to the broken adjacent segment to sufficiently distribute energy and generate force in the coordinated LNC to mitigate the altered LNC muscle activation patterns in individuals with LBP. This concept of normalization of the LNC neuromuscular chain muscle activation was derived from developmental kinesiology [16,17]. The normalization technique of the LNC involves a coordinated coactivation of the deep and superficial muscles in the LNC system [16,17]. Specifically, the deep muscle facilitation technique includes the coactivation of the diaphragm, TrA-multifidus-pelvic floor, IO, and EO muscles by asking the patent to inhale, generating internal stabilization. For superficial muscle facilitation, external core stabilization force, which is connected to hip extensor muscles [18]. Specifically, it has been theorized that the deep core stabilization muscles (diaphragm-TrA/IO-multifidus-pelvic floor) automatically create internal abdominal pressure (IAP) during the inspiration phase, which was further stabilized by reflexive coactivation of the external core stabilizers (EO, rectus abdominis, and ES) [19]. Furthermore, the integrative internal and external core stabilization forces in the proximal core segment generate sufficient stabilization or moving forces in response to external perturbation and dynamic movements of the distal extremity segment [19,20], as apparent during PHE movement. However, such LNC chain coactivation and associated motor control mechanisms remain unknown and warrant further study on participants with LBP. Thus, this research aimed to investigate the underlying motor control rationale for the LNC exercise on EMG motor control patterns in participants with and without LBP. We hypothesized that the LNC exercise would help normalize the EMG motor control patterns in terms of recruitment sequence and amplitudes in the lumbopelvic–hip muscles in participants with and without LBP during PHE. Importantly, understanding the motor control mechanism of the LNC exercise is of great interest to clinicians when developing effective intervention strategies for nonathletic participants with LBP.

## MATERIALS AND METHODS

### 1. Participants

A convenience sample of 40 nonathletic participants (mean age: 24.78 ± 1.70; 20 healthy participants and 20 participants with LBP) were included in this study. The study protocol was approved by the Institutional Review Board of Yonsei University Mirae Campus (IRB no. 1041849-201812-BM-116-02) and was conducted in accordance with the Declaration of Helsinki. All participants provided informed consent before their participation.

The inclusion criteria entailed young participants (age ranged from 20 to 30 years) with and without a history of LBP. Twenty healthy participants were those without complaint of LBP for at least 6 months. Twenty participants with mechanical LBP, who had pain with a duration over at least 6 months and a history of at least one pain of LBP, which had limited their

activities in sports and work over the past 18 months, and had experienced LBP over the past 6 months, were also recruited [21]. Exclusion criteria of all participant included: (1) any current orthopedic or neurological impairments, (2) a history of previous back surgery, (3) ankle sprain [22], (4) hypertension and diabetes, (5) hip flexor shortness (confirmed by Thomas test) [10], and (6) HAM shortness (confirmed by the 90–90 test).

## 2. Electromyography Measurement

### 1) Experimental setup

The experimental setup for EMG measurement during the PHE test is shown in Figure 1. TeleMyo DTS 542 (Noraxon Inc., Scottsdale, AZ, USA) was used and analyzed using myoResearch software (Noraxon Inc.) and was low-pass filtered (500 Hz). We set the sampling rate at 1,500 Hz. We amplified the EMG signals with an input impedance of 100 MΩ, a common mode rejection ratio of 100 dB, and a gain of 400. We prepared the participants for electrode placement by thoroughly abrading and cleaning the skin with an alcohol-soaked cotton pad. The ground was in the shape of a circle and was placed center-to-center with a distance of 20 mm. Surface electrodes (Ag/AgCl) were placed in pairs parallel to the muscular fibers to obtain EMG data. The TrA/IO electrode was located approximately 20 mm medially and inferiorly from the anterior superior iliac spine. For the GM, the electrodes were placed at the midpoint of the line running from the last sacral vertebrae to the greater trochanter; for the semitendinosus and biceps femoris, medially and laterally on the mid-distance point between the gluteal fold and the knee joint; and for the bilateral ES muscles, at the L3 level, bilaterally 2 cm lateral from the spinal processes and parallel to the lumbar spine. We measured the root mean square of a 5-second MVIC for each muscle three times to normalize the data [23].

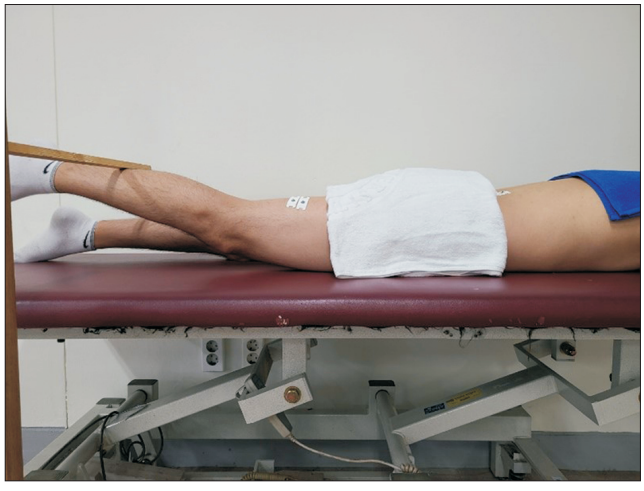

**Figure 1.** Electromyography measurement during the prone hip extension test.

### 2) Data acquisition

***Pretest (broken chain exercise, BCE).*** We conducted the PHE three times for each participant. The task required the participant to lie prone while EMG activity was measured for five seconds. Participants then extended their straight dominant leg approximately six inches using the target bar off the table. For each trial and for every participant, the data collection procedure underwent the same procedure.

The LNC is maintained broken during the pretest since it was measured before the intervention phase. Therefore, pretest measurements will be called "BCE" throughout the article.

***Intervention phase.*** The therapist introduced the participants to the connected chain exercise (CCE) steps as follows: (1) The therapist neutralizes a participant's thorax and ribcage in a quadruped position so that the subject can have diaphragmatic breathing naturally. (2) While maintaining this alignment, the therapist induces the subject to inhale descending participants' diaphragm and coactivate TrA/IO. (3) The participants are needed to correct the CCE movement surrounded that the 10–12 ribs were anteriorly and laterally expanded from the medioclavicular line and posteriorly, making sure that a cylinder barrel shape has been made. The corrective movement by the therapist must be ensured that the ribcage is not expanding toward the head direction in the participant's transverse section. All participants practiced the CCE exercise for 30 minutes. During core stabilization training, EMG was used to provide precise visual feedback on the target muscle activation and onset time of the TrA, IO, and EO [16,17].

***Posttest (CCE).*** After 30 minutes of intervention, in addition to the pretest, all participants measured EMG three times using the identical testing protocol during PHE under the CCE condition. We compared the muscle onset time and amplitude between BCE and CCE during PHE in the normal and LBP groups. Below is the experimental protocol (Figure 2).

## 3. Data Analysis

The descriptive data were expressed as mean ± standard deviations (SD). On the basis of our previous study, we computed the sample size using the G-power software (ver. 3.1; Franz

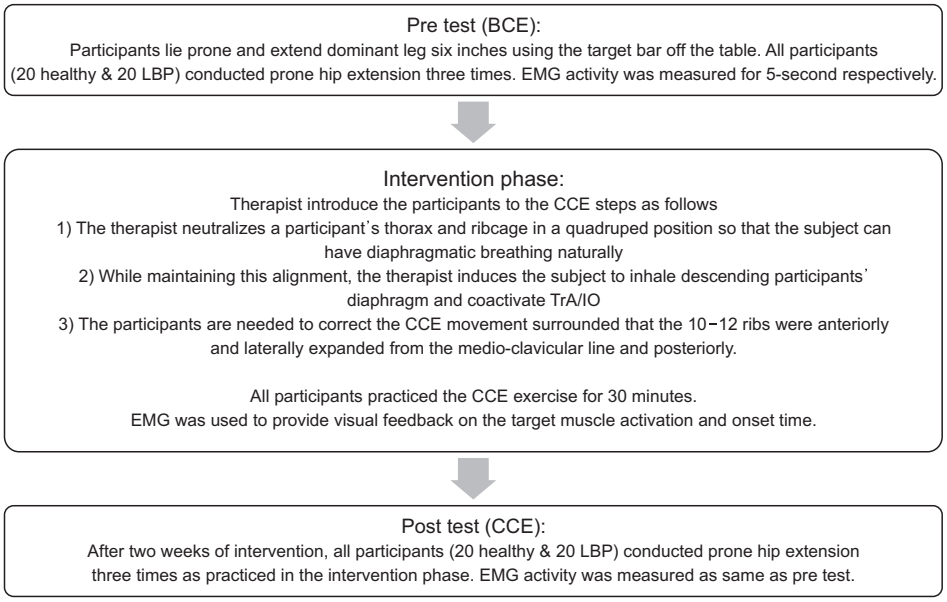


**Figure 2.** Flow chart for experimental protocol for data acquisition. BCE, broken chain exercise; LBP, low back pain; EMG, electromyography; CCE, connected chain exercise; TrA, transverse abdominis; IO, internal oblique.

Faul, University of Kiel, Kiel, Germany) based on the effect size (medium size 0.4) and power (0.8) [24]. We used the chi-squared test to analyze the categorical demographic variables and used the Kolmogorov–Smirnov test to confirm a normal distribution. We compared the general characteristics of the participants between the groups using independent t-tests. We used multivariate analysis of variance (MANOVA) to analyze the amplitude and onset time differences between groups (LBP vs. healthy controls) and within intervention (CCE vs. BCE) on eight dependent variables (EMG amplitude of the HAM, GM, bilateral TrA/IO, EO, and ES muscles and EMG onset time of the HAM, GM, and bilateral ES muscles) during PHE. When MANOVA revealed significant differences, we used univariate tests to determine a significant interaction or main effect at a significance level of $p < 0.05$. We conducted Tukey's test to find means that are significantly different from each other.

Additionally, we analyzed EMG onset times of HAM, GM, and bilateral ES with Pearson's correlation coefficient to determine any synergistic relationship among the HAM, GM, bilateral TrA/IO, EO, and ES muscles. The statistical analyses were performed using SPSS for Windows version 25.0 (IBM Co., Armonk, NY, USA).

## RESULTS

All 40 participants have successfully completed the pretest and posttest EMG measurements, and the results were included in the analysis. Table 1 shows the demographic data of the subjects. There were no significant differences in the baseline demographics.

**Table 1.** Demographic characteristics of the participants (N = 40, male)

| Characteristic | LBP group (n = 20) | Healthy group (n = 20) | p-value |
|---|---|---|---|
| Age (y) | 25.86 ± 2.79 | 26.29 ± 4.2 | 0.232 |
| Height (cm) | 172.53 ± 16.54 | 169.27 ± 12.75 | 0.171 |
| Weight (kg) | 74.42 ± 21.55 | 68.27 ± 19.37 | 0.334 |
| Dominant side (Lt/Rt) | 6/14 | 2/18 | 0.114 |
| Onset time (mo) | 5.1 ± 2.9 | None | NS |

Values are presented as mean ± standard deviation or number only. LBP, low back pain; Lt, left; Rt, right; NS, not significant.

Table 2 shows the EMG amplitude during PHE for each muscle group. For all the examined muscles, MANOVA EMG amplitude data analyses showed that there was no significant interaction effect for condition (BCE vs. CCE) × group (LBP vs. healthy). Muscle activity of ipsilateral TrA/IO, bilateral EO, and GM significantly increased, and muscle activity of bilateral ES decreased in the CCE compared with that in the BCE, which represents improved deep muscle activity after performing the CCE for subjects who suffered from LBP ($p < 0.05$). In addition, HAM, GM, bilateral ES, and contralateral TrA/IO showed a significant difference between LBP and the healthy group.

MANOVA was conducted to determine the effect of CCE and BCE in LBP and healthy participants on the four dependent variables, HAM, GM, and bilateral ES. Significant differences were found in GM and con ES onset time among the LBP and healthy participants during CCE and BCE. Table 3 shows the means and SD on the dependent variables for the two groups

**Table 2.** Electromyography amplitude during prone hip extension (N = 40)

| %MVIC | LBP group | | Healthy group | | p-value | | $R^2$ |
|---|---|---|---|---|---|---|---|
| | BCE | CCE | BCE | CCE | Condition main effect | Group main effect | |
| HAM | 86.22 ± 2.51 | 82.54 ± 3.28 | 98.35 ± 22.07 | 88.02 ± 3.83 | 0.393 | 0.016* | 0.732 |
| GM | 89.84 ± 3.90 | 95.05 ± 2.15 | 98.47 ± 4.27 | 108.77 ± 3.23 | < 0.001* | < 0.001* | 0.986 |
| Ip ES | 33.80 ± 2.62 | 23.02 ± 2.66 | 50.88 ± 3.41 | 40.74 ± 2.52 | < 0.001* | < 0.001* | 0.997 |
| Con ES | 64.74 ± 3.63 | 39.79 ± 2.21 | 51.54 ± 3.23 | 26.71 ± 3.23 | < 0.001* | < 0.001* | 0.993 |
| Ip TrA/IO | 36.76 ± 2.51 | 42.68 ± 3.87 | 36.14 ± 3.07 | 39.56 ± 3.54 | < 0.001* | 0.419 | 0.681 |
| Con TrA/IO | 42.79 ± 3.54 | 52.89 ± 3.58 | 35.86 ± 2.65 | 40.60 ± 3.59 | 0.111 | 0.031* | 0.917 |
| Ip EO | 44.75 ± 3.10 | 49.19 ± 4.21 | 38.77 ± 4.28 | 44.85 ± 7.91 | < 0.001* | 0.299 | 0.463 |
| Con EO | 44.53 ± 2.23 | 54.21 ± 3.35 | 41.51 ± 4.44 | 47.57 ± 4.94 | 0.015* | 0.192 | 0.669 |

Values are presented as mean ± standard deviation. The $R^2$ value ranges from 0 to 1 with 1 being perfect predictive accuracy. LBP, low back pain; %MVIC, %maximal voluntary isometric contraction; BCE, broken chain exercise; CCE, connected chain exercise; HAM, hamstring; GM, gluteus maximus; Ip ES, ipsilateral erector spinae; Con ES, contralateral erector spinae; Ip TrA/IO, ipsilateral transverse abdominis/internal oblique; Con TrA/IO, contralateral transverse abdominis/internal oblique; Ip EO, ipsilateral external oblique; Con EO, contralateral external oblique. *MANOVA was significant at $p < 0.05$.

**Table 3.** Electromyography onset time data during prone hip extension (N = 40)

| Onset time | LBP Group | | Healthy Group | | p-value | | $R^2$ |
|---|---|---|---|---|---|---|---|
| | BCE | CCE | BCE | CCE | Condition main effect | Group main effect | |
| HAM | 0.46 ± 0.033 | 0.32 ± 0.010 | 0.33 ± 0.019 | 0.31 ± 0.025 | < 0.001* | 0.277 | 0.565 |
| GM | 0.52 ± 0.020 | 0.34 ± 0.017 | 0.34 ± 0.021 | 0.32 ± 0.025 | < 0.001* | 0.005* | 0.754 |
| Ip ES | 0.26 ± 0.013 | 0.49 ± 0.029 | 0.25 ± 0.026 | 0.43 ± 0.052 | < 0.001* | 0.868 | 0.905 |
| Con ES | 0.21 ± 0.011 | 0.49 ± 0.023 | 0.25 ± 0.017 | 0.45 ± 0.044 | 0.005* | 0.011* | 0.921 |

Values are presented as mean ± standard deviation. The $R^2$ value ranges from 0 to 1 with 1 being perfect predictive accuracy. LBP, low back pain; BCE, broken chain exercise; CCE, connected chain exercise; HAM, hamstring; GM, gluteus maximus; Ip ES, ipsilateral erector spinae; Con ES, contralateral erector spinae. *MANOVA was significant at $p < 0.05$.

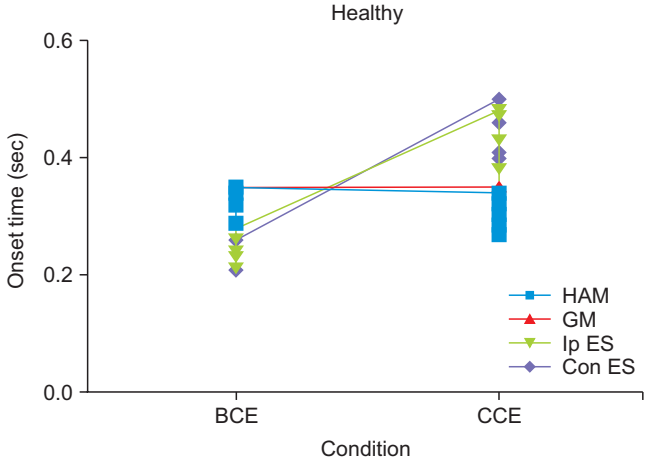


**Figure 3.** Electromyography onset time data during prone hip extension in the healthy group. BCE, broken chain exercise; CCE, connected chain exercise; HAM, hamstring; GM, gluteus maximus; Ip ES, ipsilateral erector spinae; Con ES, contralateral erector spinae.

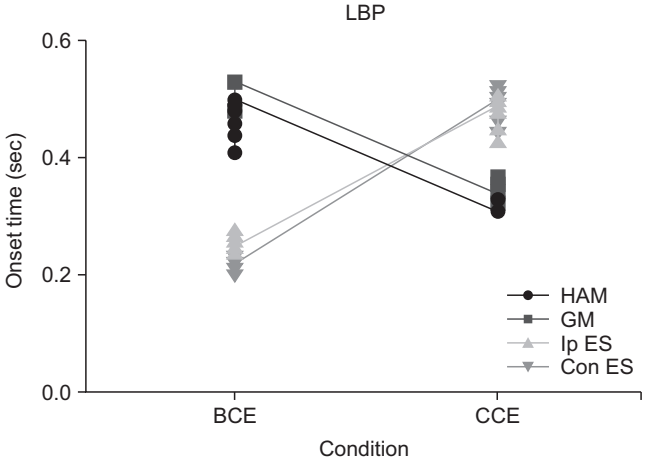


**Figure 4.** Electromyography onset time data during prone hip extension in the LBP group. LBP, low back pain; BCE, broken chain exercise; CCE, connected chain exercise; HAM, hamstring; GM, gluteus maximus; Ip ES, ipsilateral erector spinae; Con ES, contralateral erector spinae.

(Figures 3, 4).

Pearson's correlation coefficient was computed to assess the relationship between BCE and CCE on dependent variables. In most of the muscles, there was a strong, positive correlation between the two variables, and there was a significant relationship ($p < 0.001$). In both LBP and healthy participants, the EMG onset time was associated with BCE and CCE (Tables 4, 5).

## DISCUSSION

In this study, we investigated the therapeutic effects of the CCE on EMG motor control patterns in participants with and

**Table 4.** Pearson's correlation coefficient of electromyography onset time data during prone hip extension in the healthy group (N = 20)

| | HAM | | GM | | Ip ES | |
|---|---|---|---|---|---|---|
| | BCE | CCE | BCE | CCE | BCE | CCE |
| GM | 0.873*** | 0.905*** | NS | NS | NS | NS |
| Ip ES | 0.752*** | 0.420 | 0.639** | 0.333 | NS | NS |
| Con ES | 0.764*** | 0.368 | 0.863*** | 0.310 | 0.864*** | 0.870*** |

HAM, hamstring; GM, gluteus maximus; Ip ES, ipsilateral erector spinae; BCE, broken chain exercise; CCE, connected chain exercise; Con ES, contralateral erector spinae; NS, not significant. **p < 0.01. ***p < 0.001.

**Table 5.** Pearson's correlation coefficient of electromyography onset time during prone hip extension in the low back pain group (N = 20)

| | HAM | | GM | | Ip ES | |
|---|---|---|---|---|---|---|
| | BCE | CCE | BCE | CCE | BCE | CCE |
| GM | 0.789*** | 0.809*** | NS | NS | NS | NS |
| Ip ES | –0.406 | 0.753*** | –0.200 | 0.889*** | NS | NS |
| Con ES | 0.232 | 0.639*** | 0.406 | 0.922*** | 0.636** | 0.960*** |

HAM, hamstring; GM, gluteus maximus; Ip ES, ipsilateral erector spinae; BCE, broken chain exercise; CCE, connected chain exercise; Con ES, contralateral erector spinae; NS, not significant. **p < 0.01. ***p < 0.001.

without LBP during PHE. As hypothesized, CCE normalized the EMG onset time, recruitment sequence, and amplitudes in the bilateral ES, bilateral EO, bilateral IO/TrA, GM, and HAM muscles in individuals with and without LBP during PHE. Most importantly, CCE restored neuromuscular chain reconnection between the proximal core segment and distal (adjacent) hip segment, thereby improving neuromuscular coordination in terms of EMG activation onset time, recruitment sequence and amplitudes in the lumbopelvic muscles. Based on the extensive review of the literature, there has been no research concerning the effects of CCE utilizing DNS core stabilization in participants with LBP during PHE, it was difficult to compare our novel evidence with previous data.

## 1. EMG Amplitudes Between Normal and Low Back Pain Groups

Interestingly, MANOVA EMG amplitude data analyses revealed that there was no significant interaction effect for condition (BCE vs. CCE) × group (LBP vs. healthy) for all the tested muscles. However, the main effect of the conditions was significant for GM, bilateral ES, ipsilateral TrA/IO, and bilateral EO, whereas the main effect for the group was significant for HAM, GM, bilateral ES, and contralateral TrA/IO. Both healthy and LBP groups had increased activation in the ipsilateral TrA/IO and bilateral EO and GM muscles under the CCE compared with those under the BCE condition (p = 0.001, 0.015, and 0.001, respectively). HAM activation was greater in participants with LBP (3.68%) than and bilateral ES (ipsilateral 10.78%, contralateral 24.95%), and increased GM (5.21%), bilateral TrA/IO (ipsilateral 5.92%, contralateral 10.1%), and bilateral EO (ipsilateral EO 4.44%, contralateral EO 9.68%) during the BCE than the CCE (Table 2). Our findings are in line with previous EMG motor control studies. Yoon and You [25] and Lee et al. [26] demonstrated that CCE more significantly improved TrA/IO amplitude (%MVIC 11–18) than BCE in both healthy and LBP groups. Oh et al. [15] also observed decreased muscle imbalance ratio between ES and GM (1:2) and between HAM and GM (1.2:1) during the abdominal drawing-in maneuver in 20 asymptomatic adults. It is possible that CCE reconnected a broken or lose lumbopelvic chain, which in turn restored neuromuscular imbalance ratios in muscle activation amplitudes between contralateral ES and GM (BCE, 1:1.39 vs. CCE, 1:2.38 in LBP) as well as between HAM and GM (BCE, 1:1.04 vs. CCE, 1:1.15 in LBP), thereby stabilizing and equalizing the excessive spinal load during PHE movement [15]. As such, reconnected neuromuscular chain and balanced control may contribute to alleviating mechanical LBP [16,27].

## 2. Electromyography Onset Time and Sequence Between Normal and Low Back Pain Groups

MANOVA EMG onset time data analyses revealed that the main effect of the conditions was significant for all HAM, GM, and bilateral ES muscles, whereas the main effect for the group was significant only for GM and contralateral ES. Specifically, the delayed onset time was initially noted in the participants with LBP but improved during the application of the CCE.

Our findings are consistent with those of previous EMG studies [2,28]. Hodges and Richardson [2] reported that movement in each direction (shoulder flexion, abduction, and extension) resulted in contraction of trunk muscles before or shortly after the deltoid in 15 healthy adults. The TrA was invariably the first muscle to activate and was not influenced by movement direction, supporting the hypothesized role of this muscle in spinal stability generation in 15 healthy adults. Contraction of TrA was significantly delayed (5 ms) in patients with LBP with all movements. The delayed onset of contraction of TrA indicates a deficit of motor control and is hypothesized to result in inefficient muscular stabilization of the spine [2]. Tsao and Hodges [28] found that the delayed onset time of TrA was normalized during rapid shoulder flexion after the single session of isolated TrA exercise as well as sit-up exercise in participants with LBP and healthy adults. Furthermore, the earlier pre-activation of TrA training during the isolated TrA exercise more effectively produced a coordinated synergistic activation across the IO, ES, and AD compared with before training. Similarly, it is proved that such chain-connecting exercise is critical in normalizing feedforward delays of muscle activity and regaining neurophysiological control of the deep abdominal muscles during PHE or distal extremity movement [29]. Further correlation analysis was implemented to evaluate the concept of synergistic activation of LNC muscles including HAM, GM, and bilateral ES muscles. The results of Pearson's correlation coefficient demonstrated high correlations among the tested muscles during BCE in healthy participants as well as high correlations among the tested muscles during CCE in participants with LBP. Certainly, our findings support the concept of synergistic activation during anticipatory postural adjustment (APA) movement in standing, which was first investigated by Horak and Nashner [30]. Normally, APAs involve a subcortical, synergistic activation of HAM, GM, and bilateral ES in which postural core muscles stabilize the LNC against internal and external perturbation forces imposed during dynamic PHE [30-33]. Horak and Nashner [30] found that, during the first 5–20 practice trials of backward perturbation, mean EMG postural response latencies between the paraspinal and HAM muscle activation were 14 ± 13 ms, supporting the synergistic activation of the LNC muscles, which is mediated by a centrally or subcortically programed postural control mechanism. However, in LBP, the lack of synergistic activation of HAM, GM, and bilateral ES may produce an ineffective or suboptimal control of the forces associated with this postural perturbation, resulting in an inability to stabilize the spine adequately in a timely manner. Subsequently, this leads to the risk of mechanical LBP [34,35]. During the CCE, the HAM, GM, and bilateral ES muscles demonstrated more normalized synergistic activation to generate sufficient spinal stability during dynamic antigravity PHE than BCE in the LBP group, as evidenced by increased correlation among the LNC muscles (BCE, $r = -0.20$ to 0.79 vs. CCE, $r = 0.64$ to 0.96).

In addition, healthy and LBP group comparison revealed that the GM muscle onset time showed a significant difference ($p < 0.01$). This result may be explained by the fact that CCE affected the EMG onset time and sequence of the CCE group to the neutral position and normalized the alignment of the pelvis. This alignment had a positive effect on the GM muscle contraction onset time, which is the most important factor to consider during PHE exercise [36].

Taken together, our results suggest that the CCE was effective in PHE for normalizing delayed onset time and regaining neurophysiological control of the LNC muscles' synergistic activation, thereby resulting in functional spinal postural core stability improvements, which is required during dynamic PHE movements of the distal extremities in patients with LBP.

## 3. Limitations

This study has a couple of research limitations that should be considered for future studies. One limitation is that spinal stabilization-induced IAP [16,17,37], which involves a coordinated coactivation of the diaphragm, TrA-multifidus-pelvic floor, IO, and EO muscles, was not fully measured. Interestingly, Hodges and Gandevia [38] directly measured the regulation of IAP during core stabilization by inserting pressure transducers via the nose into the esophagus and stomach in vivo. Although this method is novel, it is too invasive and may pose a great risk of internal organ injury. Nevertheless, a DNS-certified physical therapist practitioner performed the CCE intervention based on the DNS treatment principles and guidelines using ultrasound imaging and surface EMG to ensure a correct activation and procedure of the CCE. Another limitation is that a careful interpretation should be made when interpreting the current data because LBP may present a variety of pathomechanics including functional impairments (e.g., muscle imbalance, inefficient muscular stabilization, or instability) and structural impairment (discogenic and degenerative stenoses) [39], which

may require a different form of interventional strategies and exercises to produce effective and sustainable results. Last, only male participants with and without LBP were included in this study because our female participants were not willing to partake in the experiment where the EMG electrode was being attached to the gluteal muscle area. Such exposure of the private area during the EMG experiment is prohibited in the current culture because it alters the existing social norms and is considered a sensitive cultural issue.

Another limitation is that target bar for PHE was set at six inches for all participants during data collection. However, differences in leg length and hip flexibility in each participants should be considered in the similar studies in the future.

## CONCLUSIONS

This clinical trial highlights the superior advantage of the CCE to produce more effective effects on EMG core muscle control in participants with and without LBP during PHE than BCE. This study provides clinical insights into therapeutic efficacy and understanding motor control mechanisms underpinning the CCE in patients with LBP with broken core chain.

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## CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

## AUTHOR CONTRIBUTIONS

Conceptualization: HP, CP, JHY. Data curation: HP, CP, JHY. Formal analysis: HP, CP, JHY. Funding acquisition: HP, CP, JHY. Investigation: HP, CP, JHY. Methodology: HP, CP, JHY. Project administration: HP, CP, JHY. Resources: HP, CP, JHY. Supervision: HP, CP, JHY. Validation: HP, CP, JHY. Visualization: HP, CP, JHY. Writing - original draft: HP, CP, JHY. Writing - review & editing: HP, CP, JHY.

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