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ORIGINAL ARTICLE

Supervised exercise therapy *versus* laser-guided exercise therapy on postural control in subjects with non-specific chronic low back pain: a randomized controlled clinical trial

Alejandro CAÑA-PINO [1], María D. APOLO-ARENAS [1] *, Pablo CARMONA DEL BARCO [2], Jesús MONTANERO-FERNÁNDEZ [3], Luís ESPEJO-ANTÚNEZ [1]

[1]Department of Medical Surgical-Therapy, Faculty of Medicine and Health Sciences, Research Group PhysioH, University of Extremadura, Badajoz, Spain; [2]School of Industrial Engineering, Service of Languages and Computer Systems, Department of Computer and Telematic Systems Engineering, University of Extremadura, Badajoz, Spain; [3]Department of Mathematics, Faculty of Medicine, University of Extremadura, Badajoz, Spain

*Corresponding author: María D. Apolo-Arenas, Department of Medical Surgical-Therapy, Faculty of Medicine and Health Sciences, Research Group PhysioH, University of Extremadura, Badajoz, Spain. E-mail: mdapolo@unex.es

ABSTRACT

BACKGROUND: Among the most effective therapeutic interventions in non-specific chronic low back pain, clinical practice guidelines highlight exercise therapy and patient education. However, the variability in the type of exercise and its dosage means that there is no clear evidence regarding the most optimal form of therapeutic exercise.
AIM: The main objective of this study was to ascertain the effects produced by two different exercise interventions (supervised exercise therapy and laser-guided exercise therapy) and pain neuroscience education on postural control measured by the displacement center of pressure (CoP) and energy spectral density (ESD) in subjects with non-specific chronic low back pain.
DESIGN: This is a single-blinded randomized clinical comparative controlled trial.
SETTING: The study was carried out in different private physiotherapy care centers.
POPULATION: We enrolled 60 subjects with non-specific chronic low back pain of at least 3-month duration, aged 18-45 years.
METHODS: Both groups performed a total of 16 therapeutic exercise sessions and 8 pain neuroscience education sessions, with the laser-guided exercise therapy group performing laser-guided exercises. The main outcome measures evaluated were ESD and displacement of CoP measured at 3 different times (baseline, post-treatment, and 3 month follow-up).
RESULTS: The most important differences for ESD and displacement of CoP variables were obtained for eyes open, unstable surface anteroposterior axis ($F(2,92)=7.36$, $P=0.001$, $d=0.71$) and eyes closed, stable surface mediolateral axis ($F(2,92)=3.24$, $P<0.001$, $d=0.76$). Further, time × group interactions showed significant statistical differences in both cases as well as significant differences between baseline and 3 month's follow-up.
CONCLUSIONS: Both exercise modalities (supervised exercise therapy and laser-guided exercise therapy) showed changes in variables related to postural control (displacement of CoP and ESD). However, the laser-guided exercise therapy program showed greater improvements in ESD.
CLINICAL REHABILITATION IMPACT: Analysis of a new approach for the quantification of data obtained from postural control assessment relying on widely used devices (accelerometers and pressure platforms).

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KEY WORDS: Low back pain; Exercise therapy; Pain; Neurosciences.

Non-specific chronic low back pain (NSCLBP) is the most common musculoskeletal condition being the main cause of disability.[1] The prevalence rate in young adults under 45 years of age is high (around 70-80%).[2] Negatively affecting their quality of life and that of the people around them.[1] In addition, NSCLBP has a large economic impact as it is related to job performance, absentism, disability and consumption of health care resources.[1, 3] Further on, among the most effective therapeutic interventions in NSCLBP, clinical practice guidelines highlight exercise therapy and patient education.[4, 5] However, the variability in the type of exercise and its dosage means that there is no clear evidence regarding the most optimal form of therapeutic exercise for the target population. Equally, pain neuroscience education (PNE) is effective and aims to help patients understand more about their pain from a biological and physiological perspective for subjects with persistent pain as NSCLBP. Although, there is heterogeneity depending on who provides it.[3, 6-8] On this line, Louw *et al.*[7] showed that for musculoskeletal pain, PNE provides compelling evidence of reductions in pain, disability, pain catastrophizing, and also physical movement improvement. On the other hand, in a recent meta-analysis, it was stated that exercise therapies such as pilates, resistance exercise, motor stabilisation/motor control exercise and aerobic exercise training, prescribed and supervised by a professional, being actively encouraged to move and exercise progressively, are the most effective.[9] Within the types of therapeutic exercise, recent systematic reviews support the effectiveness of motor control exercise in patients with NSCLBP.[4, 5] Moreover, motor control exercise programmes have shown an effect on the precision with which the movement is executed. In this sense, differences may exist when the prescribed exercise requires greater attention to the body movements performed. Internal focus (*e.g.* trunk movement) *versus* when the focus is on the effect the movement has on the environment (external focus, *e.g.*, touching objects).[10] Besides, these differences have been observed in postural control mechanisms and motor skill learning. With the derived effects being greater when exercise therapy was guided.[11, 12] On this line, Lopes *et al.*[13] obtained improvements in the displacement of centre of pressure (CoP) after applying specific spinal stabilisation exercises in subjects with NSLBP. Also, Ghasemi *et al.*[14] obtained a decrease in displacement of CoP when combining therapeutic exercise with manual therapy. In view of this, new devices have emerged, such as wireless inertial motion sensors,[15] stabilizers,[16] metronomes[10] surface electromyography (sEMG)[17] or laser-guided exercise therapy (LGET)[18, 19] which provides external feedback to exercise, achieving an improvement in range of motion and postural control in subjects with NSCLBP.[20, 21] In this regard, the LGET is a procedure applied for proprioceptive training which by means of an implement consisting of a laser pointer placed on the head/trunk/abdomen of the subject provides information on the degree of joint repositioning.[22] Also, variations in the proprioceptive system through unstable surface situations may make the postural control of subjects with NSCLBP dependent on visual information and therefore visual feedback in these patients may be relevant.[16] Abdollahipour *et al.* and Chiviacowsky *et al.* indicated that these differences might be caused by the external focus promoting a kind of automatic control allowing unconscious and rapid control processes.[11, 23] In addition, it favors goal-action coupling. Diverting concentration from oneself to the goal of the task. Abdollahipour *et al.* claim that therapeutic exercise using an external approach facilitates the establishment of effective neural connections that optimise exercise performance.[11] Although therapeutic exercise with an external focus is widely used in NSCLBP subjects, there is limited knowledge about their influence on parameters that are sensitive to change in this population, such as energy spectral density (ESD),[24] displacement of CoP,[24, 25] and CoP velocity.[26] Furthermore, to the best of the authors' knowledge, there are also no studies that simultaneously use laser as an external focus device and exercise in subjects with NSCLBP. The LGET has been shown to be effective in cervical pain,[18, 19] although studies are limited in NSCLBP. Since the most appropriate type of approach to therapeutic exercise has not yet been proven (supervised guided exercise therapy *vs.* external focus guided therapeutic exercise), the aim of this study was to to ascertain the effects produced after the application of two different exercise modalities (guided exercise therapy [SET] and laser-guided exercise therapy [LGET]) on PNE and postural control measured by the displacement of CoP and ESD in subjects with NSCLBP.

Materials and methods

Single-blinded randomized comparative clinical trial carried out in compliance with the recommendations of the CONSORT statements[27] conducted following the Declaration of Helsinki, and approved by the Ethical Research Committee of the University of Extremadura (project code 77//2018, approval date: 6/7/2018) and was registered at ClinicalTrials.gov with registration number NCT03635242.

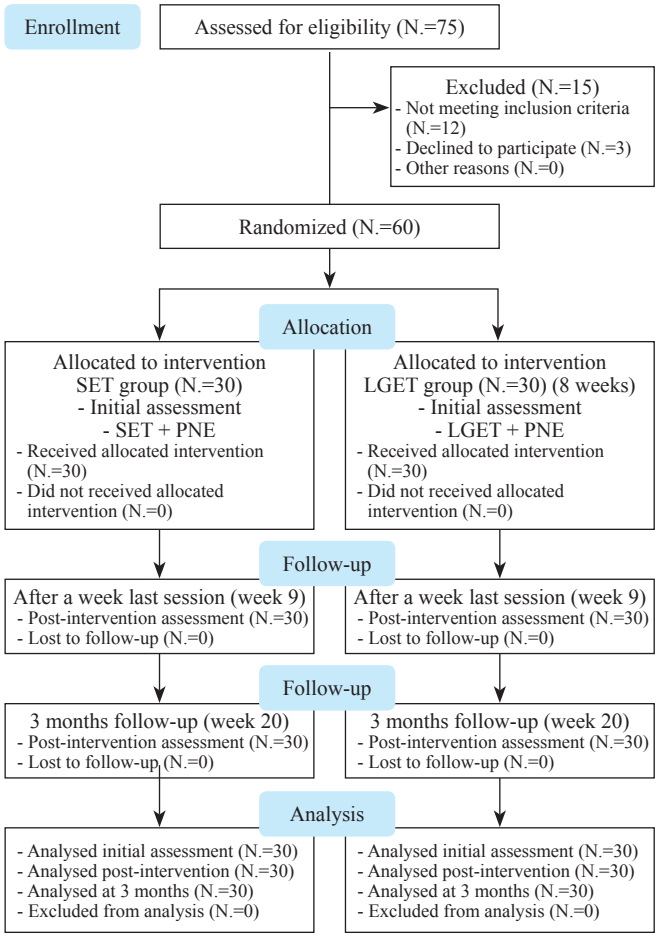

Figure 1.—Flowchart of the study.

Settings and participants

The potential sample was comprised of seventy patients with NSCLBP (Figure 1). The inclusion criteria were: 1) aged between 18-45 years;[28] 2) experiencing NSCLBP for ≥3 months[29, 30] (diagnosed by a physician) and having Spanish as their native language; 3) patients suffering from pain between the costal margins and the inferior gluteal folds with or without referred pain to the leg[31] were included, provided that they scored at least 3/10 on the Numerical Pain Rating Scale – subjective measure in which individuals rate their pain on an 10-point numerical scale ranging from 0 ("no pain at all") to 10 ("worst imaginable pain");[32] and 4) patients with NSCLBP were allowed to have referred pain in the leg above the knee as long as no neurological symptoms were present.[33] The exclusion criteria were:[29] 1) pregnancy, including 6 months postpartum; 2) chronic fatigue syndrome, fibromyalgia, complex regional pain syndrome; 3) history of back or lower limb surgery; 4) signs of neuropathic pain (*e.g.*, a painful radiculopathy[33]); 5) trauma to the back or lower extremities in the last 3 months; 6) metal spine implants; 7) neurological or vestibular disorders; 8) the consumption of analgesics 24 hours before each assessment; 9) a diagnosed psychiatric disorder or severe cognitive impairment that prevented the PNE program from being followed (in case of doubt, the mini mental test was performed with a minimum score of 25[34]); 10) a physical condition that prevented the completion of the PNE program (minimum requirement was execution in a normal time [<10″] of the timed "up and go" test[34]); and 11) patients with associated pathologies that made it impossible to perform the PNE program (myopathies and neurological disorders), and treatment with alternative therapies[2, 34] (Figure 1). Participants were asked to avoid any medication or physiotherapy treatment in the last 24 hours before the assessments, they were also informed about the nature of the study, and written informed consent was obtained from all participants prior to the first assessment. From the initial 70 patients, 60 met the inclusion criteria. Afterwards, the sample was randomly divided in 2 groups: supervised exercise therapy (SET) group, formed by 30 subjects with NSCLBP of at least 3-months duration who performed a SET and PNE intervention; and the laser-guided exercise therapy (LGET) group, formed by 30 subjects with NSCLBP of at least 3-months duration, who performed a LGET and PNE intervention (Table I).

Sample size calculation

A convenient sample of at least 30 participants per group was envisaged. This sample size was considered to be sufficient to detect an effect size of about δ=0.50 given a 2-sided level 5% paired sampled *t*-test and a statistical power of 80% as it was calculated using Jamovi 1.6 computer software, the Jamovi project (2020).[35]

TABLE I.—*Sociodemographic and clinical variables.*

Parameters	G1 (N.=30) Mean±SD	G2 (*N.*=30) Mean±SD	P value*
Mean age (years)	35.30±7.10	32.00±6.78	0.052
Height (cm)	171.30±0.08	170.23±0.10	0.544
Weight (kg)	71.77±10.11	69.50±11.42	0.668
Body mass index (kg/m²)	24.44±3.30	23.92±3.10	0.626
Pain (Numerical Pain Rating Scale)	7.38±1.18	6.97±1.05	0.095
Average duration of pain (months)	37.83±39.82	35.73±30.75	0.361
Disability (Roland-Morris)	9.80±4.72	9.08±5.77	0.289

cm: centimeters; kg: kilograms; m: meters; SD: standard deviation; G1: supervised exercise therapy; G2: laser-guided exercise therapy.
*P<0.05: statistically significant.

Randomization

Each patient evaluated was assigned a numeric code. The randomization process was performed by simulating a continuous uniform distribution by IBM SPSS 22 (IBM, Chicago, IL, USA). Then, patients were sorted according to their values so that the first 30 were assigned to SET and the rest of them to LGET.

Interventions

Two experienced independent physiotherapists who assessed the suitability of each participant based on the inclusion and exclusion criteria examined participants at baseline. However, these researchers were not involved in the interventions. The interventions were carried out by a different physiotherapist with more than 7 years of experience in exercise therapy and chronic pain. The interventions were carried out in compliance with the recommendations of the CERT[36] and TIDIER[37] statements.

Supervised exercise therapy group

Patients assigned to this group executed a therapeutic exercise program at the Faculty of Medicine and Health Sciences (Badajoz, Spain) with a total of 16 sessions (16 hours) with a frequency of 2 sessions/week for 8 weeks. In addition, they carried out a PNE program with a total of 8 sessions (8 hours), with a frequency of 1 session/week for 8 weeks.[6, 38] The program was performed in the order shown in Figure 2 and Supplementary Digital Material 1 (Supplementary Text File 1). On this line, the therapeutic exercise program was delivered in groups of 5 subjects. Where exercises progressed from supine to standing position, 4-point kneeling or sitting according to each patient's exercise tolerance. Additionally, during the sessions, reference was made to the theoretical contents learned in the PNE program. The fact that the intervention was in a group was not an obstacle for each patient to receive individualized indications on how to perform or adapt the intervention to their conditions. Seeing that patients with NSCLBP may have ineffectiveness of descending inhibitory pain pathways, the first sessions could lead to an increase in symptomatology. Therefore, patients were warned of this possibility and at no time was the onset of pain reason to stop the activity.[34] Besides, the PNE program was delivered at the same location as the exercise therapy program by the same physiotherapist. Sessions consisted of a verbal explanation with a visual presentation about aspects related to pain (acute *vs.* chronic pain, central sensitization, etc.). In addition, the content of the PNE program included concepts of the neurophysiology of pain. The patient receives PNE comparable to explain pain, adapted to the predominant pain mechanism(s) and contributing factors, using the following mode of administration:[39] 1) anatomical explanation of the main stabilising muscles of the lumbar spine (weeks 1 and 2); 2) audiovisual material through oral explanations (weeks 3 and 4); 3) written educational material (weeks 5 and 6); and 4) playful sessions (weeks 7 and 8).

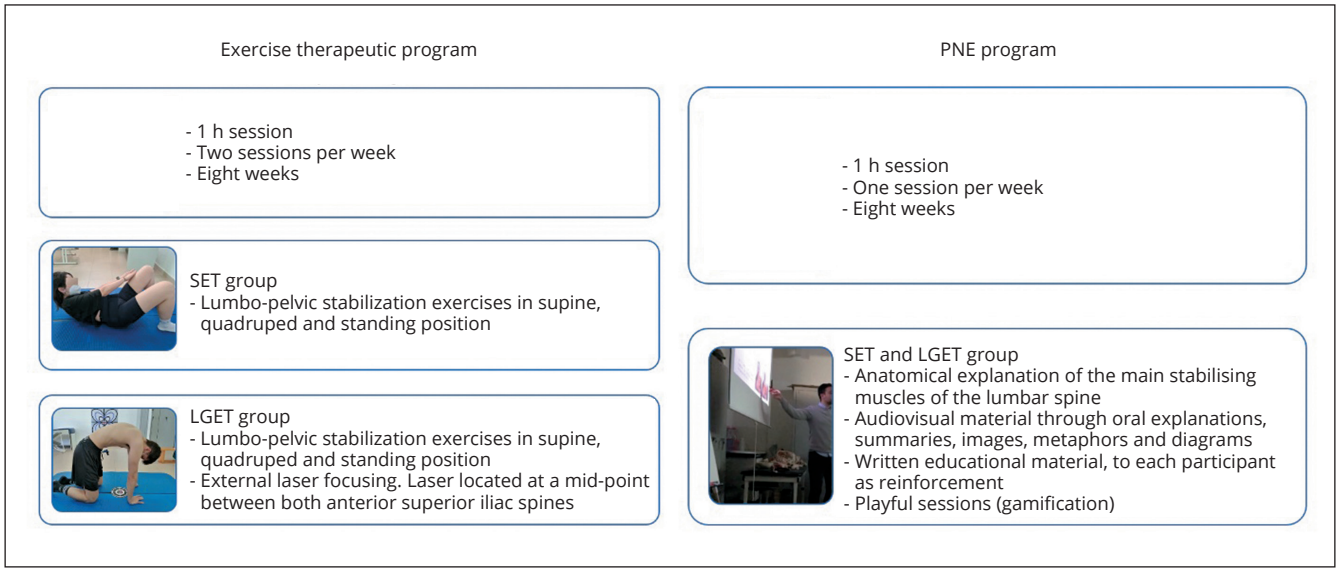

Figure 2.—Distribution of the program followed by SET and LGET group.

Laser-guided exercise therapy group

The LGET group carried out the same intervention procedure as the SET group described in point above: supervised exercise therapy (SET) group. The difference was that the LGET performed the laser-guided exercise therapy program (Supplementary Digital Material 2: Supplementary Table I), using the external focus (motion guidance system). The PNE program was the same.

Procedure of assessment

Postural control was assessed in all participants. The outcome assessor did not know the group to which each subject was allocated. Study participants were evaluated before the intervention (week 0), after the intervention (week 9), and at 3 months follow-up (week 20). Moreover, the procedure for measuring postural control was assessed by means of a pressure platform (Podoprint; Namrol, Barcelona, Spain) and triaxial accelerometer (Shimmer; Shimmer, Dublin, Ireland) similar to that used on previous studies.[24, 40-43] Each participant completed the postural task under four different consecutive conditions of increasing difficulty, in the following order without randomization:[43] 1) eyes open, stable surface (EOSS); 2) eyes closed, stable surface (ECSS); 3) eyes open, unstable surface (EOUS); and 4) eyes closed, unstable surface (ECUS). Participants stood in a neutral upright stance with both feet on the platform, arms straight to their sides, feet rotated externally relative to the line of progression and eyes on a fixed point at eye level. The accelerometer was placed on L3-L4.[24, 40] A 10-cm thick foam rubber pad (TheraBand, Akron, OH, USA) was used to provide an unstable surface.[40, 44] Frequency of data collection was set at 100 Hz for the pressure platform and 50 Hz for the accelerometer. The time for each test was 30 seconds.[24, 40]

Sociodemographic and clinical variables

Several measurements were collected for sample characterization. First, we asked about age, time since the onset of low back pain, and its intensity through the Numerical Pain Rating Scale. Secondly, bodyweight (kg) and height of participants without shoes (cm) was measured. Body mass index (BMI) was calculated according to this formula: BMI = weight (kg)/height^2 (cm^2). In addition, the following variables were registered: "pain intensity" using the numerical pain rating scale,[32] and disability, assessed through the Roland-Morris Disability Questionnaire (RMDQ).[45, 46]

Outcome variables

The main outcome measures were the differences between groups in the accelerometry (ESD) and pressure platform (displacement of CoP) variables at different times (baseline, post-treatment, and 3-month follow-up). The ESD (Joules/MHz), could be defined as the energy required by the subject to restore balance after a perturbation. Further, it is assumed that a lower ESD indicates better postural control.[24, 40] In addition, the reliability of procedures used with the technological devices was previously tested. The analysis for the pressure platform and accelerometer showed moderate-high reliability for the intraclass correlation coefficient (ICC) being the smallest value for the variable EOSS mediolateral (ML) axis =0.799 for the pressure platform and EOUS anteroposterior (Z) axis =0.506 for the accelerometry. The ICC was used to calculate the standard error of measurement (SEM). With the highest SEM value being ECUS mediolateral (ML) axis =0.74 for pressure platform and ECUS mediolateral (Y) axis =0.82 for accelerometry.[24, 40]

Statistical analysis

Data analysis was performed using SPSS version 22.0. (SPSS Inc., Chicago, IL, USA) and jamovi 1.8.4 (Jamovi project). A descriptive analysis was performed for each of the variables. Due to the weak skewness of most variables and the sample size, parametric type tests were applied. Data were reported as mean±SD. Demographic and clinical variables of the groups at baseline were compared by independent samples *t*-test. Differences between three stages were also analysed, comparing both experimental groups throughout independent samples *t*-test. Intra-group differences were analysed by paired samples *t*-test. The effect size was calculated using a Cohen's *d* coefficient. Wilcoxon Test was applied when a considerable skewness was observed. In order to avoid the increase of type I error probability due to this procedure, we also applied multivariate comparations between groups, considering jointly each kind of clinical outcome (ESD and displacement of CoP). This study guided us to focus on the most important outcomes between ESD and displacement of CoP variables. These outcomes were separately analyzed by means of a repeated measure analysis of variance (with Greenhouse-Geisser Correction) in order to compare groups SET and LGET along the treatment and controlling baseline pain and disability as potential confounding variables. Simple correlations between these outcomes and ESD and displacement of CoP variables were analyzed at each stage by Pearson's Test. A significance level of p under 0.05 was considered.

Results

Description of the sample

The final sample was 60 patients (N.=60), 30 males and 30 females; the mean age for the SET group was 35.30±7.10 and for the LGET group 32.00±6.78. Table I shows mean values and standard deviation of the main clinical variables for each group (SET and LGET) and their statistical significance (P>0.05). There were no significant baseline differences between groups.

Analysis of outcome measures about ESD and CoP within-groups

Table II includes baseline, after intervention, and follow-up values of the study variables and differences between before and after treatment measures and within-groups

TABLE II.—*Outcome measures about CoP and ESD within-groups.*

Parameters	Group G1 (N.=30) G2 (N.=30)	Baseline mean±SD	Post-treatment mean±SD	3-m follow-up mean±SD	Differences within-groups (baseline-post-treatment) 95% CI	Differences within-groups (baseline-3 month follow-up) 95% CI	Differences within-groups (post-3 month follow-up) 95% CI	Baseline posteffect size (*d*)
CoP (EOSS ML axis) (mm)	G1	5.54±2.51	3.88±2.38	4.17±2.22	1.66 (1.14. 2.18)**	1.37 (0.73, 2.01)**	0.29 (-0.77, 0.19)	1.18
	G2	6.13±2.95	4.05±1.69	4.33±1.68	2.08 (0.97, 3.20)**	1.82 (0.40, 3.24)*	0.42 (-0.94, 0.92)	0.70
CoP (EOSS AP axis) (mm)	G1	7.89±4.40	4.67±2.53	5.40±1.92	1.60 (-0.21, 3.23)**	2.49 (0.63, 4.35)*	0.73 (-1.83, 0.37)*	0.77
	G2#	10.73±11.44	5.99±2.31	5.70±1.95	4.75 (0.71, 8.78)*	5.25 (0.17, 10.34)*	0.27 (-0.70, 1.25)	0.44
CoP (ECSS ML axis) (mm)	G1	6.27±3.64	4.57±2.43	4.93±2.35	4.12 (2.71, 5.53)*	1.35 (0.60, 2.10)*	0.36 (-1.06, 0.34)	0.67
	G2	7.17±2.60	5.18±2.15	4.42±1.93	1.98 (1.00, 2.96)**	2.93 (1.95, 3.92)**	0.58 (-0.13, 1.28)	0.76
CoP (ECSS AP axis) (mm)	G1	8.69±4.03	5.54±2.50	5.50±1.96	3.15 (1.99, 4.32)**	3.19 (1.89, 4.48)**	0.03 (-0.44, 0.50)	1.01
	G2	9.57±1.02	6.18±1.82	5.67±1.89	3.39 (2.70, 4.09)**	3.93 (3.06, 4.80)**	0.51 (-0.58, 1.59)	1.83
CoP (EOUS ML axis) (mm)	G1	7.83±4.45	5.91±2.95	5.94±1.98	1.92 (0.91, 2.92)**	1.89 (0.51, 3.27)*	0.03 (-0.87, 0.82)	0.71
	G2	6.76±2.74	4.89±2.70	5.06±2.21	1.87 (0.71, 3.04)*	1.63 (0.68, 2.59)*	0.38 (-1.38, 0.63)	0.60
CoP (EOUS AP axis) (mm)	G1	9.41±4.61	5.55±2.63	6.55±2.52	3.86 (1.97, 5.76)**	2.86 (0.82, 4.90)*	1.00 (-1.78, -0.23)*	0.76
	G2	9.32±5.22	7.00±2.64	7.51±2.01	2.32 (0.30, 4.35)*	3.58 (-12.08, 4.93)	5.73 (-12.20, 2.73)	0.43
CoP (ECUS ML axis) (mm)	G1	7.43±4.53	5.63±3.22	6.08±2.67	1.80 (0.92, 2.68)**	1.35 (0.31, 2.39)*	0.45 (-1.11, 0.20)	0.76
	G2	6.66±2.72	5.18±2.02	5.42±2.43	1.48 (0.40, 2.55)*	1.23 (0.24, 2.20)*	0.28 (-1.18, 0,63)	0.51
CoP (ECUS AP axis) (mm)	G1	9.70±4.80	7.02±3.54	7.52±2.04	2.69 (0.98, 4.39)**	2.19 (0.52, 3.85)*	0.50 (-1.52, 0.52)	0.59
	G2	10.32±4.25	6.70±2.11	6.29±1.90	3.62 (0.69, 2.21)**	3.47 (2.01, 4.92)**	0.21 (-0.44, 0.86)	0.96
ESD (EOSS X axis) (J/MHz)	G1	1.11±7.19	1.08±5.63	0.92±0.72	0.03 (-0.22, 0.29)	0.19 (-0.12, 0.49)	0.16 (-0.11, 0.42)	0.05
	G2	1.27±6.92	0.95±0.59	0.93±0.74	0.32 (0.09, 0.56)*	0.49 (0.10, 0.87)*	0.12 (-0.16, 0.40)	0.51
ESD (EOSS Y axis) (J/MHz)	G1	0.97±0.49	0.99±0.40	0.92±0.70	-0.02 (-0.18, 0.13)	0.05 (-0.24, 0.34)	0.07 (-0.13, 0.28)	-0.06
	G2	1.06±0.70	0.92±0.54	0.84±0.69	0.15 (-0.13, 0.43)	0.29 (-0.11, 0.69)	0.12 (-0.09, 0.34)	0.20
ESD (EOSS Z axis) (J/MHz)	G1#	2.03±1.64	1.96±1.13	1.72±1.24	0.06 (-0.55, 0.69)	0.31 (-0.33, 0.96)	0.24 (-0.22, 0.71)	0.04
	G2	2.22±1.36	1.70±0.97	1.60±1.32	0.52 (-0.05, 1.09)	0.83 (0.08, 1.59)*	0.24 (-0.06, 0.53)	0.34
ESD (ECSS X axis) (J/MHz)	G1	1.28±0.68	1.19±0.72	1.04±0.88	0.09 (-0.13, 0.31)	0.24 (-0.01, 0.58)	0.15 (-0.14, 0.43)	0.15
	G2	1.31±0.78	1.04±0.70	0.96±0.84	0.26 (0.04, 0.47)*	0.49 (0.08, 0.91)*	0.21 (-0.14, 0.57)	0.45
ESD (ECSS Y axis) (J/MHz)	G1	0.99±0.48	0.99±0.48	0.85±0.64	-0.01 (-0.14, 0.13)	0.14 (-0.01, 0.38)	0.15 (-0.05, 0.35)	-0.03
	G2	1.23±0.91	0.98±0.56	0.92±0.84	0.26 (-0.06, 0.55)	0.44 (-0.01, 0.90)	0.15 (-0.15, 0.45)	0.30
ESD (ECSS Z axis) (J/MHz)	G1#	2.32±2.02	1.86±1.27	1.50±1.31	0.45 (-0.27, 1.18)	0.82 (0.02, 0.16)	0.37 (0.08, 0.66)	0.23
	G2	2.60±1.67	1.56±0.83	1.20±0.70	1.04 (0.44, 1.64)*	0.17 (0.97, 0.24)**	0.49 (0.10, 0.87)	0.65
ESD (EOUS X axis) (J/MHz)	G1	1.09±0.56	1.16±0.57	0.90±0.68	-0.07 (-0.24, 0.91)	0.19 (-0.67, 0.44)	0.26 (0.03, 0.49)	-0.17
	G2	1.27±0.81	0.94±0.59	0.86±0.62	0.32 (0.03, 0.61)	0.56 (0.15, 0.97)*	0.19 (-0.07, 0.45)	0.42
ESD (EOUS Y axis) (J/MHz)	G1	0.95±0.48	1.04±0.54	0.90±0.64	-0.09 (-0.28, 0.11)	0.05 (-0.21, 0.32)	0.14 (-0.12, 0.40)	-0.16
	G2	1.27±0.90	0.98±0.78	0.96±0.88	0.29 (0.02, 0.56)*	0.45 (0.07, 0.84)*	0.11 (-0.20, 0.43)	0.40
ESD (EOUS Z axis) (J/MHz)	G1	2.00±1.07	2.20±1.46	1.86±1.84	-0.21 (-0.72, 0.29)	0.14 (-0.57, 0.85)	0.35 (-0.10, 0.80)	-0.06
	G2	2.69±1.95	1.63±1.08	1.46±1.37	1.06 (0.26, 1.85)	0.15 (0.52, 0.25)	0.34 (-0.05, 0.74)	0.50
ESD (ECUS X axis)	G1	1.09±0.62	1.13±0.75	1.03±0.94	-0.04 (-0.29, 0.20)	0.06 (-0.22, 0.34)	0.11 (-0.16, 0.37)	-0.07
	G2	1.21±0.82	0.93±0.59	0.76±0.57	0.28 (-0.05, 0.62)	0.60 (0.20, 0.99)*	0.27 (0.02, 0.53)	0.31
ESD (ECUS Y axis) (J/MHz)	G1	1.01±0.45	0.93±0.50	0.71±0.50	0.09 (-0.06, 0.23)	0.31 (0.15, 0.47)**	0.22 (0.05, 0.39)	0.23
	G2	1.38±0.89	0.88±0.59	0.71±0.56	0.51 (0.21, 0.80)*	0.84 (0.49, 0.12)**	0.24 (0.01, 0.48)	0.64
ESD (ECUS Z axis) (J/MHz)	G1	2.12±1.22	2.31±1.65	1.87±1.27	-0.19 (-0.72, 0.34)	0.25 (-0.31, 0.81)	0.44 (-0.22, 0.11)	-0.13
	G2	2.83±2.13	1.79±0.74	1.51±1.06	1.04 (0.28, 1.81)*	1.65 (0.71, 2.60)*	0.39 (0.01, 0.77)*	0.71

* and ** indicate significant intragroup differences between baseline post-treatment and between 3-month follow-up post-treatment and baseline (P<0.05 and P<0.001, respectively); #Wilcoxon's Test has been carried out instead *t*-test due to big skewness.
EOSS: eyes open stable surface; ECSS: eyes closed stable surface; EOUS: eyes open unstable surface; ECUS: eyes closed unstable surface; X: vertical axis; Y: mediolateral axis; Z: anteroposterior axis; ML: mediolateral axis; AP: anteroposterior axis; SD: standard deviation; CI: confidence interval; G1:supervised exercise therapy; G2: laser-guided exercise therapy.

TABLE III.—*Outcome measures about CoP and ESD between groups.*

Parameters	Differences between groups (baseline-post treatment) (G1-G2) 95% CI	Differences between groups (baseline 3-m follow-up) (G1-G2) 95% CI	Differences between groups (post-treatment 3-m follow-up) (G1-G2) 95% CI
CoP (EOSS ML axis) (mm)	0.42 (-1.63, 0.78)	0.45 (-1.90, 1.00)	0.13 (-0.56, 0.82)
CoP (EOSS AP axis) (mm)	1.53 (-5.76, 2.70)	2.77 (-7.77, 2.23)	1.00 (-2.46, 0.45)
CoP (ECSS ML axis) (mm)	0.28 (-1.62, 1.07)	1.59 (-2.78, -0.40)*	0.94 (-1.91, 0.04)
CoP (ECSS AP axis) (mm)	0.24 (-1,57, -1.09)	0.74 (-2.32, -0.83)	0.47 (-1.57, 0.62)
CoP (EOUS ML axis) (mm)	0.04 (-1.46, 1.55)	0.26 (-1.43, 1.95)	0.35 (-0.92, 1.62)
CoP (EOUS AP axis) (mm)	1.54 (-1.17, 4,25)	6.44 (-1.56, 14.43)	4.73 (-2.98, 12.43)
CoP (ECUS ML axis) (mm)	0.33 (-1.04, 1.69)	0.12 (-1.28, 1.53)	0.18 (-1.25, 0.89)
CoP (ECUS AP axis) (mm)	0.94 (-3.10, 1.23)	1.29 (-3.48, 0.91)	0.71 (-1.94, 0.52)
ESD (EOSS X axis) (J/MHz)	0.29 (-0.63, 0.05)	0.30 (-0.77, 0.17)	0.04 (-0.34, 0.41)
ESD (EOSS Y axis) (J/MHz)	0.17 (-0.48, 0.14)	0.24 (-0.72, 0.23)	0.05 (-0.34, 0.25)
ESD (EOSS Z axis) (J/MHz)	0.45 (-1.28, 0.37)	0.52 (-1.49, 0.44)	0.01 (-0.56, 0.57)
ESD (ECSS X axis) (J/MHz)	0.17 (-0.47, 0.14)	0.26 (-0.78, 0.27)	0.06 (-0.50, 0.38)
ESD (ECSS Y axis) (J/MHz)	0.25 (-0.58, 0.08)	0.30 (-0.78, 0.18)	0.01 (-0.35, 0.34)
ESD (ECSS Z axis) (J/MHz)	0.59 (-1.50, 0.33)	0.86 (-1.9, 0.20)	0.12 (-0.59, 0.34)
ESD (EOUS X axis) (J/MHz)	0.39 (-0.72, -0.07)*	0.37 (-0.83, 0.09)	0.07 (-0.27, 0.41)
ESD (EOUS Y axis) (J/MHz)	0.38 (-0.70, -0.05)*	0.40 (-0.85, 0.04)	0.03 (-0.37, 0.43)
ESD (EOUS Z axis) (J/MHz)	1.27 (-2.2, -0.35)*	1.36 (-2.53, -0.20)*	0.01 (-0.59, 0.60)
ESD (ECUS X axis) (J/MHz)	0.33 (-0.73, 0.08)	0.54 (-1.00, -0.08)*	0.17 (-0.53, 0.19)
ESD (ECUS Y axis) (J/MHz)	0.42 (-0.74, -0.10)*	0.53 (-0.90, -0.17)*	0.03 (-0.31, 0.25)
ESD (ECUS Z axis) (J/MHz)	1.23 (-2.15, -0.32)*	1.40 (-2.44, -0.37)*	0.05 (-0.72, 0.82)

*Significant intergroup differences between baseline post-treatment and between 3-m follow-up post-treatment and baseline (P<0.05).
EOSS: eyes open stable surface; ECSS: eyes closed stable surface; EOUS: eyes open unstable surface; ECUS: eyes closed unstable surface; X: vertical axis; Y: mediolateral axis; Z: anteroposterior axis; ML: mediolateral axis; AP: anteroposterior axis; CI: confidence interval; G1:supervised exercise therapy; G2: laser-guided exercise therapy.

follow-up. The SET group showed statistically significant differences before and after the intervention for all displacement of CoP variables (P<0.05) (Table II). However, no statistically significant differences were found for any of the ESD variables (P>0.05). In the LGET group, within-group analyses showed statistically significant differences before and after the intervention for all displacement of CoP variables (P<0.05) and for the variables ESD EOSS X axis (*d*=0.51), ESD ECSS X axis (*d*=0.45); ESD ECSS Z axis (*d*=0.65), ESD EOUS Y axis (*d*=0.40), ESD ECUS Y axis (*d*=0.64) and ESD ECUS Z axis (*d*=0.71) (Table II).

Analysis of outcome measures about ESD and CoP between groups

Table III includes the differences between measures before and after treatment and follow-up between groups. Differences between both groups taking baseline values jointly for ESD were analysed by one-way multivariate analysis of variance (manova) with significant results (F(12,47)=3.03, P=0.003). In fact, it can be checked in Table II that LGET showed worse values in all ESD measures (which is not surprising since they are highly correlated[24]). Multivariate differences for displacement of CoP did not turn out to be significant (F(8,51)=1.786, P=0.102). Therefore, both groups can be considered homogeneous in this sense. Nevertheless, further comparisons for both ESD and displacement of CoP were made based on stage differences. In this sense, and considering the differences between baseline and post intervention, manova gave a significant result (F(12,47)=2.16, P=0.031). Wich means that evolutions in ESD were different for each experimental group. We can see on Table III the variables that gave significant differences. Namely, ESD EOUS X axis (P=0.018, *d*=0.63), ESD EOUS Y axis (P=0.025, *d*=0.59), ESD EOUS Z axis (P=0.008, *d*=0.71), ESD ECUS Y axis (P=0.012, *d*=0.67) and ESD ECUS Z axis (P=0.009, *d*=0.70). Comparisons between evolution in displacement did not turn out to be significant acoording to manova (F(8,51)=1.084 P=0.389). Also, no multivariate differences between groups were found when we considered assessment in ESD from just after intervention and follow-up (F(12,47)=0.791, P=0.657). Nevertheless, when considering the displacement of CoP parameter, the result was significant (F(8,51)=2.46, P=0.026). Although, no single parameters turned significant. Taking into account the previous analysis (Table I, II) and multivariate analysis, study was focused on ESD ECUS Z and CoP ECSS ML as ESD as displacement of CoP main variables, respectively. Each

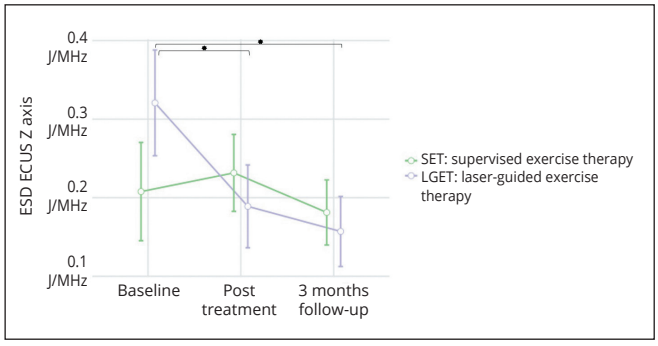

Figure 3.—Comparison between groups for the ESD ECUS Z axis variable controlling for EVA and Roland-Morris.
*Statistically significant.

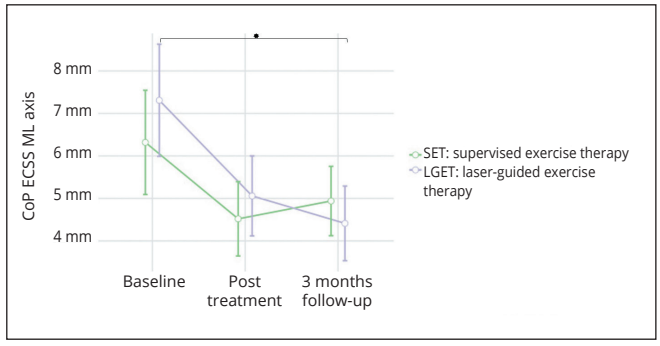

Figure 4.—Comparison between groups for the CoP ECSS ML axis variable controlling for EVA and Roland-Morris.
*Statistically significant.

measure was analysed separately throughout a repeated measures model. Being considered as an intra-subject factor measured at the three stages (pre, post and follow-up), the treatment (SET or LGET) as intergroup factor and, as clincal covariates, baseline pain and disablity. As a result, interaction time × treatment was significant ($F(1.8,91.7)=7.36$, $P=0.004$), which means that evolution in ESD depended on treatment. In fact, according to Tukey's HSD *post-hoc* comparisons, while there was no significant difference from pre to post for the SET group ($P=0.978$). The difference was significant for the LGET group ($P=0.006$). This can be checked in Figure 3. The same analysis for CoP ECSS ML also showed interaction ($F(1.7,90.7)=3.24$, $P=0.050$) in this sense we can check in Table III and Figure 4.

Correlations between clinical variables outcome variables

A correlation analysis between clinical (pain and disability) and ESD-CoP outcomes has been carried out. Taking into account pain variables, we can observe that when displacement of CoP decreases, pain postintervention (EOUS ML axis [R=0.479]); ECUS AP axis [R=0.374]) and 3 month's follow-up (ECUS AP axis [R=0.310]) also decreases in a statistically significant way ($P<0.01$). On the other hand, taking into account the disability variable, we can observe that when displacement of CoP decreases, disability postintervention (EOSS AP axis [R=0.334]) and 3 month's follow-up (EOUS AP axis [R=0.383]) also decreases in a statistically significant way ($P<0.01$). There is also a statistically significant positive correlation ($P<0.01$) when ESD (ECUS Y axis [R=0.406]; ECSS Y axis [R=0.368]) decreases, also observed in the disability variable at 3-months follow-up. The rest of the associations between variables showed no statistically significant differences ($P>0.05$).

Discussion

The aim of the present study was to ascertain the changes occurred after two exercise modalities were implemented (supervised exercise therapy [SET] and laser-guided exercise therapy [LGET]) combined with PNE on postural control measured by displacement of CoP and ESD in subjects with NSCLBP. Overall, both groups achieved improvements in the variables analyzed. However, the LGET program combined with PNE showed greater improvements in ESD compared to the SET group. Although, there are several studies on the effectiveness of therapeutic exercise in NSCLBP,[3, 13, 16, 17, 20, 38, 47-49] to date, this is the first clinical trial to analyse postural control-related variables that have previously been shown to be sensitive to change between subjects with NSCLBP and healthy subjects after the application of therapeutic exercise modalities.[24] Results showed statistically significant changes in displacement of CoP and ESD ($P<0.05$) for both groups (SET and LGET group). Both just after the end of the 8-week programme and at 3-month follow-up. Although, effect sizes in all conditions were higher in the LGET group (Table II). Regarding the displacement of CoP, the observed improvements could be explained by two reasons: 1) by the hypoalgesic effects produced by therapeutic exercise in chronic musculoskeletal disorders[3, 34] – the desensitisation of nociceptive mechanisms could be associated with a reduction of disruptive stimuli on postural control mechanisms;[16, 26] 2) by the recruitment of the local muscles of the spine and hip, especially extensor muscles reported by an exercise programme similar to the one proposed in the present study.[13] In this sense, Lopes *et al.*[13] achieved statistically significant improvements ($P<0.05$) in displacement of CoP in the ECUS situation in both ML (Dif pre-post: 0.6 cm) and AP (Dif pre-post: 0.6 cm) axes after applying specific

spinal stabilisation exercises in subjects with NSLBP. Our results were superior to those shown by these authors (ML axis: group SET: Dif pre-post: 1.80; group SET: Dif pre-post LGET: 1.48; axis AP: group SET: Dif pre-post: 2.69; group SET: Dif pre-post LGET: 3.62) which may be due to the longer duration of the programme (single session *vs*. 16 ss). On the other hand, Ghasemi *et al.*[14] analyzed the combination of a therapeutic exercise programme with manual therapy techniques (muscle energy technique and craniosacral therapy) applying 10 sessions for 5 weeks (2 sessions/week). For the eyes open/eyes closed stable surface situation, the results obtained by these authors indicated positive effects on the displacement of CoP in the ML and AP axis, the effect sizes being lower than those reported in the present study for any of the conditions analyzed. On this line, wee have not found any study analysing displacement of CoP after combining any therapeutic exercise modality and PNE. Therefore, future studies are needed. On the other hand, for the ESD variable post-treatment, the difference in means between groups was 1.27 J/MHZ for the variable ESD EOUS Z axis and 1.23 J/MHZ for the ESD ECUS Z axis, with statistically significant differences for the LEGT group. In this sense, Caña Pino *et al.*[24] established as a cut-off value through the ESD ECUS Z variable, differences between healthy and NSCLBP populations. This cut-off value was set at 1.6 J/MHZ. This could be clinically relevant, given that in the present study, the LGET group obtained baseline values of 2.83 J/MHZ, post-treatment of 1.79 J/MHZ, and a 3-month follow-up of 1.51 J/MHZ. In the SET group, although there was a decrease in the ESD ECUS Z axis at all assessment moments, there were no significant differences. Our results are consistent in that externally focused exercise improves parameters related to postural control.[12] The LGET group maintained the positive effects of the intervention at 3 months follow-up with a reduction in ESD. Ghasemi *et al.*[14] found an improvement at 2 months after intervention in displacement of CoP with a therapeutic exercise programme. Given that improvements are made over time, the assessment with the 3-month follow-up seems to be important to detect the extent of the intervention carried out. It has been suggested that visual input is the most reliable source of information necessary for the execution of a motor command.[16, 50] On this line, visual dependence, as with LGET, can improve postural stability, which depends on the interactions of visual information with the environment. In this sense, NSCLBP has been associated with postural control through alterations in sensory and proprioceptive afferents.[50, 51] Variation in the proprioceptive system through unstable surface situations may make the postural control of subjects with NSCLBP dependent on visual information, and therefore visual feedback in these patients may be relevant.[16] Exercises used in this study may have helped in the maintenance and adjustment of trunk position by improving muscle activity and improving trunk muscle activation patterns.[13] Variability in the results in terms of correlations between clinical variables and postural control variables may be due to the variability that exists in patients with NSCLBP in subjective variables (pain and disability) at different times in the evolution of the pathology. Previosly knowing that ESD and CoP are variables with good reliability in subjects with NSCLBP, allows us to give greater objectivity to subjective variables that patients indicate to us, such as the intensity of perceived pain and disability. Furthermore, in the study by Caña Pino *et al.*[24] they observed that the less displacement of CoP, the better the postural control. In this sense, the fact that the clinician can indirectly monitor the evolution of clinical variables of a subjective nature that have not traditionally been associated with instrumental assessment instruments, it may be possible to have variables that can be controlled by their association with other variables.[13, 25, 52] Although, other recent studies indicate that the use of a pressure platform in the four conditions assessed does not seem to be suitable for the diagnosis of postural control disorders in subjects with NSCLBP,[24, 51] the results shown in the present study indicate that its use could be considered of interest in the clinical evolution of subjects with NSCLBP when a therapeutic exercise program and PNE are applied.

Clinical implications

These results may provide a new therapeutic exercise modality in NSCLPB subjects, as postural control deficits are not usually addressed in the treatment of NSCLBP.[4] In addition, the assessment using technological devices makes the results objective and guides therapeutic interventions, and therefore, gives greater objectivity to subjective variables that patients indicate to us, such as the intensity of perceived pain and disability. On the other hand, LGET-based motor control exercises could be a useful tool for both patients and therapists to guide them in correct ranges of movement.

Limitations of the study

Although the variability in these technological devices is well known and the difficulty of translating these variables related to postural control to clinical settings,[26, 51] we consider that the homogeneity observed between groups

in our study concerning variables such as BMI will not influence the results obtained. The main limitation of this study is the absence of a control group that did not receive an intervention. This would have allowed us to compare results from both treatment groups with the natural history of NSCLBP. On the other hand, our results cannot be extrapolated to adults presenting specific causes of low back pain and no sociodemographic factors with potential effect on the results (*e.g.*, occupation). Finally, the effect of the intervention was only assessed at 3-month follow-up. Future studies are necessary to study the long-term effects of this combined intervention; to analyze other clinical variables (kinesiophobia, catastrophic pain, propioception, pressure pain threshold, etc.) after applying various exercise modalities (SET and LGET) plus PNE, as well as the correlation with variables related to postural control. Another future line of research would be to apply the studied exercise modalities in other populations with chronic pain (*e.g.*, cervical pain).

Conclusions

Both exercise modalities (SET and LGET) showed changes in variables related to postural control (displacement of CoP and ESD); however, the LGET program combined with PNE showed greater improvements in ESD compared to the SET plus PNE program.

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