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Rowers with a recent history of low back pain engage different regions of the lumbar erector spinae during rowing

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1 **ABSTRACT**

2 **Objectives:** Despite the high prevalence of low back pain (LBP) in rowers, there are few
3 studies investigating changes in lumbar muscle activation in rowers with a recent history of
4 LBP. Such knowledge is relevant to understand potential mechanisms contributing to the
5 maintenance and recurrence of LBP in rowers. For the first time, we evaluate the spatial
6 distribution of erector spinae (ES) activity in rowers with and without a recent history of LBP,
7 using a novel application of high-density surface electromyography (HDEMG).

8 **Design:** Cross-sectional study.

9 **Methods:** Asymptomatic rowers (N=10) and rowers with a recent history of LBP (N=8)
10 performed 7x4-min exercise bouts (rowing ergometer) until volitional exhaustion. HDEMG
11 signals were acquired bilaterally over the lumbar ES and the root mean square (RMS)
12 amplitude and entropy were analyzed. In addition, the y-axis coordinate of the barycentre
13 (RMS-map) was used to assess changes in ES spatial activation.

14 **Results:** As the load increased, rowers with LBP showed higher amplitude ($p<0.01$) and less
15 complexity (entropy) of the HDEMG signals ($p<0.001$). In addition, rowers with LBP showed
16 opposite displacements of the barycentre, specifically showing a caudal shift of muscle
17 activity at high intensities ($p<0.001$).

18 **Conclusion:** Both the magnitude of activation and distribution of ES activity were altered in
19 rowers with a recent history of LBP. The lower complexity of signals together with the caudal
20 displacements of the barycentre suggest an inefficient recruitment of the ES as the load
21 progressed. Modification of the rowing technique in conjunction with feedback from HDEMG
22 might prove useful in future studies.

23 **Keywords:** Rowing; Low back pain; High-density surface electromyography; Erector Spinae

24 INTRODUCTION

25 Rowing is a sport with a high incidence of low back pain (LBP), with an annual rates of
26 occurrence between 32 and 51%^{1,2}. Rowing is defined by cyclical lumbar flexion through the
27 'drive' phase of the stroke when the rower applies work and the hips, knees and trunk move
28 from full flexion to relative extension. The 'recovery' phase of the stroke is when no work is
29 applied and the rower moves from relative extension back to full flexion to another work
30 cycle; in a boat, this is when the oars are out of the water. It has been recognized that cyclical
31 lumbar flexion, particularly when combined with fatigue, may alter joint mechanics and
32 loading patterns of the lumbar spine, possibly leading to risk of tissue failure and injury³. In
33 the latest review of Wilson et al., it was discussed that the volume of training on an ergometer
34 is one of the most important risk factors for LBP¹. Studies have shown that flexion of the
35 lumbar spine is increased by approximately 10% at the end of an incremental test in a rowing
36 ergometer compared to that of the rowing boat⁴. High-intensity rowing on an ergometer also
37 increases L5/S1 joint loading⁵, probably due to increased fatigability of the lower limb
38 muscles⁶, showing that athletes likely involve the lower lumbar spine to compensate for the
39 overall decrease in stroke force. These changes are also observed during submaximal rowing,
40 as lumbar spinal motion increased during the course of a 60-min steady-state ergometer trial
41⁷. It is expected that these changes in lumbar spine kinematics and kinetics are accompanied
42 by different activation patterns of the lumbar muscles. However, there is a paucity of studies
43 quantifying lumbar muscle activation patterns in rowers with a recent history of LBP, and the
44 studies that have been conducted, have applied classic bipolar surface electromyography
45 (EMG)^{8,9}, which is known to have large variability and low reliability¹⁰. High-density EMG

46 (HDEMG), using grids of tens of electrodes, increases the reliability and sensitivity of
47 amplitude estimates ^{11 12 13}. In addition, HDEMG measures the spatial distribution of muscle
48 activity and can identify relative adaptations in the intensity of activity within regions of a
49 muscle ^{14 15}. Changes in homogeneity/heterogeneity of HDEMG have been previously used to
50 evaluate changes in muscle behaviour in LBP ¹⁴; nevertheless, this technique has never been
51 used in rowers or during a rowing task. Here we apply this methodology to assess the
52 topographical distribution of lumbar erector spinae (ES) activity of rowers with and without a
53 recent history of LBP during an incremental rowing test. It was hypothesized that the rowers
54 with a recent history of LBP would show altered activity within regions of the lumbar ES
55 revealing a sub-optimal pattern of ES muscle use.

56

57 **METHODS**

58 *Study design and participants*

59 A cross-sectional study was conducted on 18 rowers (junior, senior, and veteran)
60 recruited via print and social media advertisements. All participants were rowing at a
61 competitive level (club or international level) for at least one year and training a minimum of
62 7 sessions per week. Participants with a recent history of LBP (n=8, 5 male/3 female, age 32.1
63 (17.6) years, BMI= 24 (3), % body fat= 19.0 (6.8) %, years rowing= 15 (13.3)) experienced
64 rowing-related symptoms within the last 6 months [Oswestry Disability Index of 21 (2) %] but
65 not during the 6 weeks prior to the study. Rowers free of LBP (n=10, 8 male/2 female, age
66 27.0 (14.3) years, BMI= 23 (1), % body fat= 16.3 (4.9) %, years rowing= 12.1 (13.5)) for >12
67 months served as controls. The study was conducted in accordance with the declaration of
68 Helsinki and the Ethics Committee of Trinity College Dublin Faculty of Health Sciences

69 approved the study. Participants provided informed written consent and a pre-participation
70 screening tool was applied to confirm that the participants were fit to test.

71 *Testing protocol*

72 Following the application of testing equipment (HDEMG electrodes, electro-
73 goniometer and heart rate monitor; see below for details), the rowers performed a 5-minute
74 warm up on a Concept2 (Model D, USA) rowing ergometer at a power output equivalent to
75 the first stage of the subsequent test. Participants then performed the 7x4-min incremental
76 rowing test, devised by the Australian Institute of Sport (see appendix A). Starting workload
77 and incremental load were determined by participant's previous best 2000m time. Thus, the
78 rowers started at a load ranging between 140 and 200W and an incremental increase of 15
79 to 45 W. Stage duration was 4-min with 1-min recovery, during which blood lactate was
80 measured from the earlobe via Lactate Pro 2 monitor (COSMED, Italy). Lactate threshold was
81 quantified using the V-Slope method ¹⁶.

82 *Electromyography (EMG) and kinematics data acquisition*

83 During the test, both HDEMG signals and knee motion were recorded continuously.
84 Surface HDEMG signals were detected with semi-disposable adhesive grids of electrodes (OT
85 Bioelettronica, Italy). Each grid consisted of 13 rows and 5 columns of electrodes (1-mm
86 diameter, 8-mm inter-electrode distance in both directions). Following skin preparation, the
87 electrode grids were placed bilaterally over the lumbar ES, 2cm lateral to the lumbar spinous
88 processes, starting at the level of L5, extending approximately to the level of L3 ^{17, 18}. The
89 electrode grid covers the iliocostalis lumborum pars lumborum and the iliocostalis lumborum
90 pars thoracis, with the muscular portions of the longissimus being too medial or too cranial
91 to be covered by the electrode grid ¹⁸.

92 Conductive paste was inserted into each cavity of the grid to provide electrode-skin
93 contact. A reference electrode was placed over the 7th cervical vertebra. EMG signals were
94 amplified (400-channel HDEMG amplifier, OT Bioelettronica, Torino, Italy; -3dB bandwidth 10-
95 500 Hz) by a factor of 150, sampled at 2048 Hz, and converted to digital form by a 16-bit AD
96 converter. HDEMG signals were recorded in monopolar mode (64 channels), however, these
97 signals were then re-referenced offline to form 59 bipolar derivations, as the difference
98 between adjacent electrodes in the direction of the muscle fibres.

99 Pairs of bipolar electrodes (15 cm inter-electrode distance, Spes Medica, Italy) were
100 mounted over the rectus abdominis (RA) and external oblique (EO) muscles bilaterally
101 according to guidelines ¹⁹. This was done with the aim of assessing co-activation (formula:
102 right EO + right RA + left EO + left RA RMS: right ES + left ES RMS x 100) during the drive phase.
103 Sagittal motion of the rowing task was measured with a twin axis SG150B electrogoniometer
104 placed over the knee joint (Biometrics Ltd., UK) with one axis (sagittal plane) used for analysis.
105 The goniometer was attached to the knee joint since movement in the lumbar region is
106 typically small ⁴ and does not allow to accurately distinguish the different phases of rowing
107 (drive and recovery). The goniometer signal was acquired by the EMG amplifier (OT
108 Bioelettronica, Italy) and sampled at 2048 Hz to ensure synchronisation of the data.

109 *EMG and kinematic data analysis*

110 EMG signals were firstly band-pass filtered (20-350 Hz, second order, zero lag
111 Butterworth). Root mean square (RMS) values were computed from each bipolar recording
112 (59 bipolar EMG signals) obtained from the grid (12 longitudinal bipolar recordings in each
113 column except the far left and right, which had 11 electrode pairs) ¹⁴. For graphical
114 representation, the 59 values were interpolated by a factor of 8, but only the original values
115 were used for data processing and statistical analysis. To characterize the spatial distribution

116 of muscle activity, the following variables were extracted from the 59 bipolar signals: RMS
117 (averaged over the 59 signals), modified entropy (measure of uniformity that can be used to
118 indicate the degree of homogeneity in muscle activation ^{15, 20}. A reduction in entropy values
119 means that signals are becoming more heterogeneous) and the y-axis coordinate of the
120 barycentre of the RMS map (cranial-caudal direction) ¹⁴. Values of RMS, modified entropy and
121 y-axis coordinate of the barycentre were calculated during two different epochs during the
122 drive phase, therefore, EMG signals were analysed from full flexion to 33% of extension
123 (epoch 1) and 33% of extension to 66% of extension (epoch 2). These epochs were selected
124 because they represent the phases of the drive where the ES is mostly active (epoch 1
125 represents the beginning of ES activation while epoch 2 represents the period of peak force
126 production) ²¹. Co-activation between the flexors and extensors was quantified on the first
127 two epochs and in a third epoch [66% of extension to full extension (epoch 3)] as presented
128 previously ²¹. The windows where these phases were calculated was adjusted to kinematic
129 changes (i.e. if the participants increased stroke rate, the windows still represent full flexion
130 to 33% of extension and 33% of extension to 66% of extension) and were not fixed in time as
131 done in previous studies ^{21, 22}. This was necessary as we instructed participants to perform the
132 incremental test as naturally as possible (with their preferred stroke rate) in order to mimic a
133 competitive scenario.

134 Extracted EMG values were averaged for the full duration of each incremental step (7
135 values of RMS, y-axis barycentre and entropy); however, only the first five steps were
136 considered for further analysis, since in some cases large movement artefacts and/or
137 sweating during the last two steps affected signal quality. To allow comparisons between
138 rowers with and without a recent history of LBP, the RMS values were expressed as a
139 percentage relative to the initial value, which was the average value of RMS in the first minute

140 of the first exercise step of each epoch. This method of normalization was employed due to
141 two reasons; first, individuals with a history of low back pain may not be able to activate their
142 muscles maximally and therefore normalization against a submaximal contraction is
143 preferable²³. Second, this method has shown to be the most sensitive to measure changes in
144 muscle activation during incremental exercise (cycling)²⁴.

145 All EMG variables extracted from the right and left electrode grids were compared
146 within each group before the EMG variables were compared between groups. This was
147 necessary to determine the most appropriate side of comparison between groups (e.g. LBP
148 right vs. control right or LBP painful side, e.g. left vs. control left).

149

150 *Statistical analysis*

151 The normality of the data was tested using the Shapiro–Wilk’s test. The assumption of
152 sphericity was checked by the Mauchly test, and in case of violation, the Greenhouse–Geisser
153 correction was applied. Homogeneity of variances was checked with a Levene test. Data was
154 analysed descriptively via means and standard deviations (SD). Anthropometrics, lactate and
155 power output between groups were compared by independent t-tests. Potential side
156 differences in EMG variables were examined within the group with a recent history of LBP
157 using 3-way repeated measures analysis of variance (ANOVA) with factors side (left, right),
158 load (15, 30, 45, 60, 75% peak power output) and epoch (one and two). Statistical comparison
159 for EMG variables between groups (RMS, entropy, y-axis barycentre) was performed using 4-
160 way ANOVA with factors side, load, epoch and group. The degree of co-activation between
161 the lumbar ES and abdominal muscles was evaluated using 3-way ANOVA with factors of
162 group, epoch and load. Student-Newman-Keuls test was used for pairwise comparisons when
163 ANOVA was significant. Finally, the partial eta-squared (η^2) for ANOVA was used to examine

164 the effect size of the differences in EMG parameters between groups. A η^2 less than 0.06
165 was classified as “small”, 0.07-0.14 as “moderate”, and greater than 0.14 as “large”. Alpha
166 level was set at 5%.

167

168 **RESULTS**

169 There were no differences in anthropometrics or rowing profiles between groups
170 ($p>0.27$ in all cases). Moreover, there were no group differences in the intensity where the
171 lactate threshold was identified (control= 3.20 (0.46) w/kg vs. LBP= 2.92 (0.50) w/kg, $p=0.49$)
172 and peak power output (control= 3.94 (0.76) w/kg vs. LBP= 3.96 (0.83) w/kg, $p= 0.97$).

173 *Evaluation of side to side differences*

174 In the group with a recent history of LBP, 4 participants had a history of pain on the
175 right, 3 on the left and one central, however, there were no side differences for any of the
176 EMG variables in either group ($p>0.13$ in all cases). Therefore, for all further analyses the EMG
177 variables were assessed between groups according to the same side (e.g. right ES LBP vs. right
178 ES control).

179 *Y-axis barycentre*

180 Asymptomatic rowers showed cranial displacements of the barycentre at high loads,
181 while rowers with a recent history of LBP showed a caudal shift of the barycentre throughout
182 the rowing trial. **Figure 1** presents representative topographical maps of the EMG RMS value
183 recorded from the ES for a rower in the control group and a rower with a recent history of
184 LBP throughout the incremental test (5 steps, epoch 2). These representative results were
185 confirmed for the group of participants (**Figure 2**) as there was a significant interaction
186 between group and load ($F=5.49$, $p=0.001$, $\eta^2=0.26$).

187 *Average EMG amplitude*

188 The rowers with a recent history of LBP showed higher ES EMG amplitude compared
189 to the rowers without LBP during epoch 2, while the asymptomatic rowers maintained their
190 level of activation relatively constant on both sides (right, left) in both epochs (**Figure 3a**) (3-
191 way interaction between epoch, load and group; $F= 2.81$, $p=0.032$, $\eta p^2=0.15$).

192 *Modified entropy*

193 The asymptomatic rowers showed increased heterogeneity of their EMG signals as the
194 load progressed, in comparison to the rowers with a recent history of LBP (group x load
195 interaction, $F=3.66$, $p=0.01$, $\eta p^2=0.19$) (**Figure 3b**). The right ES was the side showing the
196 largest reduction in entropy for the control group (group, muscle side and load interaction,
197 $F=2.81$, $p=0.033$, $\eta p^2=0.15$).

198 *Co-activation*

199 There were no differences in the level of co-activation between groups during the
200 drive phase in all epochs ($F=1.68$, $p=0.109$, $\eta p^2=0.095$) (see supplementary figure).

201

202 **DISCUSSION**

203 This study is the first to demonstrate that both the level and distribution of activation
204 of the lumbar ES muscle is altered in rowers with a recent history of LBP. This, in addition to
205 the lower complexity of the EMG signals, suggests an inefficient recruitment of the ES muscle
206 in rowers with a recent history of LBP, which likely has significant implications for the
207 perpetuation of LBP.

208 *Spatial changes in the distribution of lumbar ES activity*

209 Previous studies using classic bipolar EMG techniques have evaluated lumbar ES
210 activation in healthy rowers^{3, 25-27}, but no previous study has investigated the distribution of
211 activity within the lumbar ES during rowing. During incremental exercise, healthy rowers

212 show no significant displacement of ES activity up to loads ~45% of the peak power output.
213 Activation patterns change at higher loads as rowers show a cranial shift in ES activity (Figures
214 2 and 3). These findings can be interpreted according to the results obtained from studies
215 evaluating lumbar kinetics and kinematics. Previous studies investigating asymptomatic
216 rowers show increased levels of lumbar flexion over the course of a 2000m race simulation
217 ²², incremental rowing test ⁴ and prolonged steady-state rowing ^{3, 7, 21, 22}. It is very likely that
218 increased fatigability of lower limb muscles influenced spinal motion. Accordingly, Buckeridge
219 et al ⁵. found that shear and compressive forces on the L5/S1 segment increases at higher
220 loads, while peak knee extensor moments decreased. Taken together, these observations
221 suggest that it is not entirely possible to avoid the increased movement of the lumbar spine
222 at high rowing intensities; therefore, it would be necessary to transfer movement to higher
223 lumbar areas, which would place the ES in greater mechanical advantage, thus potentially
224 protecting the spine from compressive and shear forces. These observations are important
225 considering our findings in symptomatic rowers, since they showed a consistent caudal
226 displacement of ES activity as the load progressed. This likely placed the ES in a mechanical
227 disadvantage, possibly increasing load on lower lumbar areas. Accordingly, in one of the few
228 studies that evaluated rowers with LBP, Ng et al. ²⁸ showed that rowers with LBP have less
229 excursion of the upper lumbar spine during the drive phase, maintaining a flexed posture of
230 the lower lumbar spine while rowing at high intensities. Although ES activity was not assessed
231 in that study, it is very likely that these rowers would also show a caudal displacement of
232 lumbar ES activity as the rowing trial progressed, given the differences in lumbar movement
233 observed.

234 *Changes in the amplitude of ES activity*

235 Previous research using bipolar EMG showed that asymptomatic rowers do not
236 increase their level of ES activation as workload increases ^{21, 22, 25, 29}. Our findings are in
237 agreement, as the asymptomatic rowers maintained their activation levels throughout the
238 trial. In contrast, the rowers with a recent history of LBP showed higher ES activity compared
239 to the control participants during the epoch where the ES was most active (2nd epoch, **Figure**
240 **3a**). Recent research using HDEMG in people with chronic non-specific LBP has also shown
241 increased levels of ES activation relative to asymptomatic people ¹⁴. The authors suggested
242 that the increased amplitude could be due to an increased excitatory drive to painful muscles.
243 However, in the present study, none of the rowers experienced pain during the trial.
244 Therefore, these changes are most likely attributed to an altered rowing pattern requiring
245 higher activation of the more distal regions of the ES, compensating for increased flexion ²⁸.
246 This observation can be confirmed by the co-activation data since there was no difference in
247 the level of co-activation between groups, suggesting that the increased activation of the ES
248 in the LBP group was not due to increased activation of the antagonists but rather due to
249 changes in lumbar kinematics (increased lumbar flexion).

250 *Changes in the uniformity of muscle activity*

251 An increase in the heterogeneity of HDEMG signals (lower entropy) is thought to be
252 related to altered spatial reorganization of muscle activity potentially with the aim of reducing
253 muscle fibre overload during fatiguing contractions ^{15, 30}. Therefore, while the barycentre
254 provides an estimate of where the average activity of the muscle activity is positioned in space
255 (centre of mass of the HDEMG map), entropy provides an estimate of homogeneity between
256 signals of the whole 2D HDEMG map.

257 As predicted, rowers with a recent history of LBP showed higher values of entropy
258 compared to the asymptomatic rowers as the load progressed (**Figure 3b**). As appreciated in

259 Figure 1, the rower with a recent history of LBP increases ES activity (RMS amplitude) during
260 the trial but the regions that were activated are maintained (with a higher increase in
261 amplitude in caudal regions of the map as shown in the barycentre results). On the contrary,
262 the rower without a recent history of LBP shows a clear difference in homogeneity, as regions
263 that were active during the beginning of the trial (caudal regions at 15, 30 and 45% of peak
264 power output) become less active at 60 and 75% of peak power output.

265 These results reinforce our findings that rowers with a recent history of LBP fail to
266 recruit their lumbar ES muscle efficiently. They shift activity towards lower lumbar regions,
267 but also maintain motor output by a more homogenous activation of the ES, thereby
268 activating regions which are at greater mechanical disadvantage.

269 *Implications*

270 Optimising the rowing technique is essential to avoid the development or
271 maintenance of LBP. One of the most important factors to correct is lumbo-pelvic positioning
272 during the stroke phase ^{1, 4, 7, 31}. Neutral positioning of the pelvis allows efficient movement
273 of the hips, decreasing both flexion of the lumbar spine and the activation of the lumbar
274 muscles. The development of new HDEMG methods allowing visualization of ES spatial
275 changes in real time could prove a useful biofeedback tool in helping rowers to reduce the
276 activity of their lower lumbar ES and facilitate more efficient lumbo-pelvic motion.

277 *Limitations*

278 There are some methodological considerations which warrant attention. Firstly, the study was
279 conducted on a relatively small and heterogeneous sample of elite rowers. It was not possible
280 to assess differences between rowers with different levels of experience. Future
281 investigations should aim to evaluate differences between rowers with different levels of
282 expertise since it could be expected that more refined use of the ES would be present in

283 internationally competitive rowers versus those with less experience. Secondly, a Concept II
284 stationary ergometer was used for all trials. Differences in stroke mechanics have previously
285 been reported comparing stationary and dynamic ergometers^{4, 32-34}. However, no differences
286 in ES activity were observed comparing rowing ergometer designs to each other²⁷ or to on-
287 water sculling²⁵. Nonetheless, it is possible that results may differ when performed on a
288 dynamic ergometer (i.e. Rowperfect) or on-water. Thirdly, due to the complexity of recording
289 HDEMG from the participants under such demanding conditions, it was not possible to
290 quantify lumbar kinematics. Finally, it is important to mention that the participants in this
291 study did not experience pain during the rowing trial. Longitudinal studies, specifically
292 evaluating muscle behaviour whilst rowing during periods with and without pain should be
293 conducted in order to better understand the relationship between LBP symptoms and an
294 altered spatial distribution of muscle activity in rowers. Furthermore, prospective studies
295 examining risk of LBP should assess EMG longitudinally to examine if specific recruitment
296 patterns can predict LBP onset or response to rehabilitation.

297 *Conclusion*

298 Both the magnitude of activation and the distribution of ES activity were altered in
299 rowers with a recent history of LBP compared to back pain-free rowers. The lower complexity
300 of signals together with the caudal displacement of the centre of activity at high work
301 intensities suggests an inefficient recruitment of the ES as the load progressed. Modification
302 of the rowing technique in conjunction with feedback from HDEMG might prove useful in
303 future studies.

304 **PRACTICAL IMPLICATIONS**

- 305 - During an incremental rowing test, rowers with a recent history of low back pain show
306 increased activation of the lumbar erector spinae

- 307 - This increase in activation was accompanied by recruitment of mechanically inefficient
308 portions of the lumbar erector spinae (lower regions of the lumbar erector spinae) at
309 high loads
- 310 - Rowers with a history of low back pain show more homogeneous activation of the
311 lumbar erector spinae compared to controls, which likely relates to preferential
312 recruitment of a smaller portion of the muscle as the load progresses

313 **Disclosure statement**

314 The authors report no potential conflicts of interest

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427 **FIGURE LEGENDS**

428 **Figure 1.** Representative topographical maps (interpolation by a factor 8) of the EMG root
429 mean square (RMS) value recorded from the right lumbar erector spinae (ES) for a rower
430 without a recent history of low back pain (NLBP, up) and with a recent history of LBP (LBP,
431 down) during the rowing incremental test (15, 30, 45, 60 and 75% peak power output).
432 HDEMG maps were extracted from the second epoch (peak force phase). The white circle in
433 the middle of each map represents the barycentre (centre of activity). The full displacement
434 of the barycentre across the task for both rowers can be seen on the right side of the Figure.
435 For the NLBP rower, the barycentre started to shift cranially from 45% peak power output
436 while for the LBP rower the barycentre it displaced caudally from 30% peak power output.

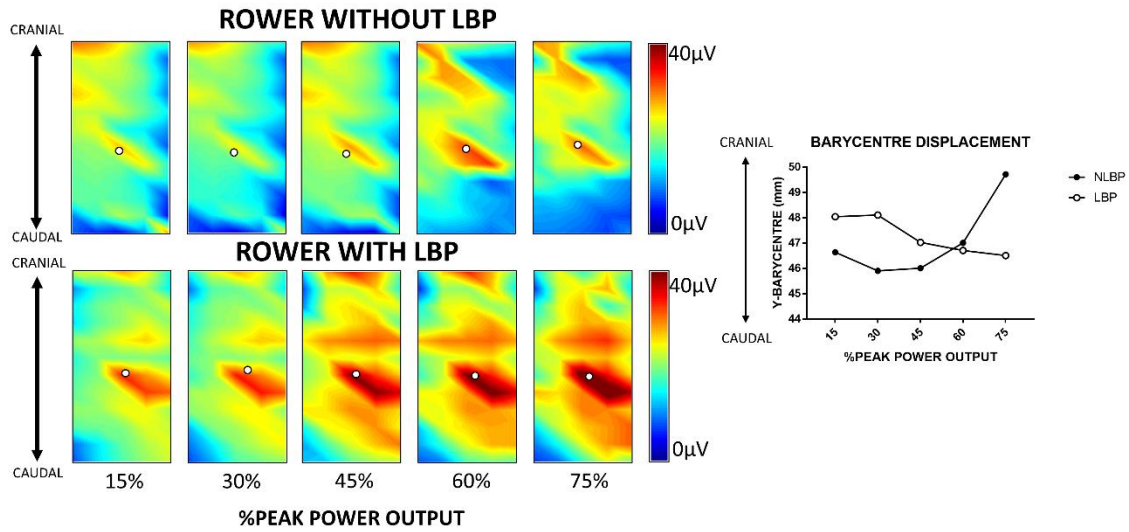
437 **Figure 2.** Displacement of the barycentre of the left and right erector spinae (ES) at five steps
438 (15, 30, 45, 60, 75% peak power output) of the incremental test in the three different epochs.
439 Rowers without a recent history of low back pain (NLBP, black circles); rowers with a recent
440 history of low back pain (LBP, white circles). *, significant caudal displacements of the
441 barycentre in the LBP group ($p < 0.001$). #, significant cranial displacements of the barycentre
442 in the NLBP group ($p < 0.001$).

443 **Figure 3.** Root mean square (RMS, A) and entropy (B) values of the left and right erector
444 spinae (ES) at five steps (15, 30, 45, 60, 75% peak power output) of the incremental test in
445 the first and second epochs. Rowers without a recent history of low back pain (NLBP, black
446 circles); rowers with a recent history of low back pain (LBP, white circles). *, significant
447 increase in RMS amplitude in the LBP group ($p < 0.001$). #, significant decrease in entropy in
448 the NLBP group ($p < 0.001$). A.U., arbitrary units.

449

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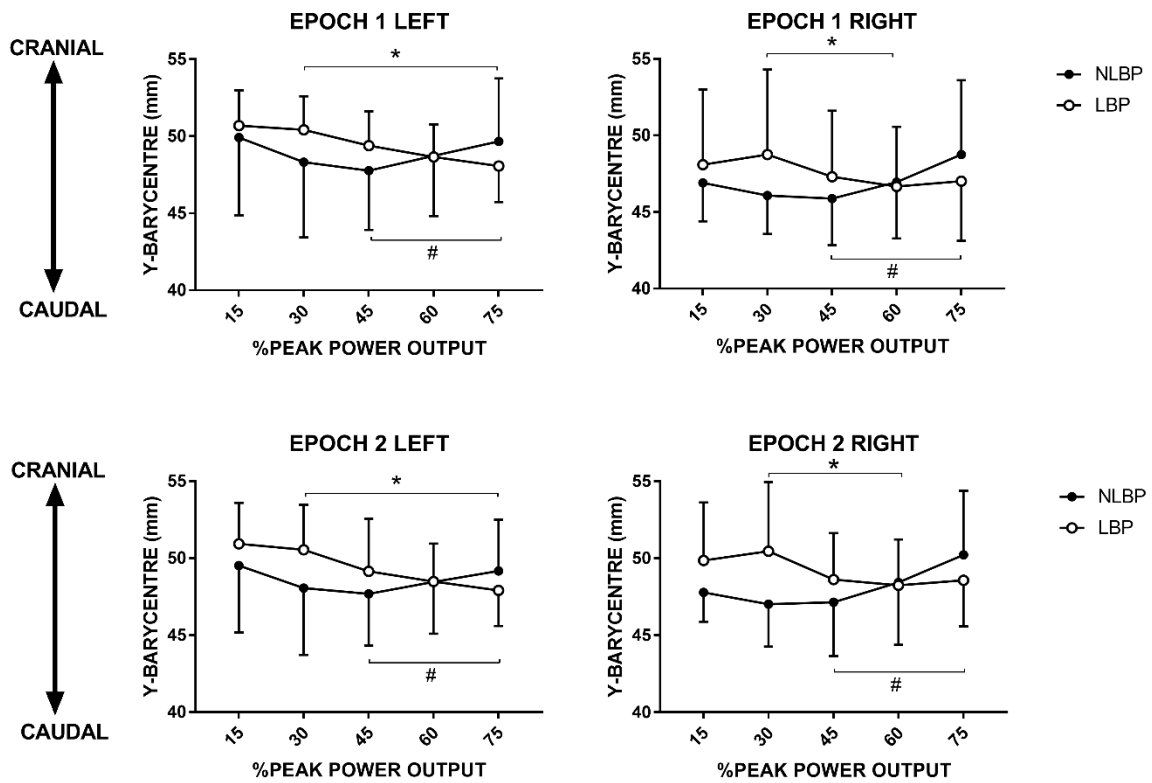
451 FIGURE 1



452

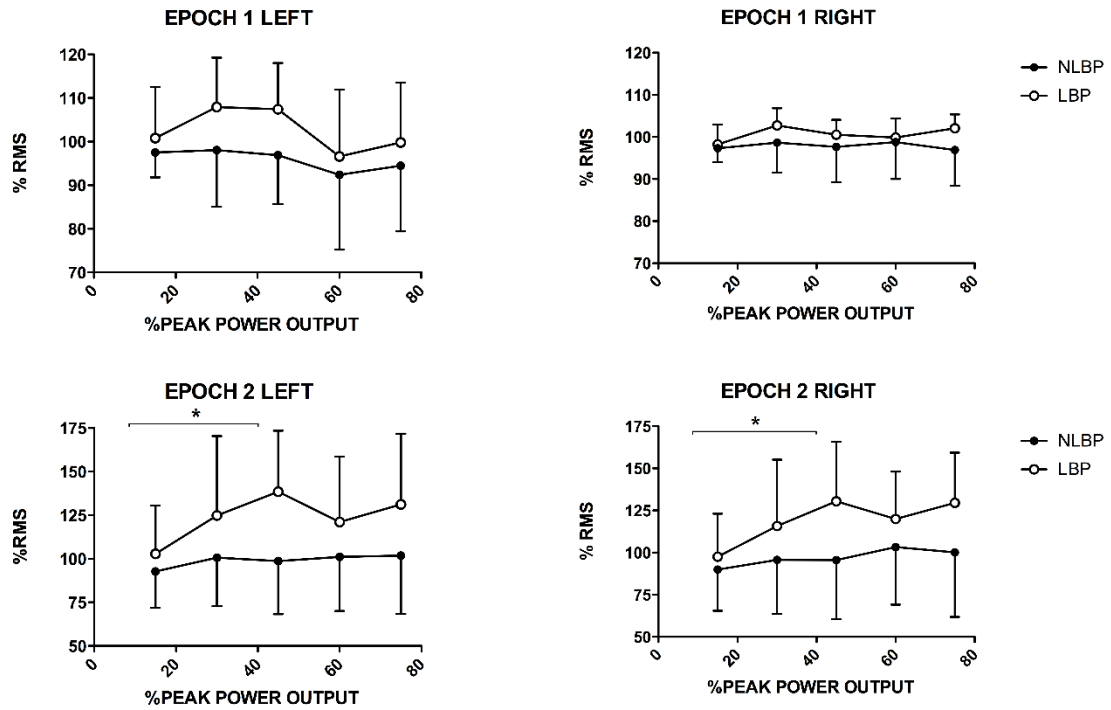
453 FIGURE 2

Y-AXIS BARYCENTRE



454

A) AMPLITUDE



B) ENTROPY

