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**The onset of deep abdominal muscles activity during tasks with different trunk
rotational torques in subjects with non-specific chronic low back pain**

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Aizuwakamatsu, Japan.**

1 **ABSTRACT**

2 **Background:** Although delayed onset of the deep abdominal muscles activity in subjects with
3 non-specific chronic low back pain (CLBP) has been suggested to be related to trunk
4 rotational torque, no study has examined the onsets associated with non-specific CLBP during
5 a variety of tasks with different trunk rotational torque. The aim of this study is to compare the
6 onsets of deep abdominal muscles activity among tasks with different trunk rotational torques
7 in subjects with and without non-specific CLBP.

8 **Methods:** Twelve subjects with non-specific CLBP and 13 control subjects were included.
9 They performed 8 types of upper limb movements. The onsets of muscular activity of bilateral
10 internal oblique-transversus abdominis (IO-TrA) and trunk rotational torque due to the upper
11 limb movements were measured using a surface electromyography and a three-dimensional
12 motion analysis system.

13 **Results:** In non-specific CLBP group, right IO-TrA activities were significantly delayed
14 during tasks with left trunk rotational torque compared with the control ($P < 0.05$), while
15 onsets of the left IO-TrA activities were significantly later than those of the control during
16 tasks with right rotational torque of the trunk ($P < 0.05$). There were no significant differences
17 in onsets of both sides IO-TrA during tasks without trunk rotational torque between
18 non-specific CLBP and control groups ($P > 0.05$).

19 **Conclusions:** The onsets of IO-TrA activities in subjects with non-specific CLBP were

20 delayed during tasks with rotational torque of the trunk in the opposite direction, suggesting a
21 possibility that delayed onset of the deep abdominal muscles during rotational torque of the
22 trunk might be etiology of chronic low back pain.

23 **1. Introduction**

24 Non-specific low back pain with no identifiable underlying pathology accounts for
25 90% of cases of low back pain [1]. Patients with non-specific acute low back pain demonstrate
26 a favorable improvement rate in the first six weeks [2], but approximately 40% of patients will
27 develop non-specific chronic low back pain (CLBP) [3]. One proposed mechanism for
28 non-specific CLBP is instability of the spine due to the lack of neuromuscular control [4].

29 The deep abdominal muscles play an important role in lumbopelvic stability [5].

30 Several studies indicated that the onsets of transversus abdominis activities in response to
31 movements with rotational torque of the trunk were prior to those of other trunk muscles [6, 7,
32 8]. Early activation of the deep abdominal muscles is therefore considered to contribute to the
33 control of spinal stiffness [7, 9]. Dysfunction of the deep abdominal muscles activities could
34 contribute to persistence of low back pain [10].

35 The onset latency of deep abdominal muscles in rapid movements was delayed in
36 subjects with non-specific CLBP compared to healthy subjects [6, 8]. On the other hand, there
37 were no significant differences in onsets of transversus fibers of the internal oblique and
38 transversus abdominis (IO-TrA) activation during bilateral shoulder flexion between healthy
39 individuals and subjects with non-specific CLBP [11]. Therefore, we hypothesized that the
40 delay of the onset of the deep abdominal muscles would be related to the magnitude of trunk
41 rotational torque during limb movement in subjects with non-specific CLBP and that the

42 delayed onsets of the deep abdominal muscles in response to movements with rotational
43 torque of the trunk might be etiology of CLBP. The purpose of the present study was to
44 compare the onsets of deep abdominal muscles activities among tasks with different rotational
45 torque in the subjects with and without non-specific CLBP.

46

47 **2. Methods**

48 **2.1 Participants**

49 Twelve subjects with non-specific CLBP and 13 healthy control individuals
50 participated in this study. All subjects were recruited in our University thorough advertisement.
51 Non-specific CLBP was defined as pain and discomfort localized between the 12th rib and the
52 inferior gluteal folds for at least 3 months. Subjects with non-specific CLBP were excluded if
53 they had neurologic symptoms, spinal and abdominal surgery in the past 12 months,
54 pregnancy, observable spinal deformity (e.g., scoliosis or kyphosis) and suspected or
55 diagnosed serious spine pathology. Subjects reported minimal or no pain at the time of testing.
56 All participants provided informed consent and completed the Oswestry disability index
57 (ODI). A visual analogue scale was used to characterize their pain. Descriptive statistics for
58 the two groups are reported in Table 1. There were no differences in age, height, weight, and
59 BMI between groups. This research has been approved by the Institutional Review Board of
60 the authors' affiliated institutions.

61

62 **2.2 Experimental procedure**

63 All participants stood on the floor with their arms at their sides while standing
64 comfortably with the eyes directed straight forward. They performed 5 trials of 8 types of
65 rapid upper limb movements: weighted left shoulder flexion and right shoulder extension
66 (WLFRE) (Fig. 1A); left shoulder flexion and right shoulder extension (LFRE) (Fig. 1B); left
67 shoulder flexion (LF) (Fig. 1C); bilateral shoulder extension (LERE) (Fig. 1D); bilateral
68 shoulder flexion (LFRF) (Fig. 1E); right shoulder flexion (RF) (Fig. 1F); left shoulder
69 extension and right shoulder flexion (LERF) (Fig. 1G); weighted left shoulder extension and
70 right shoulder flexion (WLERF) (Fig. 1H). A weight of 1 kg was selected in order to increase
71 the torque without substantially altering the pattern of movement [12]. The participants were
72 instructed to flex their shoulders to approximately 60 degrees and extend them to
73 approximately 40 degrees as fast as possible in response to a beep signal [6]. The emphasis
74 was on the speed of limb movement rather than the accuracy of the angle. The order of each
75 upper limb movement was randomized for each subject. Participants had 3-5 practice trials for
76 each task prior to the measurements.

77

78 **2.3 Data collection**

79 EMG recordings of the bilateral IO-TrA, anterior deltoid (AD), and posterior deltoid

80 (PD) were acquired using a wireless surface EMG system (WEB-1000; Nihon Kohden
81 Corporation, Tokyo, Japan) with surface-type electrode telemeters that sampled at 1000 Hz.
82 Surface electrodes of the IO-TrA were positioned approximately 20 mm medial and inferior to
83 the anterior superior iliac spine [13], and “surface EMG for a non-invasive assessment of
84 muscles (SENIAM)” recommendations were followed for the AD and PD [14]. Surface
85 electrodes were placed in parallel with the muscles fibers.

86 Kinematic data were collected using a motion analysis system (Cortex-64 5.5.0.1579,
87 Motion Analysis Corporation, Santa Rosa, CA, USA) with 7 digital cameras (Hawk cameras,
88 Motion Analysis Corporation) sampling at 200 Hz. Ten reflective markers were attached to
89 each segment with double-sided tape bilaterally at the following locations: acromion, medial
90 epicondyle of the humerus, lateral epicondyle of the humerus, radial styloid process, and ulnar
91 styloid process.

92

93 **2.4 Data analysis**

94 The raw EMG signals were band-pass filtered at 20-500 Hz, full-wave rectified, and
95 filtered using a fourth-order Butterworth low-pass filter with a cut off of 10 Hz. Following
96 data reduction, the onsets of EMG signals were identified using MATLAB software (The
97 MathWorks, Natick, MA, USA) as the point at which the rectified EMG signal first rose
98 above baseline by 1 standard deviation (SD) and lasted for at least 50 msec [11]. The baseline

99 activity was defined as the average of the 500 msec period preceding the beep signal. In
100 addition, each onset of EMG signal was confirmed visually [15]. Onset of IO-TrA activity was
101 expressed relative to the onset of deltoid EMG signal (t=0) (Fig. 2). For BE and BF, the
102 ipsilateral anterior or posterior deltoid onset was determined as t=0 [11]. For the other six
103 tasks, the onset of activity of the anterior deltoid on the flexing side was established as t=0
104 [12].

105 Each marker trajectory was filtered using a fourth-order Butterworth low-pass filter
106 at 12 Hz. Kinematic data were condensed to two-dimensional coordinates in the sagittal plane.
107 Joint reaction forces at the shoulder joint were estimated using free-body diagram force
108 analysis [16]. Trunk rotational torque around a midpoint of the bilateral acromia was
109 calculated from transversus components of the shoulder joint reaction forces. The trunk
110 rotational torque was normalized to body weight for each subject (Nm/kg). The peak
111 rotational torque was determined for analysis of maximum torque during the acceleration
112 phase, defined as the duration from when the angular acceleration at the shoulder first rose
113 above baseline by 2 SD and stayed above this level for 20 msec, to the instant when the
114 angular acceleration crossed the zero line [17]. In addition, peak shoulder angle and peak
115 shoulder angular velocity were calculated during each task.

116

117 **2.5 Statistical analysis**

118 A priori power analysis was performed in G-power 3.1. The sample size was
119 estimated from a pilot study we carried out with 8 subjects (4 subjects with non-specific
120 CLBP and 4 healthy control individuals), for a calculated effect size of partial $\eta^2 = 0.472$.
121 We performed the power analysis using F-test model of G*Power 3.1. Eleven subjects in
122 each group were deemed sufficient to detect significant differences in the onset of right
123 IO-TrA between groups with a power ($1-\beta$) of 0.8..

124 Unpaired t-tests were used to examine differences in age, height, weight, body mass
125 index (BMI), peak shoulder angle, and peak shoulder angular velocity between the
126 non-specific CLBP and control groups. The effects of group factor (non-specific CLBP and
127 control) and task factor (WLFRE, LFRE, LF, LERE, LFRF, RF, LERF and WLERF) on the
128 onsets of right and left IO-TrA activity and peak rotational torque were analyzed using
129 two-way analysis of variance (ANOVA). Where significant main effects were observed,
130 post hoc Tukey's HSD tests were performed. Significance was set at $\alpha = 0.05$. Statistical
131 analyses were performed using IBM SPSS Statistics 22 software (IBM, Chicago, IL, USA).

132

133 **3. Results**

134 There was a significant task effect on the peak rotational torque ($P < 0.05$, [effect size
135 (ES) = 0.96]), but ANOVA did not detect a significant group effect (Fig. 3). LERE and LFRF
136 were the smallest and were not significantly different from each other (Fig. 3). WLFRE and

137 LFRE were not significantly different, nor were LERF and WLERF (Fig. 3). On the other
138 hand, LERF and WLERF produced significantly greater amounts of peak rotational torques
139 than RF ($P < 0.05$, [ES = 1.51]) (Fig. 3). WLFRE and LFRE caused greater peak rotational
140 torques than LF ($P < 0.05$, [ES = 2.09]) (Fig. 3). RF and LF produced greater absolute peak
141 rotational torques than LERE and LFRF ($P < 0.05$, [ES = 4.28 - 4.58]) (Fig. 3). The peak left
142 shoulder angle in LFRF and WLERF were significantly different between groups but there
143 were no significant differences of shoulder angle in other tasks (Table 2). There were no
144 significant differences of peak shoulder angular velocity in all tasks as well as peak rotational
145 torque (Table 2)."

146 There were significant main effects of group on the onsets of right and left IO-TrA
147 activity ($P < 0.05$, [ES = 0.40, 0.50]) (Fig. 4, 5). The onsets of right IO-TrA activity were
148 delayed in the non-specific CLBP group in comparison with the control group in WLFRE,
149 LFRE, and LF ($P < 0.05$, [ES = 0.89 - 1.94]) (Fig.4). The onsets of left IO-TrA activity were
150 delayed in the non-specific CLBP group in comparison with the control group in RF, LERF,
151 and WLERF ($P < 0.05$, [ES = 1.18 - 1.48]) (Fig. 5). However, in LERE and LFRF, there are
152 not significant differences for the onsets of IO-TrA activity on both sides between groups ($P =$
153 0.09 - 0.27, [ES = 0.50 - 0.63]).

154 There were significant main effects of task on the onsets of right and left IO-TrA
155 activity ($P < 0.05$, [ES = 0.44, 0.54]). In the control group, the onsets of right IO-TrA activity

156 in WLFRE, LFRE and LF were significantly earlier than the other five tasks ($P < 0.05$, [ES =
157 1.13 - 3.77]) (Fig. 4). The onset of left IO-TrA activity in RF was significantly earlier than the
158 onsets in WLFRE, LFRE, and LF ($P < 0.05$, [ES = 1.39 - 2.25]) (Fig. 5) and the onsets of left
159 IO-TrA activity in LERF and WLFRE were significantly earlier than the onsets in WLFRE,
160 LFRE, LF, LERE, and LFRF ($P < 0.05$, [ES = 1.11 - 3.17]) (Fig. 5). In the non-specific CLBP
161 group, the onsets of right IO-TrA activity in WLFRE, LFRE, and LF tasks was significantly
162 earlier than the onsets in LERF and WLERF ($P < 0.05$, [ES = 1.15 - 1.70]) (Fig. 4). The onsets
163 of left IO-TrA activity in RF, LERF, and WLERF was significantly earlier than those WLFRE,
164 LFRE, and LF ($P < 0.05$, [ES = 1.40 - 2.34]) (Fig. 5).

165

166 **4. Discussion**

167 In a present study, we found that the onsets of IO-TrA activity were significantly
168 delayed during tasks with trunk rotational torque in the non-specific CLBP group but there
169 were no significantly differences between groups during LERE and LFRF. In addition, the
170 results of this study demonstrated that the onsets of IO-TrA activity were significantly related
171 not to the magnitude of trunk rotational torque, but to direction of trunk rotational torque.

172 To our knowledge, this is the first study to show the onsets of the deep abdominal
173 muscles activity are significantly delayed during tasks with trunk rotational torque but are not
174 significantly delayed during tasks without trunk rotational torque in the non-specific CLBP

175 group. These results supported our first hypothesis. Previous studies reported that anticipatory
176 activity of the deep abdominal muscles was delayed in subjects with non-specific CLBP
177 during RF [6, 8]. However, the onsets of IO-TrA activity during bilateral shoulder flexion
178 were reported no significant difference between subjects with non-specific CLBP and healthy
179 individuals [11]. These different results may be explained by our study's findings, as
180 neuromuscular dysfunction of the deep abdominal muscles during upper limb movement in
181 subjects with non-specific CLBP may be related to rotational torque of trunk. For example,
182 during RF, the upper limb movement generated right trunk rotational torque on the trunk in
183 acceleration phase of right shoulder flexion. Delayed onset of IO-TrA may lead to instability
184 of the lumbosacral segment for trunk rotational torque. These results suggest that feedforward
185 mechanism for trunk rotational torque is altered in the subjects with non-specific CLBP. The
186 trunk rotation has been identified as a significant risk factor for low back injury [18, 19] and
187 produces the onset of microdamage in the elements of the lumbar spine even if the amount of
188 trunk rotation is small [20, 21]. Therefore, although it is important to maintain lumbosacral
189 segmental stability for trunk rotation, the stability may be decreased in the non-specific CLBP
190 subjects due to the delayed onset of the deep abdominal muscles activity. Hodges et al. [22]
191 have investigated a cause-effect relationship between the delayed onset of the deep abdominal
192 muscles and pain. They have shown that pain produces delayed onset of transversus
193 abdominis activity and suggested that this delay appears to be a result rather than a cause of

194 pain [22]. However, these changes may increase the likelihood of chronicity of non-specific
195 low back pain because dysfunction of trunk muscles would lead to spinal instability, repeated
196 microtrauma and pain [4, 10]. The present findings suggest that clinicians need to pay
197 attention to the deep abdominal muscles activity against trunk rotational torque.

198 Our results failed to find any significant differences in the onsets of IO-TrA activity
199 between tasks with the same directional torque in the control group. This may indicate that the
200 onset of IO-TrA activity is not influenced by the magnitude of trunk rotational torque. Hodges
201 and Richardson [8] recorded the onsets of trunk muscle activity in upper limb movements at
202 three different speeds (fast, intermediate, and slow), and found that the onset of TrA and IO
203 activity at fast speeds was significantly different from the onset at slow speeds, but not
204 significantly different from the onset at intermediate speeds. They explained these results by
205 stating that the trunk muscles respond to upper limb movement above a threshold velocity or
206 postural disturbances at fast or intermediate speeds, while TrA and IO are not needed to react
207 to slow upper limb movements [8]. Therefore, because the three tasks with the same direction
208 of rotational torque in our study (WLFRE- LFRE- LF, RF-LERF-WLERF) provided enough
209 velocity or postural disturbance due to upper limb movement, it is possible that there are no
210 significant differences between the onsets of IO-TrA activity in these tasks.

211 Also, in the present study, the onsets of IO-TrA activity were directionally specific
212 not only in the control group but also in the non-specific CLBP group. Except for LERE and

213 LFRF, the contraction of the ipsilateral IO-TrA for the direction of trunk rotational torque
214 lagged behind the contralateral side. The sequences of IO-TrA recruitment were consistent
215 with previous studies indicating that left TrA and IO had earlier onset than right TrA and IO in
216 right shoulder flexion [23]. An opposite and equal torque needs to be created to maintain the
217 trunk position of horizontal plane during tasks with trunk rotational torque. Morris et al. [12]
218 and Allison et al. [23] suggested that the TrA asymmetrically contracted to balance the
219 rotational torque generated by rapid upper limb movement in healthy subjects. The present
220 study suggests that the deep abdominal muscles activity in the subjects with non-specific
221 CLBP is also specific to the direction.

222 In this study, there were significant differences of left shoulder angle in LFRF and
223 WLERF. However, there were no significant differences of left shoulder angle in WFRE,
224 LFRE and LF which had the significant delayed onset of right IO-TrA activity. The onsets of
225 IO-TrA activity delayed in non-specific CLBP group were observed during tasks which had no
226 significant differences of shoulder angle between groups. Therefore, these significant
227 differences of left shoulder angle would have little effect on the results in this study.

228 There were limitations to this study. First, EMG recordings were made from the
229 surface EMG system, and this system may pick up other muscular activity. However, a
230 previous study showed that the use of surface EMG to measure IO-TrA activity could acquire
231 data independent of other trunk muscle activity [13]. Second, we hypothesized from a

232 previous study that WLERF and WLFRE had increased trunk rotational torque compared with
233 other tasks [12]. However, there were nonsignificant differences between peak rotational
234 torque in WLFRE and LFRE as well as in LERF and WLERF. In this study, the acceleration
235 of the center of mass in each segment was used for calculation of trunk rotational torque. In a
236 later analysis, we clarified that accelerations of the forearm and hand in WLFRE and WLERF
237 were significantly decreased compared with LFRE and LERF. The nonsignificant findings of
238 peak rotational torque could be explained by the delayed accelerations of the forearm and
239 hand in WLFRE and WLERF with a 1 kg weight. Third, the subjects with non-specific CLBP
240 in this study had relatively mild symptoms. The average ODI score in Japanese people in their
241 20s who have low back pain who were unable to engage in routine work was reported to be
242 15.86 [24]. In this study, which recruited subjects in a similar generation, the average ODI
243 score was 13.0 (SD 6.6). In other words, the subjects in our non-specific CLBP group would
244 be regarded as having mild non-specific CLBP. Therefore, the results of this study may be
245 generalizable to subjects with mild non-specific CLBP and may be related to aggravation of
246 non-specific CLBP.

247

248 **5. Conclusion**

249 The onsets of IO-TrA activities in subjects with non-specific CLBP were delayed
250 during tasks with rotational torque of the trunk in the opposite direction, suggesting a

251 possibility that delayed onset of the deep abdominal muscles during rotational torque of the
252 trunk might be one of the characteristics of CLBP.

253

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321 **Figure captions**

322 Fig. 1. The upper limb movements of 8 types were performed by all subjects.

323

324 Fig. 2. An example of onset of right deltoid (top) and left IO-TrA (bottom) during a trial of
325 LERF in control group. The dotted line means onset of right deltoid. The thick vertical line
326 means onset of left IO-TrA.

327

328 Fig. 3. The peak trunk rotational torque (mean and SD) for the 8 rapid upper limb movements
329 in the control group and non-specific CLBP group. Positive value means right trunk rotational
330 torque and negative value means left trunk rotational torque.

331

332 Fig. 4. Onset latency (\pm SD) of right IO-TrA in non-specific CLBP and control group.

333 * indicates significant differences between groups.

334 † indicates significantly earlier than LERE, LFRE, RF, LERF, and WLRF.

335 ‡ indicates significantly earlier than LERF and WLRF.

336

337 Fig. 5. Onset latency (\pm SD) of left IO-TrA in non-specific CLBP and control group.

338 * indicates significant differences between groups.

339 † indicates significantly earlier than WLFR, LFRE, LF, LERE and LFRE.

340 ‡ indicates significantly earlier than WLFRE, LFRE, LF.

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1 Figures

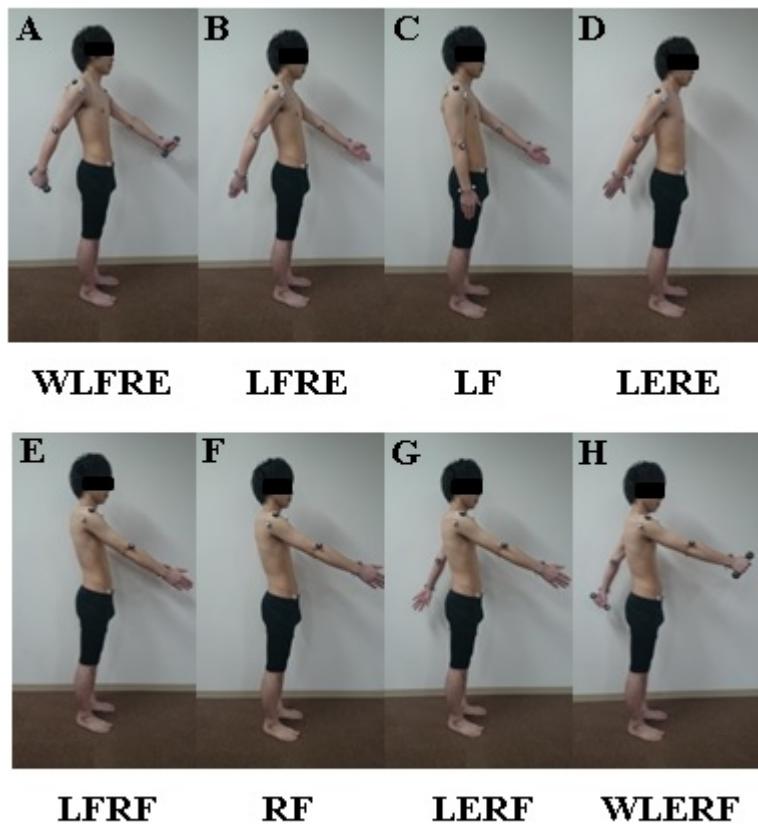


Fig. 1. The upper limb movements of 8 types
were performed by all subjects.

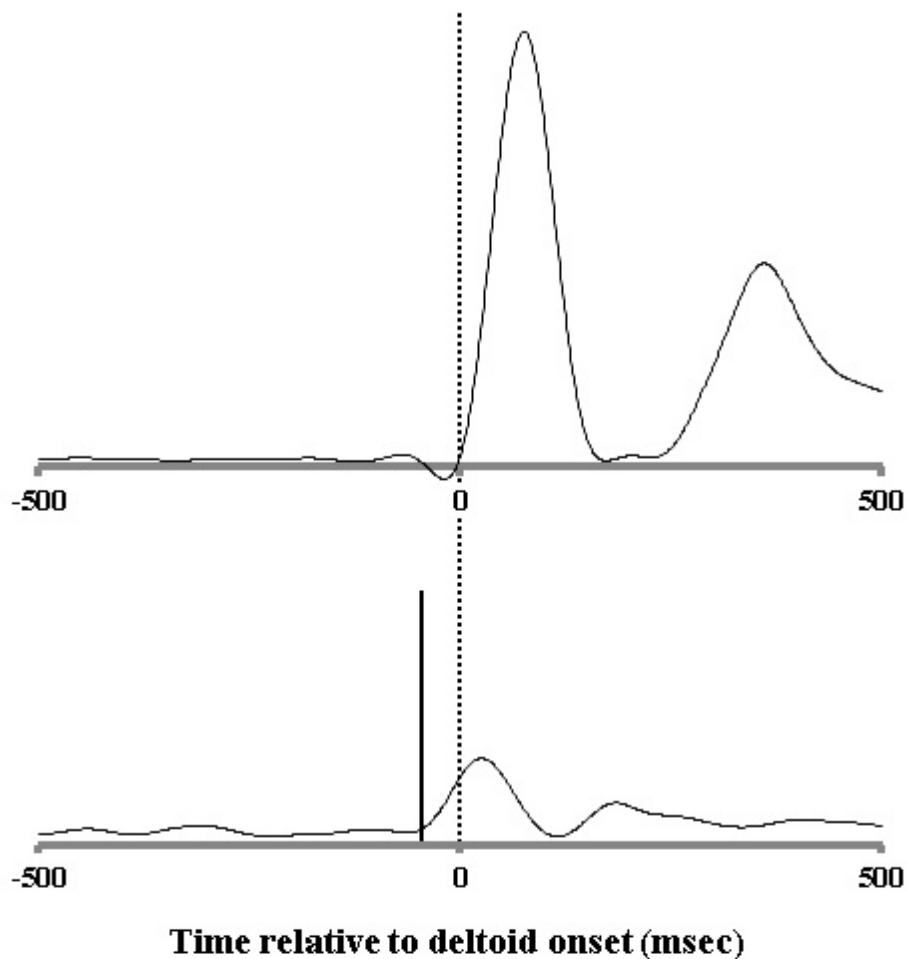


Fig. 2. An example of onset of right deltoid (top) and left IO-TrA (bottom) during a trial of LERF in control group. The dotted line means onset of right deltoid. The thick vertical line means onset of left IO-TrA.

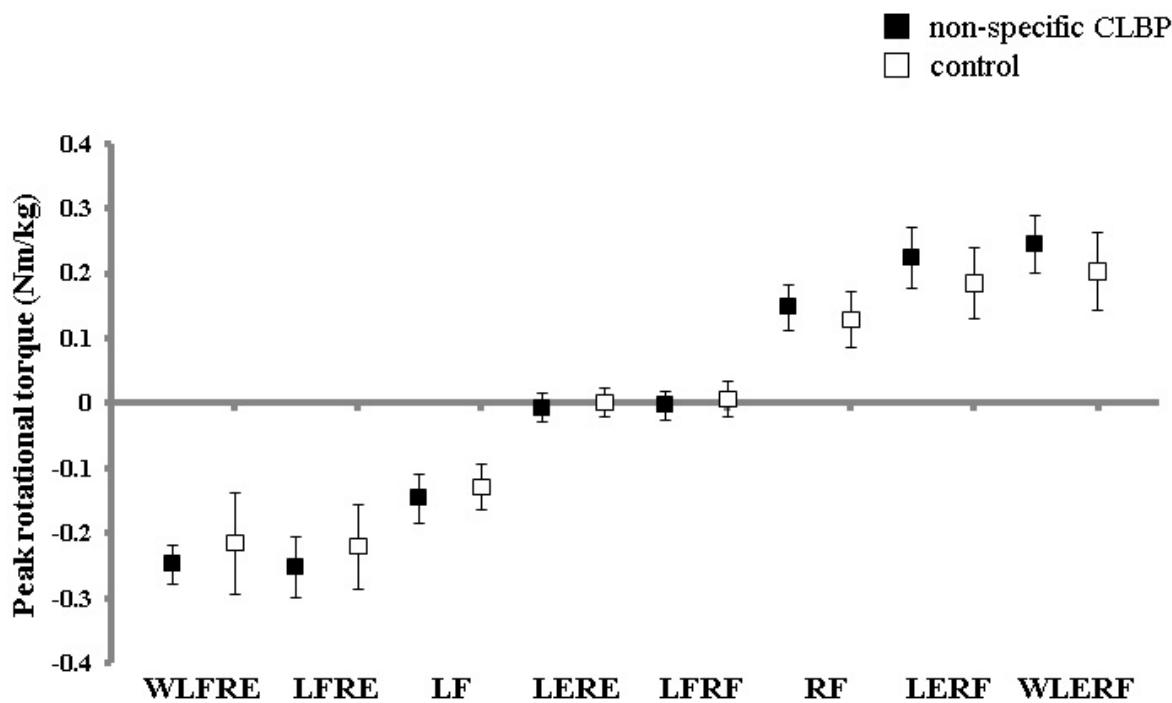


Fig. 3. The peak trunk rotational torque (mean and SD) for the 8 rapid upper limb movements in the control group and non-specific CLBP group. Positive value means right trunk rotational torque and negative value means left trunk rotational torque.

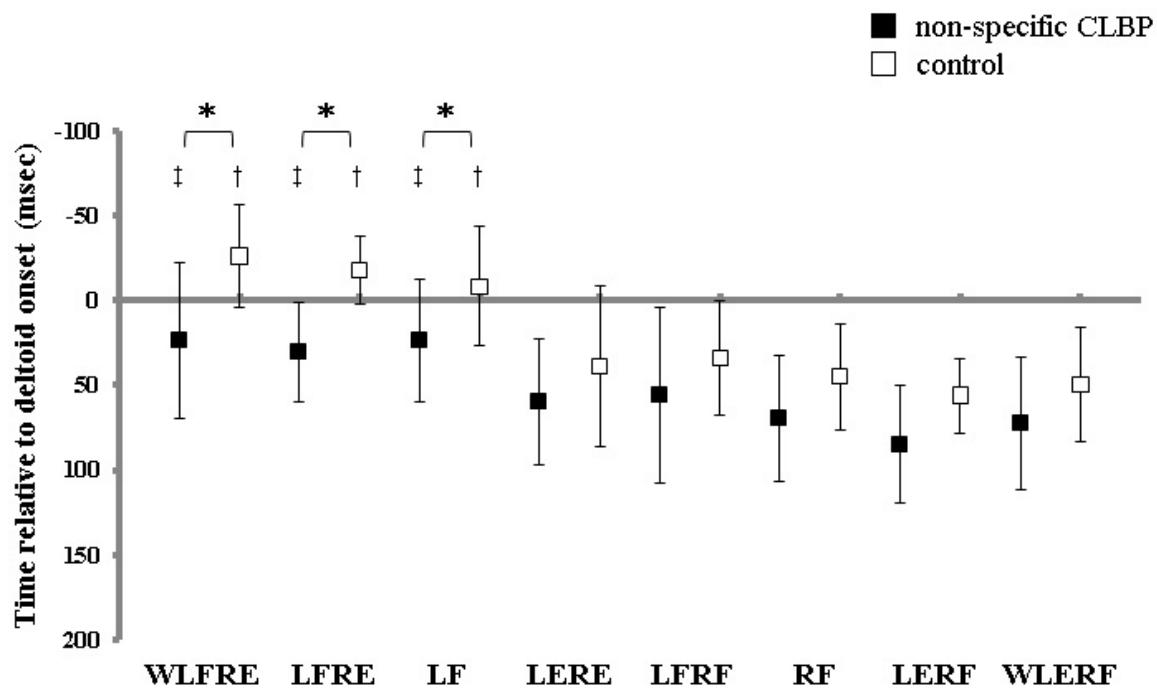


Fig. 4. Onset latency (\pm SD) of right IO-TrA in non-specific CLBP and control group.

* indicates significant differences between groups.

† indicates significantly earlier than LERE, LFRF, RF, LERF, and WLERF.

‡ indicates significantly earlier than LERF and WLERF.

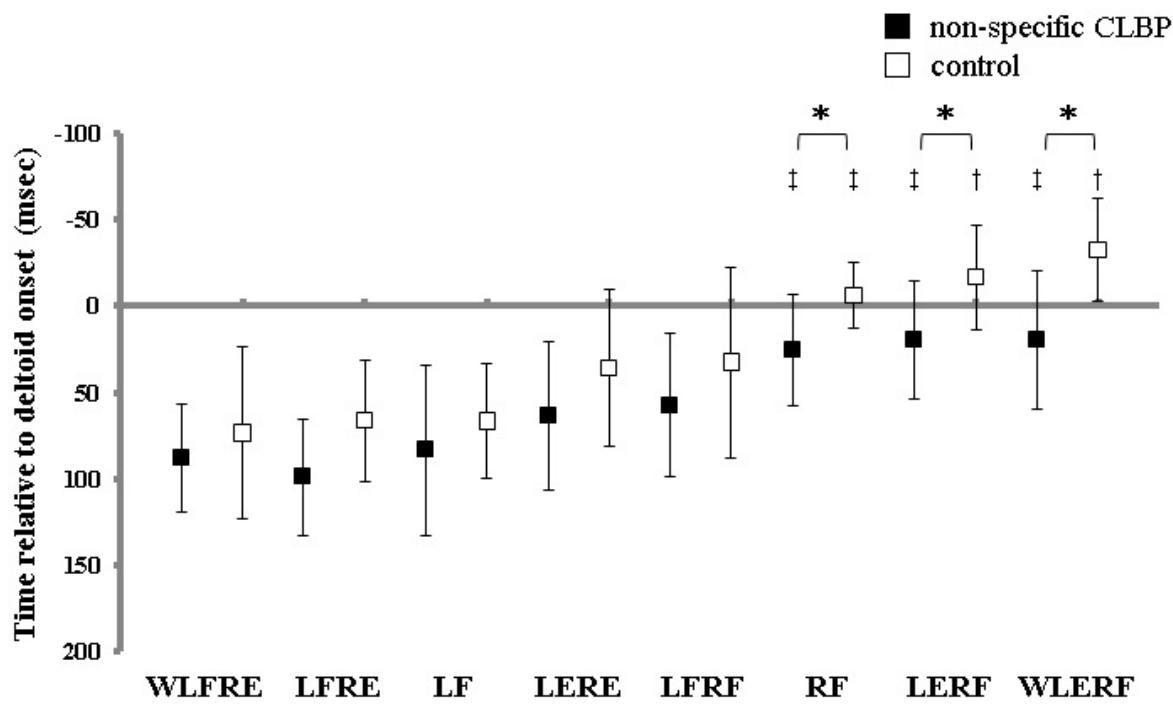


Fig. 5. Onset latency (\pm SD) of left IO-TrA in non-specific CLBP and control group.

* indicates significant differences between groups.

† indicates significantly earlier than WLFRE, LFRE, LF, LERE and LFRF.

‡ indicates significantly earlier than WLFRE, LFRE, LF.

1 **Tables**

2 Table 1. Subject characteristics

	non-specific CLBP	control	P value
Participants (n)	12	13	-
Gender (m : w)	9:3	7:6	-
Age (years)	21.2 (2.2)	21.5 (1.5)	0.506
Height (cm)	168.3 (8.3)	167.2 (9.6)	0.754
Weight (kg)	60.2 (8.2)	57.5 (9.3)	0.448
BMI (kg/m^2)	21.2 (2.2)	20.5 (1.4)	0.294
VAS (%)	36.7 (25.1)	-	-
ODI (%)	13.0 (6.6)	-	-

3 Abbreviations: CLBP, chronic low back pain. BMI, body mass index.

4 VAS, visual analog scale. ODI, oswestry disability index

5 Values expressed as mean (standard deviation).

7 Table 2. Peak shoulder angle and peak shoulder angular velocity during each task.

Task	Side	Peak shoulder angle (deg)			Peak shoulder angular velocity (deg/sec)		
		non-specific CLBP	Control	P value	non-specific CLBP	Control	P value
WLFRE	Rt	47.5 (6.4)	48.4 (8.1)	0.758	184.3 (32.5)	199.2 (37.7)	0.311
	Lt	57.8 (6.7)	60.3 (9.6)	0.469	264.7 (33.4)	253.1 (34.8)	0.416
LFRE	Rt	46.9 (6.0)	47.1 (8.9)	0.942	258.6 (38.5)	261.5 (52.9)	0.877
	Lt	56.1 (7.5)	59.7 (7.9)	0.273	331.7 (45.1)	331.1 (56.7)	0.981
LF	Rt	-	-	-	-	-	-
	Lt	55.7 (5.2)	61.7 (9.7)	0.073	333.1 (42.1)	341.4 (57.7)	0.692
LERE	Rt	52.9 (9.0)	51.4 (6.4)	0.638	252.6 (72.3)	224.8 (101.5)	0.448
	Lt	54.0 (8.6)	50.7 (5.8)	0.284	286.5 (39.7)	270.5 (39.6)	0.333
LFRF	Rt	54.3 (6.7)	58.6 (8.9)	0.197	309.5 (38.3)	322.3 (40.9)	0.434
	Lt	54.2 (4.8)	60.5 (7.9)	*0.027	309.9 (36.5)	326.7 (41.6)	0.304
RF	Rt	57.3 (7.0)	58.9 (10.2)	0.656	340.7 (52.2)	341.3 (66.0)	0.981
	Lt	-	-	-	-	-	-
LERF	Rt	57.8 (4.8)	57.9 (14.2)	0.986	330.3 (46.1)	312.0 (61.8)	0.418
	Lt	48.7 (9.2)	47.0 (10.2)	0.657	263.5 (43.5)	248.0 (44.7)	0.398
WLERF	Rt	58.6 (6.8)	59.2 (11.2)	0.880	262.9 (44.6)	258.6 (43.8)	0.814
	Lt	50.8 (9.8)	42.9 (7.2)	*0.035	200.4 (57.9)	176.7 (33.6)	0.232

8 Abbreviations: CLBP, chronic low back pain. Rt, right. Lt, left.

9 Values expressed as mean (standard deviation).

10 * indicates significant difference in peak shoulder angle between groups.