



Bilateral experimental neck pain reorganize axioscapular muscle coordination and pain sensitivity

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Published in:
European Journal of Pain

DOI (link to publication from Publisher):
[10.1002/ejp.972](https://doi.org/10.1002/ejp.972)

Publication date:
2017

Document Version
Accepted author manuscript, peer reviewed version

[Link to publication from Aalborg University](#)

Citation for published version (APA):
Christensen, S. W., Hirata, R. P., & Graven-Nielsen, T. (2017). Bilateral experimental neck pain reorganize axioscapular muscle coordination and pain sensitivity. *European Journal of Pain*, 21(4), 681-691.
<https://doi.org/10.1002/ejp.972>

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1 **BILATERAL EXPERIMENTAL NECK PAIN REORGANISE AXIOSCAPULAR MUSCLE**
2 **COORDINATION AND PAIN SENSITIVITY**
3

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9 **Original article** for European Journal of Pain

10 **Running title:** *Axioscapular motor control during experimental neck pain*

11
12 **Funding source:** The Research Foundation of the Danish Physiotherapists Association supported
13 the study. Center for Neuroplasticity and Pain (CNAP) is supported by the Danish National
14 Research Foundation (DNRF121).
15
16

17 **What's known about the topic and what does this study add?**

- 18 • Bilateral clinical neck pain alters axioscapular muscle coordination but only effects of
19 unilateral experimental neck pain has been investigated.
- 20 • Bilateral experimental neck pain causes task-dependent reorganised axioscapular and trunk
21 muscle activity in addition to widespread decrease in pressure pain sensitivity.
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25 **Conflict of interest:** The authors have no conflicts of interests.
26

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1 **ABSTRACT**

2 *Background* Neck pain is a large clinical problem where reorganised trunk and axioscapular muscle
3 activities have been hypothesised contributing to pain persistence and pain hypersensitivity. This
4 study investigated the effects of bilateral experimental neck pain on trunk and axioscapular muscle
5 function and pain sensitivity.

6 *Methods* In 25 healthy volunteers, bilateral experimental neck pain was induced in the splenius
7 capitis muscles by hypertonic saline injections. Isotonic saline was used as control. In sitting,
8 subjects performed slow, fast, and slow-resisted unilateral arm movements before, during, and after
9 injections. Electromyography (EMG) was recorded from eight shoulder and trunk muscles
10 bilaterally. Pressure pain thresholds (PPTs) were assessed bilaterally at the neck, head, and arm.
11 Data was normalised to the before-measures.

12 *Results* Compared with control and post measurements, experimental neck pain caused (i)
13 decreased EMG activity of the ipsilateral upper trapezius muscles during all but slow-resisted down
14 movements ($P<0.001$), and (ii) increased EMG activity in the ipsilateral erector spinae muscle
15 during slow and fast movements ($P<0.02$), and in the contralateral erector spinae muscle during all
16 but fast-up and slow-resisted down movements ($P<0.007$). The PPTs in the painful condition
17 increased at the head and arm compared with post measurements and the control condition
18 ($P<0.001$). In the post-pain condition, the neck PPT was decreased compared with the control
19 condition ($P<0.001$).

20 *Conclusion* Acute bilateral neck pain reorganised axioscapular and trunk muscle activity together
21 with local hyperalgesia and widespread hypoalgesia indicating that acute neck pain immediately
22 affects trunk and axioscapular function which may affect both assessment and treatment.

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1 INTRODUCTION

2 Neck pain is a major problem in the general population (Fejer et al., 2006) with multiple symptoms
3 and involved structures (Bogduk 2011). Developing effective treatment strategies requests an
4 improved understanding of the mechanisms behind neck pain (Michaleff et al., 2014). So far, the
5 main focus has been directed towards the painful neck area.

6 Based on abnormal axioscapular muscle function in neck pain patients, dysfunctional
7 shoulder girdles have been suggested as an important factor in persistent neck pain (Behrsin and
8 Maguire 1986; O'Leary et al., 2009) although it is not known if it is an effect of neck pain or a
9 causal factor for neck pain. Emerging studies show altered alignment of the shoulder girdle in neck
10 pain patients, displaying protracted shoulders and a forward head posture in rest (Helgadottir et al.,
11 2011b) and during functional tasks (Helgadottir et al., 2010). Although electromyographic studies
12 during upper limb tasks reported reorganised axioscapular muscle activity in neck pain patients
13 compared with healthy controls, the results are conflicting. In neck pain patients one study found
14 reduced activity of the upper trapezius muscle during arm movement (Falla et al., 2004) whereas
15 another study found no change in activity of the upper trapezius muscle but a task dependent
16 increase in the lower trapezius muscle during isometric contractions (Zakharova-Luneva et al.,
17 2012). Wegner et al. (2010) found reduced activity in the lower trapezius muscle and increased
18 activity of the middle trapezius muscle during a typing task in neck pain patients compared with
19 controls while others reported no changes for the trapezius muscle but delayed onset and reduced
20 duration of the serratus anterior muscle activity during arm movement (Helgadottir et al., 2011a).
21 Different tasks or heterogeneous patient groups may explain the previous contrasting findings.
22 Clinical confounding factors have been eliminated in studies using experimental neck pain where
23 saline-induced neck pain in healthy subjects demonstrate reduced upper trapezius muscle activity
24 during arm movement (Christensen et al., 2015; Falla et al., 2007) and increased trunk muscle
25 activity (Christensen et al., 2015). Since neck pain patients often present with bilateral pain
26 (Fernandez-de-las-Penas et al., 2008) the unilateral experimental pain models may not be sufficient.

27 Hyperalgesia to pressure in the neck region has been reported for acute and chronic neck pain
28 compared with healthy controls (La Touche et al., 2010; Scott et al., 2005) while widespread
29 hyperalgesia were only reported for chronic neck pain (Javanshir et al., 2010). Experimental neck
30 pain did not cause local hyperalgesia after a unilateral injection of hypertonic saline into the
31 trapezius muscles (Ge et al., 2003; Schmidt-Hansen et al., 2006) but after bilateral injections,
32 hypoalgesia due to descending pain modulation were observed outside the injected areas (Ge et al.,
33 2003).

34 This study aimed to investigate the effects of bilateral experimental neck pain on axioscapular
35 muscles coordination during standardised arm movements as well as the pressure pain sensitivity.

1 Experimental neck pain was expected to reorganise axioscapular and trunk muscle activity and
2 cause hypoalgesia to pressure away from the painful area.

5 **MATERIALS AND METHODS**

6 *Subjects*

7 Twenty-five healthy volunteers were recruited from a university setting (13 women, one left-
8 handed). Women had a mean age of 24.4 ± 3.4 years (\pm standard deviation) and a mean body mass
9 index (BMI) of 21.3 ± 2.1 kg/m². For men, the mean age was 24.3 ± 3.0 years and mean BMI was
10 23.6 ± 2.5 kg/m². Exclusion criteria were persistent or recurring neck or shoulder pain within the
11 past year, deviations in spinal posture such as significant scoliosis, kyphosis, or forward head
12 posture. In addition, any signs or symptoms indicating rheumatologic or neurological disorders that
13 may influence the outcome of the study, use of pain medication, or pregnancy were cause of
14 exclusion. Subjects were given written and verbal information about the study after which informed
15 consent was obtained. The study was approved by the local ethics committee (N20120018) and
16 performed according with the Helsinki declaration.

18 *Experimental protocol*

19 The study used a single blinded randomised crossover design (Fig. 1) with data collection
20 performed in a single session. The muscle activity was assessed by electromyography (EMG)
21 recorded during arm movements where subjects were seated in an upright position (Fig.2) while the
22 pressure pain thresholds (PPTs) were measured with subjects leaning over a bench. All
23 measurements were performed bilaterally, starting with the EMG measurements, and were done
24 before (baseline), during (immediately after experimental neck pain was induced by bilateral
25 injections of hypertonic saline into the splenius capitis muscles or the control injections by isotonic
26 saline), and after (i.e. 5 min after any potential pain had vanished). A 10 min. pause was included
27 between the post measurements after the first series of injections (e.g. hypertonic saline) and the
28 baseline before the second series of injections (e.g. isotonic saline). The sequence of experimental
29 pain and control sessions was randomised in a balanced way with participants a priori blinded to the
30 sequence.

32 *Experimental neck pain*

33 The splenius capitis muscle was identified between the posterior border of the sternocleidomastoid
34 muscle and the lateral border of the upper trapezius muscle using ultrasonography (Logiq S7 Expert
35 mounted with a ML6-15L transducer; GE Medical Systems, Milwaukee, Wisconsin, USA). A

1 hypodermic needle was inserted into the splenius capitis muscle and the initial injection of
2 hypertonic saline (5.8%, 0.75 ml) was injected. The time between the bilateral injections was
3 approximately 45 s and to compensate for this delay the pain duration of the first injection was
4 prolonged by a larger volume (Graven-Nielsen et al., 1997) than the subsequent injection (0.5 ml).
5 This novel approach ensured that bilateral pain was kept during the experimental session and
6 avoided that pain in one side would be fading away before the other during the recordings. For the
7 control condition, isotonic saline (0.9%) was used with volumes of 0.75 ml and 0.5 ml for the two
8 sequential injections. The side of the initial injection was randomised in a balanced way for both
9 injection types.

10 The pain intensity profile was recorded using a 10 cm electronic visual analogue scale (VAS)
11 labelled with “no pain” (0 cm) and “maximum pain” (10 cm). Participants were continuously
12 reminded to update the VAS throughout the experiment. Peak, duration and area under the VAS-
13 time curve were extracted for further analysis. The duration of pain was defined as the time
14 difference between the first and last VAS score exceeding 0.1 cm and was defined as 0 s if the VAS
15 remained zero. Subjects drew areas of perceived pain on a body chart and the pain areas were
16 extracted (VistaMetrix v.1.38.0, SkillCrest, LLC) in arbitrary units (a.u.).

17

18 *Standardised arm movements*

19 To ensure a standardised arm movement, subjects sat on a custom built chair, supporting only the
20 sacrum. The starting position was an upright sitting position with arms hanging relaxed by the side
21 and feet flat on the floor. Adjustable walls, angled 30° from the frontal plane, allowing movement
22 in the scapular plane (scaption), were placed on the side of the chair; Subjects were asked to keep
23 the back of their hand in contact with the wall at all times during movements. Movements were
24 done with outstretched arm and thumbs pointing up. A physical upper marker was placed on the
25 vertical surface allowing for abduction to 140°. Arm movements were performed bilaterally,
26 alternating between sides, with a 6 s break in-between. To guide when the movement should 1)
27 start, 2) be at the upper marker, and 3) returned to the resting position, a custom made program
28 (Aalborg University, DK) was set to make 3 beep cues separated by 3 s thereby giving a 3 s
29 window for both up and down movement without any breaks at the upper marker. Each movement
30 series was initiated with 3 slow movements in each side, followed by 3 fast movements in each
31 side. During fast movements, subjects were instructed to move the arm as fast as possible from the
32 starting position to the upper marker. The down movements for the fast movements were not
33 recorded. Immediately after the fast movements another slow resisted movement series (3 in each
34 side) was conducted with the addition of a 1 kg wrist cuff attached to each arm. The total movement

1 series, consisting of slow, fast and resisted movements in both side, lasted 3.5 min. Subjects were
2 reminded to keep an upright posture and this was visually inspected throughout the study.

3 Accelerometers (ACC; EVAL-ADXL327Z; Analog Devices, Norwood, Massachusetts, USA)
4 placed over the lateral epicondyle of each arm were used to monitor the timing of each movement.
5 For the slow movements, time parameters were extracted showing the time from the first cue (i.e.
6 start) to maximum angle and from maximum angle to the third cue (i.e. return position) while for
7 the fast movements only the time from the first cue to maximum angle was recorded. For the
8 accelerometer analysis an average of the 3 movements for each movement type (slow up, slow
9 down, fast up, resisted slow up, resisted slow down) was used.

10 Participants rated the perceived difficultness of the arm movements on a 6-point Likert scale
11 after each movement series (0: “no problems”, 1: “minimally difficult”, 2: “somewhat difficult”, 3:
12 “fairly difficult”, 4: “very difficult” 5: “unable to perform”).

13

14 *Electromyography recordings*

15 Eight muscles were assessed bilaterally with surface EMG: Serratus anterior (SA), upper trapezius
16 (UT), middle trapezius (MT), lower trapezius (LT), anterior deltoid (AD), middle deltoid (MD),
17 obliquus externus (OE), and erector spinae (ES) muscles. Adhesive bipolar surface electrodes
18 (Neuroline 72001-k; AMBU, Denmark) were mounted pairwise on the skin which had been
19 prepared according to the SENIAM recommendations (Hermens et al., 2000). A ground electrode
20 (OT Bioelettronica, Italy) was mounted on the right wrist. The EMG recordings were amplified
21 (gain 500) and sampled at 2048 Hz (OT Bioelettronica, Italy). The raw EMG signal was rectified
22 and filtered (Butterworth 2nd order, band pass 25-450Hz) in matlab (R2012a; The MathWorks Inc.,
23 Natick, MA). Root mean square (RMS) EMG values from 2 epochs of 3 seconds (i.e. between
24 cues), representing slow up and down movements were extracted for further analysis. For the fast
25 up movements the epochs were defined as the time from the first cue to the maximum angle (based
26 on the accelerometer data). The average RMS-EMG values from the 3 movement repetitions from
27 the respective series were used for further analysis and normalised to baseline (100%).

28

29 *Pressure algometry*

30 A 1-cm² probe enclosed by a disposable latex sheet was mounted on a handheld pressure algometer
31 (Somedic, Hörby, Sweden). A steady increasing pressure was applied at a rate of 30 kPa/s. The
32 pressure pain threshold (PPT) was defined, as the time point where the pressure was first perceived
33 as painful. When the PPT was reached, participants were instructed to push a button in order to
34 record the specific pressure at the time point.

1 Bilateral PPTs (six in total) were measured above 1) the injection site, over the splenius
2 capitis muscle (neck), 2) over the middle part of the temporalis muscle (head) (Kasch et al., 2001),
3 and 3) above the extensor carpi radialis brevis (arm) muscle (Slater et al., 2005). The PPT was
4 always assessed first at the side of the first injection, starting proximal and moving distal before
5 assessing the contralateral side. This was done three times, giving an interval of approximately 25 s
6 before re-assessing the same site again. An average of the three values for each site was used for
7 further analysis and normalised to baseline (100%).

8 9 *Statistics*

10 Data are presented as mean and standard error of the mean (SEM). A Wilcoxon test was used to
11 compare pain areas, VAS parameters and Likert scores after the two injections. Accelerometer, PPT
12 and EMG data was inspected using QQ plots and log-transformed (\log_{10}) if not normally
13 distributed (RMS-EMG) before two steps were taken to analyse data: i) a comparison of baselines
14 (one before each injection) before data was normalised to baseline ii) analysis of the normalised
15 data.

16 For accelerometer, PPT, and RMS-EMG the baseline recordings were compared separately
17 for each movement type using a repeated-measure analysis of variance (RM-ANOVA) with *session*
18 (baseline recordings before hypertonic and isotonic saline injections) along with *site* (PPT sites [3];
19 EMG recording sites [16 muscles]) where relevant. Data was then normalised to baseline (100%)
20 and analysed using a RM-ANOVA with *time* (during, after) and *saline* (hypertonic or isotonic)
21 along with *side* (left or right) as within factors for each movement type in order to investigate
22 *saline*time*side* or *saline*time* interactions. To compensate for the use of multiple ANOVAs in the
23 analysis of EMG data (16 muscles) the P-value for ANOVA effects was Bonferroni corrected to
24 $P < 0.0031$ (i.e. $0.05/16$). The Newman-Keuls post-hoc test adjusting for multiple comparisons was
25 applied if a significant interaction was detected in the RM-ANOVA or ANOVA. Significance level
26 was set at a P-value of 0.05. Analysis was carried out using STATISTICA 10 (StatSoft Inc., Tulsa,
27 USA).

28 29 30 **RESULTS**

31 *Experimental neck pain*

32 Injection of hypertonic saline compared with isotonic saline caused higher VAS peaks (5.7 ± 0.4 cm
33 vs. 0.6 ± 0.2 cm, $P < 0.001$), longer duration (597.6 ± 53.4 s vs. 52.3 ± 14.7 s, $P < 0.001$), and larger
34 area under the VAS-time curve (1524.4 ± 188.8 cm·s vs. 65.6 ± 19.8 cm·s, $P < 0.001$). One subject
35 indicated higher pain intensity on the side of the initial injection of hypertonic saline while the

1 remaining subjects felt no difference. The hypertonic saline also caused larger perceived area of
2 pain in posterior (1.149 a.u. vs. 0.113 a.u., $P<0.001$) and side view (0.204 a.u. vs. 0.001 a.u.,
3 $P=0.011$). The saline-induced pain generally covered an area extending from the level of the
4 external occipital protuberance and down to the level of the spinous process of Th3 (Fig. 3). For the
5 upper pain area, it extended to the side of the neck while one subject felt it extending to the
6 temporal region. For the lower pain area, the lateral border reached the level of the acromio-
7 clavicular joint.

8

9 *Performance of arm movements*

10 Due to technical problems, accelerometer data from two participants were discarded. Average
11 timings for slow movements in the two baseline recordings from the first cue signal to maximum
12 angle and from maximum angle to the last cue signal were 3.16 ± 0.02 s and 2.84 ± 0.02 s for up
13 and down movements, respectively. For the resisted slow movements the same parameters were
14 3.07 ± 0.02 s and 2.93 ± 0.02 s. During the fast movements the time recorded from the cue signal to
15 the maximum angle was on average 1.04 ± 0.02 s. No significant timing differences were found
16 between baseline recordings or between the normalised data for the isotonic/hypertonic conditions
17 and post measurements.

18 During the painful session, 24% of the participants felt an increase in the perceived
19 difficulty of lifting the arm indicated by a Likert score of 1 or above whereas all subjects scored
20 0 for the non-painful session ($P=0.027$).

21

22 *Muscle activity during baseline measurements*

23 Average RMS-EMG for the two baseline recordings during fast, slow, and slow-resisted
24 movements are presented in supplementary material (Fig. S6). Comparing RMS-EMG in the two
25 baseline sessions (before each injection) for the different movement types (slow up & down, fast
26 up, and slow-resisted up & down movements) found one significant interaction between sessions
27 and side when lowering the arm during slow movements (RM-ANOVA: $F[15,73] = 1.7$, $P=0.032$);
28 post-hoc test revealed that the baseline RMS-EMG for the lower trapezius muscle contralateral to
29 arm movement for the control condition was increased (by $15.8 \pm 1.9\%$) compared with the baseline
30 for the painful condition (NK: $P<0.001$).

31

32 *Muscle activity during painful slow movements*

33 The interactions between saline, time and side in the RM-ANOVAs of RMS-EMG were all non-
34 significant whereas Table S1 presents all saline*time interactions. Significant interactions for the
35 normalised RMS-EMG is shown in Fig. 4.

1 For the upper trapezius muscle on the side of movement (ipsilateral) an interaction between
2 saline and time was found with the post-hoc test revealing decreased RMS-EMG activity during the
3 painful condition when compared with both post and control conditions (NK: $P < 0.001$, Fig. 4a). On
4 the side of movement an increased RMS-EMG was found in the anterior deltoid muscle during the
5 painful condition when compared to the post and control conditions (NK: $P < 0.001$). For the
6 ipsilateral middle deltoid muscle, the post-hoc test revealed a reduction in the post painful condition
7 (NK: $P < 0.001$) compared with both immediately after the injection and the control condition.
8 However, for the control condition an increase was seen when compared with immediately after the
9 isotonic saline injection (NK: $P = 0.039$). In the trunk muscles, the bilateral erector spinae muscles
10 showed an increase during the painful condition when compared with post and control conditions
11 (NK: $P < 0.003$). In addition, an increase in the ipsilateral erector spinae muscles was observed for
12 the post measurement in the control condition compared with the immediately after recordings in
13 the control condition and with the post recording after the painful condition (NK: $P < 0.03$).

14 In the slow, down movement a decreased RMS-EMG activity of the upper trapezius muscle
15 during the painful condition was found when compared with post and control conditions (Fig. 4b,
16 NK: $P < 0.001$). A similar pattern, although with a smaller RMS-EMG reduction during the painful
17 condition, was seen for the middle trapezius muscle (NK: $P < 0.001$). The bilateral erector spinae
18 muscles showed an increase in RMS-EMG during the painful condition compared to the post
19 recording and control condition (NK: $P < 0.002$).

20

21 *Muscle activity during painful fast up movements*

22 Reduced RMS-EMG was found in the side of movements during the painful condition when
23 compared with the post and control condition for the upper and middle trapezius muscles (Fig. 4e
24 NK: $P < 0.001$). A decrease in RMS-EMG was also found during the painful condition for the
25 contralateral upper trapezius muscle when comparing this to the post recording and control
26 condition (Fig. 4c, NK: $P < 0.001$). The ipsilateral erector spinae muscle showed an increase in
27 RMS-EMG during the painful condition compared with post and control conditions in addition to
28 an increase in the post measurement after the control injections of isotonic saline (NK: $P < 0.02$).

29

30 *Muscle activity during slow resisted movements*

31 Data from one participant for the upper trapezius had to be discarded due to technical problems. For
32 the slow, resisted up movement a decreased RMS-EMG was found for the ipsilateral upper
33 trapezius muscles during the painful condition compared with post and control measurements (Fig.
34 4c; NK: $P < 0.001$). For the erector spinae muscle a bilateral increased RMS-EMG was found during
35 the painful condition compared with the post measurement (NK: $P < 0.002$) although only

1 significantly different from the control condition in the contralateral side (NK: $P < 0.001$). However
2 for the ipsilateral side the post RMS-EMG measurement in the erector spinae muscle was decreased
3 compared with the post measurement of the control condition (NK: $P = 0.010$).

4 For the slow, resisted down movements no significant differences were detected.

6 *Pressure algometry*

7 The PPT at the neck site was higher (RM-ANOVA: $F[2,98] = 5.6$, $P = 0.004$; NK: $P = 0.018$) in the
8 baseline recordings before the hypertonic injection (214 ± 8 kPa) compared with the baseline before
9 the isotonic injections (199 ± 11 kPa). For the head (356 ± 8 kPa) and arm (371 ± 9 kPa) sites such
10 differences were not found.

11 No interactions was found for the saline, time and side analysis for the normalised PPTs
12 however, a saline and time interaction (Fig. 5) was found for the neck site (RM-ANOVA: $F[1,48] =$
13 53.0 , $P < 0.001$) with post-hoc testing revealing a drop in PPTs in the post-pain condition compared
14 with all other recordings for this site (NK: $P < 0.001$). Increased PPT was observed during
15 experimental pain compared with post and control measurements for both the head (RM-ANOVA
16 $F[1,48] = 7.2$, $P = 0.009$; NK: $P < 0.001$) and arm sites (ANOVA: $F[1,48] = 11.1$, $P = 0.001$; NK:
17 $P < 0.001$).

20 **DISCUSSION**

21 This study demonstrated that acute bilateral experimental neck pain reorganised the activity of
22 axioscapular and trunk muscles simultaneously with increased perceived difficulty of lifting the
23 arm. Additionally, the painful condition caused widespread hypoalgesia as well as localized
24 hyperalgesia.

26 *Performance of arm movements*

27 No timing differences were found between the different movement types although performance of
28 movements were perceived as more difficult during the painful condition, indicating that the
29 observed RMS-EMG changes may cause the altered perception. However, the RMS-EMG for the
30 contralateral lower trapezius, during the slow unresisted down movements, was 15.8% lower during
31 baseline before the painful condition compared to the baseline before the control condition.

32 Although significant, this difference was not thought to have influenced the results since data was
33 normalized to baseline and therefore adjusting for this discrepancy. Although one subject felt more
34 pain in the side of the first injection, the injection sequence did probably not affect the current
35 results since the side of first injection with respect to arm movement was randomized.

1
2 *Axioscapular muscle activity during neck pain*
3 During experimental neck pain a consistent reduction in muscle activity was found for the
4 ipsilateral upper trapezius muscle, which was present during all but the slow resisted down
5 movement. Based on a similar protocol, unilateral experimental pain reduced activity of
6 approximately 12% in the upper trapezius muscle activity (Christensen et al., 2015) whereas the
7 present study, bilateral pain caused a drop of approximately 20%. Another finding supporting a
8 larger impact on the motor system by bilateral neck pain is the significant adaptations seen for the
9 trapezius and erector spinae muscles during the fast movements (Fig. 4e) in the present study which
10 was not found in the study by Christensen et al. (2015). In line with the present study, only bilateral,
11 and not unilateral knee-related pain, caused alterations in the EMG activity of leg muscles during
12 standing (Hirata et al., 2012). Interestingly, when assessing the different movements, the amount of
13 reduced EMG activity for the ipsilateral upper trapezius muscle varied, with the resisted movement
14 showing the least decrease in muscle activity, indicating that changes may be task dependent
15 (Wegner et al., 2010). The activity in the other muscles during both slow resisted and fast up
16 movements (Fig. 4c & e) were less effected by pain, displaying smaller increase or decrease in
17 activity, compared with the slow unresisted up movements (Fig. 4a). A similar pattern, with less
18 effect of pain, was found when comparing the slow resisted down movements with the unresisted
19 down movements (Fig. 4b & d). The added weight for the resisted movements or faster speed
20 probably caused recruitment of additional motor units with higher thresholds to produce the force
21 needed and may have caused less pronounced pain-adaptation effects (Hodges et al., 2008). Such
22 additional recruitment during more demanding movements, may explain the stable motor
23 performance observed in both resisted and fast movements compared to the slow unresisted
24 movements (Fig. 4). A final explanation for less reductions of axioscapular muscle activity during
25 fast or resisted painful movements may indicate that an optimal strategy for these movements had
26 already been obtained during the baseline movements and therefore a pain-related adjustment to
27 optimise or compensate the movement strategy was not possible without compromising the
28 performance.

29 A factor unaccounted for in this study is the possible internal redistribution of activity within
30 a muscle, which previously have been shown for the upper trapezius during experimental pain
31 (Falla et al., 2009; Madeleine et al., 2006). Furthermore, knowledge of the activity of the deeper
32 layers of axioscapular muscles such as levator scapula and pectorals minor muscles are warranted in
33 order to understand the complex relationship between the reorganised muscle activities while
34 moving the arm during a painful condition. The changes in axioscapular muscle activity in this
35 study and the different motor performance during different movement types may explain some of

1 the contrasting findings in patient studies studying the axioscapular muscle activity during different
2 tasks. For instance, the upper trapezius activity have been studied during different tasks where some
3 find a reduction (Falla et al., 2004) while others do not (Helgadottir et al., 2011a; Wegner et al.,
4 2010) even though similar patient groups were included. Different strategies for motor adaptations
5 of axioscapular muscle activity due to neck pain have been identified in experimental and clinical
6 studies. Although it may serve as a protective strategy, the long-term effect of such adaptations
7 could be detrimental (Hodges and Tucker 2011).

8

9 *Trunk muscle activity during neck pain*

10 The erector spinae muscles showed increased activity bilaterally during all but the fast up painful
11 and slow resisted down movements, with the maximal mean increases of approximately 25% during
12 the painful condition. If the increased trunk muscle activity was only found contralateral it could be
13 argued that participants used a strategy with lateral flexion of the trunk to help lifting the arm.
14 However, the additional increased ipsilateral erector spinae muscle activity would counteract such
15 approach and it therefore seems as an unlikely strategy. Nonetheless, altered trunk movement
16 during pain cannot be ruled out without movement analysis, which was not used in the present
17 study. The bilateral increased activity may serve as a protective strategy by increasing the overall
18 stiffness of the spine to protect from further harm (Hodges et al., 2013; Hodges and Tucker 2011).
19 A recent study also found increased activity of a trunk muscle, the contralateral external oblique
20 muscle, while lowering the arm during experimental neck pain (Christensen et al., 2015). The
21 increased activity for erector spinae muscle in the present study and not the external oblique muscle
22 as previously seen is unclear. However, the increased muscle activity distant to the painful area,
23 could indicate a disrupted motor planning, causing an overestimation of the force needed to
24 accomplish the task (Palsson et al., 2015).

25 Although the results of the present study indicates that pain may cause an increase in muscle
26 activity for the erector spinae muscles, a similar increase is seen for in the post measurement after
27 the control injection, particularly during the fast up movements for which explanations remain
28 elusive. Despite the observation of increased activity in the erector spinae muscle during a condition
29 that should not be painful, the remaining findings from the present study, in combination with
30 previous experimental findings and observations of altered trunk muscle activity in clinical neck
31 pain (Moseley 2004), it seems likely that a link between neck pain and alter motor control of trunk
32 muscles exist.

33 In general this study shows that experimental neck pain reorganizes axioscapular and trunk muscle
34 activity, but it is unknown if people experiencing an acute episode of clinical neck pain, causing

1 various acute changes and adaptations, will be predisposed for later development of on-going neck
2 pain.

3

4 *Pressure pain sensitivity*

5 A small difference in PPTs between baselines (14.8 kPa) at the neck site was found but not believed
6 to influence the results since data was normalised to baseline and thereby adjusting for this.

7 Hyperalgesia in the post measurements at the neck site is contrasting finding to that made in a
8 previous study using unilateral experimental pain (Christensen et al., 2015) and other studies on
9 experimental pain in the neck/shoulder area (Ge et al., 2003; Schmidt-Hansen et al., 2006).

10 Nonetheless, in another study on experimental pelvic girdle pain a similar response with
11 hyperalgesia at the injection site was observed in the post-pain condition (Palsson and Graven-
12 Nielsen 2012).

13 For the head and arm sites, increased PPTs were found during the painful condition when
14 compared to the post and control conditions. Such hypoalgesia away from the injection site are in
15 line with a previous study using bilateral experimental pain (Ge et al., 2003) and could indicate the
16 importance of the spatial summation (bilateral versus unilateral pain) in triggering a conditioned
17 pain modulation. The local hyperalgesia is similar to clinical neck pain where reduced PPT has
18 been observed in some subgroups of neck pain patients while the hypoalgesia away from the neck
19 site is a contrasting finding to those done in patients (La Touche et al., 2010; Sterling et al., 2002)
20 most likely due to impaired mechanisms for conditioning pain modulation in patients due to the
21 persistent pain condition (Yarnitsky 2010).

22

23 *Conclusion*

24 Bilateral experimental neck pain in healthy subjects reorganised the activity of axioscapular and
25 some trunk muscles with adaptations being linked to the type of arm movement. In addition,
26 experimental neck pain caused localised hyperalgesia along with widespread hypoalgesia. Together,
27 these results demonstrate complex adaptations of the sensory and motor systems, which could be a
28 protective mechanism. Although this study only shows the immediate effect of bilateral
29 experimental neck pain on healthy subjects, the findings may help clinicians making mechanism-
30 based decisions by supporting the inclusion of the shoulder girdle and trunk muscles in the
31 examination and rehabilitation of clinical neck pain.

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33

34 **AUTHOR CONTRIBUTION**

1 Steffan Wittrup Christensen was in charge of planning and executing data collection, statistical
2 analyses, and writing the first draft of the manuscript. Rogerio Pessoto Hirata and Thomas Graven-
3 Nielsen contributed to the planning of the study, statistical analyses and development of the final
4 version of the manuscript. All authors discussed the results, commented on the manuscript and
5 agreed on the final version.

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1 **FIGURE LEGENDS**

2 **Figure 1.** Study design: Electromyographic (EMG) recordings were followed by pressure pain
3 threshold (PPT) measurements at Baseline, During (i.e. immediately after the injection), and Post
4 (i.e. 5 min after any potential pain had vanished). The order of saline injections was randomised in a
5 balanced way.

6
7 **Figure 2.** Photographic depiction of a subject performing the standardized arm movements from an
8 upright sitting position.

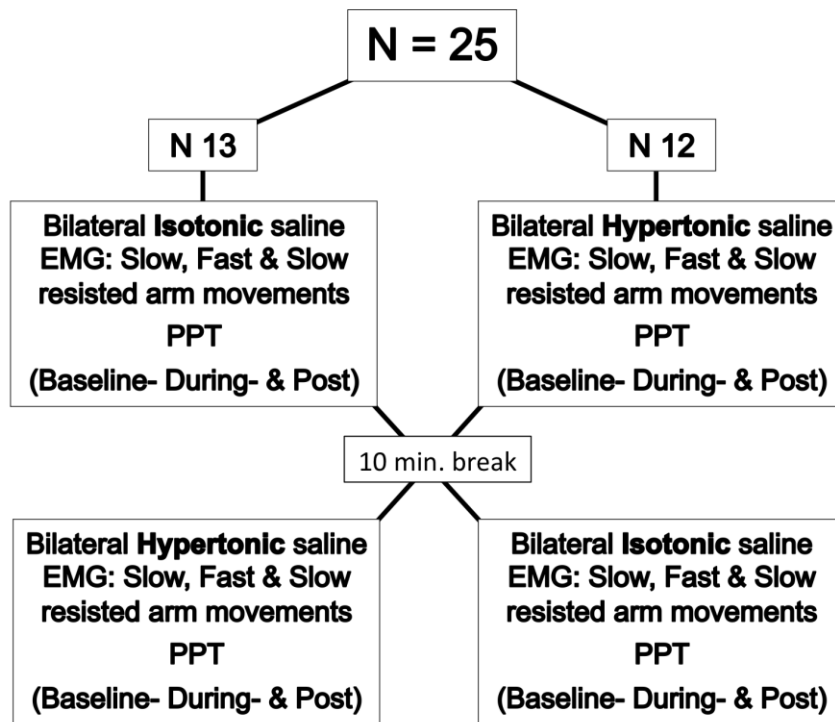
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10 **Figure 3.** Superimposed body chart drawings (N = 25) after the hypertonic saline (a) and isotonic
11 saline injections (b). Transparency in colours indicates that these were less frequently marked by
12 the subjects.

13
14 **Figure 4.** Mean normalised RMS-EMG (+ SEM, N = 25 for slow & slow resisted movements; N =
15 23 for fast movements) recorded immediately after injection of hypertonic (Hyp) or isotonic (Iso)
16 saline and in a post session 5 min after any potential pain had vanished. RMS-EMG was extracted
17 from the arm movements during the slow up (a), down (b), slow resisted up (c), down (d) and fast
18 up (e) phases. EMG recordings from ipsilateral (Ipsi) and contralateral (Contra) muscles: Upper
19 trapezius (UT), middle trapezius (MT), anterior deltoid (AD), middle deltoid (MD), and erector
20 spinae (ES). Significantly different RMS-EMG from post recordings following either the painful or
21 the control condition (*, NK: $P < 0.05$) or compared with the same time (immediately after injection
22 or post) for the control condition (#, NK: $P < 0.05$) is illustrated.

23
24 **Figure 5.** Mean (averaged for both sides) normalized pressure pain thresholds (PPTs; +SEM,
25 N=25) for the Neck (injection site), Head (m. temporalis muscle), and the Arm (m. extensor carpi
26 radialis brevis) immediately after the injection of hypertonic (Hyp) or isotonic (Iso) saline and in a
27 post measurement 5 min after any potential pain had gone. Significantly different PPT from post
28 recordings following either the painful or the control condition (*, NK: $P < 0.05$) or compared to the
29 same time (immediately after injection or post) for the control condition (#, NK: $P < 0.05$) is
30 illustrated.

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1 **Figure 1**



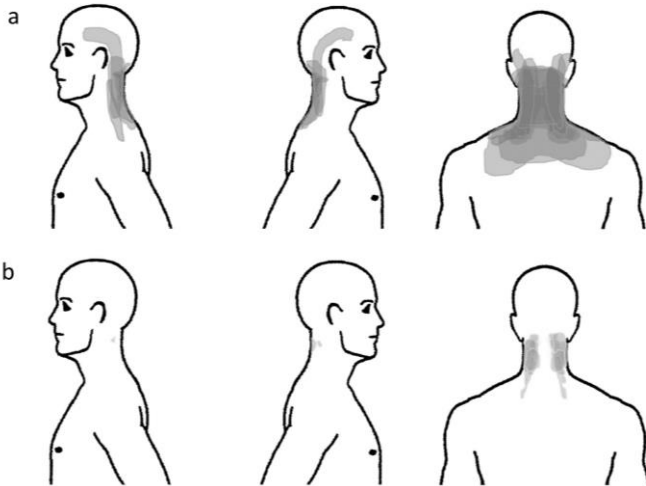
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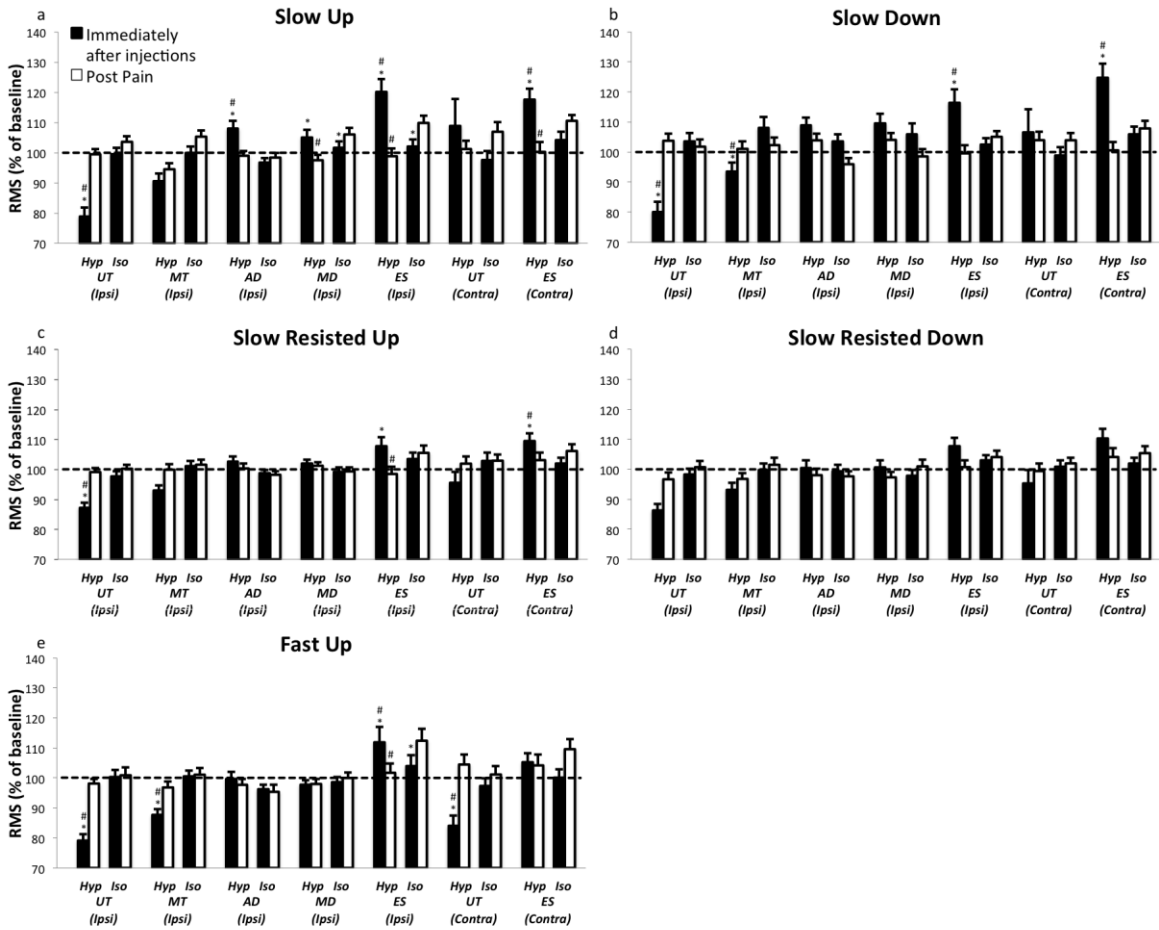
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1 **Figure 3**



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3 **Figure 4**

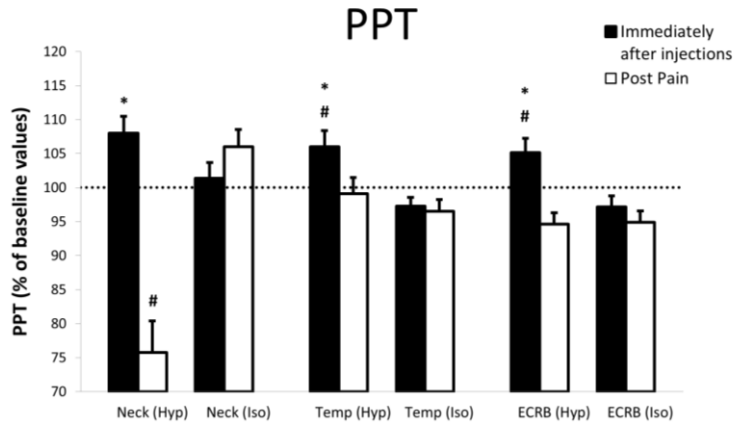


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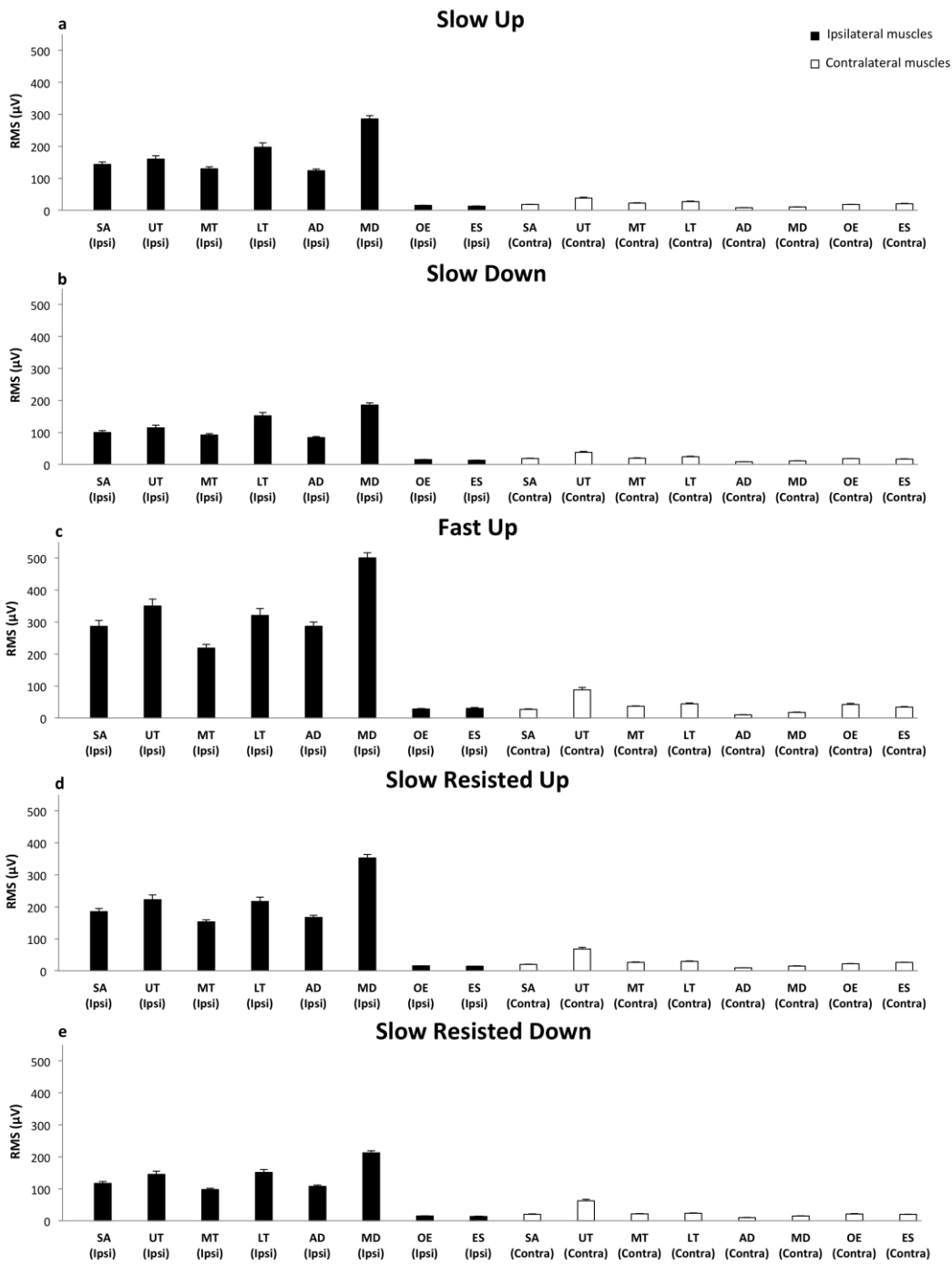
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1 **Figure 5**



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Figure S6



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2 Figure S6: Mean RMS-EMG values (+ SEM, N = 25 for slow & slow resisted movements;
 3 N = 23 for fast movements) of baseline recordings (mean of right and left movements) for
 4 slow up (a), slow down (b), fast up (c), slow resisted up (d) and slow resisted down (e).

5 Root-mean-square electromyographic (RMS-EMG) parameters from ipsilateral (Ipsi) and
 6 contralateral (Contra) muscles: Serratus anterior (SA), upper trapezius (UT), middle
 7 trapezius (MT), lower trapezius (LT), anterior deltoid (AD), middle deltoid (MD), external
 8 oblique (OE), and erector spinae (ES).”
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1 **Table S1:** The RM ANOVA interactions between saline and time for RMS-EMG recordings for all muscles and all movements. Ipsilateral (Ipsi) and
 2 contralateral (Contra) muscles with respect to the movement: Serratus anterior (SA), upper trapezius (UT), middle trapezius (MT), lower trapezius
 3 (LT), anterior deltoid (AD), middle deltoid (MD), external oblique (OE), and erector spinae (ES). Significant ANOVA interactions ($P < 0.003$,
 4 Bonferroni corrected due to multiple ANOVAs) followed by significant post-hoc testing is indicated (**, $P < 0.05$).

Table S1	Movement Type				
Muscle:	Slow Up	Slow Down	Fast Up	Resisted up	Resisted down
SA, Ipsi	F[1,48] = 0.1 P=0.713	F[1,48] = 2.8 P=0.100	F[1,44] = 6.7 P=0.012	F[1,48] = 0.4 P=0.485	F[1,48] = 0.6 P=0.418
UT, Ipsi	**F[1,48] = 37.9 P<0.001	**F[1,48] = 40.3 P<0.001	**F[1,44] = 56.1 P<0.001	**F[1,47] = 21.3 P<0.001	F[1,48] = 7.3 P=0.009
MT, Ipsi	F[1,48] = 0.004 P=0.948	**F[1,48] = 14.9 P<0.001	**F[1,44] = 11.1 P=0.001	F[1,48] = 8.5 P=0.005	F[1,48] = 0.8 P=0.356
LT, Ipsi	F[1,48] = 1.4 P=0.229	F[1,48] = 3.6 P=0.061	F[1,44] = 7.3 P=0.009	F[1,48] = 1.1 P=0.261	F[1,48] = 0.1 P=0.665
AD, Ipsi	**F[1,48] = 10.6 P=0.002	F[1,48] = 0.8 P=0.350	F[1,44] = 0.009 P=0.921	F[1,48] = 0.3 P=0.570	F[1,48] = 0.01 P=0.888
MD, Ipsi	**F[1,48] = 22.1 P<0.001	F[1,48] = 0.3 P=0.557	F[1,44] = 0.1 P=0.706	F[1,48] = 0.2 P=0.605	F[1,48] = 3.4 P=0.070
OE, Ipsi	F[1,48] = 0.1 P=0.669	F[1,48] = 0.01 P=0.889	F[1,44] = 0.6 P=0.438	F[1,48] = 1.5 P=0.220	F[1,48] = 0.08 P=0.778
ES, Ipsi	**F[1,48] = 34.3 P<0.001	**F[1,48] = 16.0 P<0.001	**F[1,44] = 18.0 P<0.001	**F[1,48] = 11.9 P=0.001	F[1,48] = 7.5 P=0.008
SA, Contra	F[1,48] = 2.3 P=0.134	F[1,48] = 0.01 P=0.908	F[1,44] = 1.0 P=0.319	F[1,48] = 2.6 P=0.113	F[1,48] = 2.59 P=0.113
UT, Contra	F[1,48] = 3.6 P=0.061	F[1,48] = 0.6 P=0.419	**F[1,44] = 32.6 P<0.001	F[1,48] = 4.7 P=0.034	F[1,48] = 0.9 P=0.332
MT, Contra	F[1,48] = 2.7 P=0.102	F[1,48] = 0.1 P=0.752	F[1,44] = 0.2 P=0.622	F[1,48] = 0.01 P=0.896	F[1,48] = 3.7 P=0.058
LT, Contra	F[1,48] = 3.7 P=0.057	F[1,48] = 0.09 P=0.761	F[1,44] = 0.8 P=0.370	F[1,48] = 4.7 P=0.033	F[1,48] = 0.4 P=0.521
AD, Contra	F[1,48] = 3.5 P=0.067	F[1,48] = 0.1 P=0.730	F[1,44] = 0.002 P=0.963	F[1,48] = 0.1 P=0.707	F[1,48] = 0.6 P=0.412
MD, Contra	F[1,48] = 0.03 P=0.858	F[1,48] = 0.1 P=0.667	F[1,44] = 0.3 P=0.531	F[1,48] = 6.0 P=0.017	F[1,48] = 4.3 P=0.043
OE, Contra	F[1,48] = 1.2 P=0.273	F[1,48] = 0.1 P=0.722	F[1,44] = 0.7 P=0.400	F[1,48] = 0.001 P=0.964	F[1,48] = 0.6 P=0.793
ES, Contra	**F[1,48] = 24.7 P<0.001	**F[1,48] = 22.3 P<0.001	F[1,44] = 5.8 P=0.019	**F[1,48] = 18.1 P<0.001	F[1,48] = 9.5 P=0.003