

Active Trigger Points in the Cervical Musculature Determine Altered Activation of Superficial Neck and Extensor Muscles in Women with Migraine

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1 **Active trigger points in the cervical musculature determine**
2 **altered activation of superficial neck and extensor muscles in**
3 **women with migraine**
4

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33 **ABSTRACT**

34 **Objective:** Previous studies have demonstrated the presence of active TrPs in women
35 with migraine reproducing their headache attacks. No study has investigated if these
36 TrPs can alter muscle function in the cervical spine in migraine. Our objective was to
37 analyze differences in activation of superficial neck flexor and extensor muscles in
38 women with migraine considering the presence of active trigger points (TrP) in splenius
39 capitis (SC), upper trapezius (UT), and sternocleidomastoid (SCM) muscles. **Methods:**
40 Surface EMG was recorded from superficial flexor (SCM and anterior scalene) and
41 extensor (SC) muscles bilaterally as subjects performed a staged task of cranio-cervical
42 flexion (CCF; 5 contractions representing a progressive increase in CCF range of
43 motion) in 70 women with migraine. They were stratified according to presence or
44 absence of active TrPs in SCM, SC or UT musculature. Comparison of normalized root
45 mean square (RMS) values was conducted with 2x5 ANCOVA with task level as the
46 within-subject variable, group stratified by active TrPs as the between-subjects variable
47 and the presence of neck pain as a co-variable. **Results:** All patients exhibited active
48 TrPs in their cervical muscles which reproduced their migraine. Women with migraine
49 exhibiting active TrPs in the SCM ($P<0.01$), UT ($P<0.05$) or SC ($P<0.05$) muscles had
50 lower normalized RMS values of their superficial neck flexors than those without active
51 TrPs in the same muscles. In addition, subjects exhibiting active TrPs in the SC and UT
52 (both, $P<0.05$) muscles had higher normalized RMS values in the SC muscle than those
53 without active TrPs in the same muscles. **Conclusion:** The presence of active TrPs in
54 the cervical musculature determines altered activation of superficial neck and extensor
55 muscles during low-load, isometric CCF contractions in women with migraine.

56 **Key words:** migraine, cranio-cervical flexion test, trigger points, electromyography.

57 **Active trigger points in the cervical musculature determine altered**
58 **activation of superficial neck flexor and extensor muscles in women**
59 **with migraine**
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61 **INTRODUCTION**

62 Migraine is a disabling primary headache described as a chronic disorder with
63 recurrent attacks. Migraine has worldwide prevalence ranging from 5 to 12%.¹ Although
64 migraine pain is mostly perceived in the ophthalmic distribution of the trigeminal nerve,
65 neck pain is also a prevalent concomitant symptom in this population.²⁻⁴ In fact,
66 approximately 76% of migraine patients also report the presence of neck pain,⁵ which
67 can occur as a premonitory manifestation, during the headache phase or even in the
68 interictal period.⁶

69 It has been suggested that the association between neck pain and migraine
70 occurs because the trigeminal-cervical convergence provides an anatomical and
71 neurophysiological path for interaction via the convergence of cervical and trigeminal
72 nociceptive afferents in the trigemino-cervical nucleus caudalis.^{7,8} In addition, central
73 sensitization presenting in most individuals with migraine may facilitate neck pain and
74 related disorders.⁹ The presence of neck pain has a negative influence on migraine by
75 reducing the pharmacological treatment response.^{10,11}

76 Experience of pain or even the anticipation of pain may promote a variety of
77 motor control changes involving redistribution of activity within and between
78 muscles.¹² Previous studies investigating neck muscle activity in patients with migraine
79 revealed varying results. For instance, during maximal voluntary isometric contractions
80 of the neck musculature, an increased co-activation of antagonist muscles was observed
81 in girls¹³ and women with either episodic or chronic migraine¹⁴ while maximal strength
82 seems to be affected only in women with chronic migraine.¹⁴ However, during low-load
83 tasks such as the cranio-cervical flexion test (CCFT) no significant differences in

84 activation of superficial neck flexors were observed in individuals with migraine.^{15,16}
85 These varying results may reflect the different tasks examined (strength versus motor
86 control) but may also suggest that changes in the activation the cervical musculature are
87 only present in some patients with migraine, rather than being unequivocally associated
88 with most migraine patients.

89 Interestingly, these previous studies have not taken into account the presence of
90 trigger points (TrPs) in the neck musculature. Yet, it is well described that patients with
91 migraine exhibit more active TrPs, those which reproduce the migraine attack when
92 stimulated,¹⁷ in the cranio-cervical muscles compared to subjects without headache.^{18,19}
93 The presence of TrPs has been associated with motor disturbances as they can promote
94 fatigue, altered coordination, and altered pattern of intramuscular activity.²⁰⁻²²

95 No previous study has investigated the potential influence of active TrPs in the
96 neck musculature on electromyographic activity of superficial neck flexor and extensor
97 muscles during the CCFT in individuals with migraine. Therefore, the aim of the current
98 study was to investigate differences in activation of superficial neck flexor and extensor
99 muscles during the CCFT in migraine patients considering the presence of active TrPs
100 in splenius capitis (SC), upper trapezius (UT) and sternocleidomastoid (SCM) muscles.
101 We hypothesized that the presence of active TrPs in the cervical musculature would be
102 associated with altered activity of the superficial neck muscles.

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109 **METHODS**

110 **Participants**

111 Patients with migraine without aura were recruited from an urban regional hospital
112 between November 2014 and October 2015. Patients were diagnosed following the third
113 edition of International Headache Society criteria by an experienced neurologist.²³
114 Migraine features including location, quality of pain, years with disease, the frequency
115 and intensity of attacks, family history and medication intake were collected as clinical
116 history. No abnormalities were detected in routine blood analyses with ESR or urine
117 analyses. An X-ray examination of the skull and cervical spine and a CT scan or MRI of
118 the head were invariably performed, and did not show any structural lesion. They were
119 excluded if they presented any of the following criteria: 1, other concomitant primary or
120 secondary headache; 2, medication overuse headache; 3, history of cervical or head
121 trauma (i.e., whiplash); 4, pregnancy; 5, history of cervical herniated disk or cervical
122 osteoarthritis on medical records; 6, any systemic degenerative disease, e.g., rheumatoid
123 arthritis, lupus erythematosus; 7, diagnosis of fibromyalgia syndrome; 8, anesthetic block
124 in the past 3 months; or, 9, receiving physical therapy intervention in the head and neck
125 the previous 6 months. A careful clinical examination of each participant was conducted
126 to determine inclusion and exclusion criteria.

127 All participants signed the informed consent form before their inclusion in the
128 study. The local Ethics Committee of Hospital Rey Juan Carlos (HRJ 07/14) approved
129 the study design.

130 **Clinical measures**

131 Clinical data including years with migraine, migraine frequency (days per month),
132 intensity of pain attacks (numerical pain rate scale, 0-10), headache duration (hours per

133 attack), as well as presence of self-reported neck pain, including report of the frequency,
134 intensity and years with neck pain were systematically collected.

135 **Cranio-cervical flexion test (CCFT)**

136 The CCFT is a low-load graded test of deep cervical flexor muscle performance
137 with five progressive stages guided by a pressure biofeedback unit (Stabilizer[®],
138 Chattanooga Group Inc. South Pacific, USA, **Fig. 1**). It is performed with the subject in
139 supine, with the head and neck in a neutral position. The pressure biofeedback unit is
140 placed behind the subject's neck in the suboccipital region, with an initial inflation
141 pressure of 20 mmHg.²⁴

142 First, participants were familiarized with the test. Subjects were instructed to
143 perform a gentle head-nodding action of cranio-cervical flexion over five incremental
144 stages of increasing range of motion (2 mmHg each stage) and each stage was
145 maintained for 10 seconds. Head extension, head lift or opening the mouth, described as
146 compensations strategies,²⁴ were discouraged at familiarization time.

147 After the familiarization phase, a rest period of 1 minute was permitted. Subjects
148 then performed the CCFT by holding each target level for 10s with 30s rest between
149 levels. During the holding phase, surface electromyography of selected neck flexors and
150 extensor muscles was acquired. The full CCFT was repeated twice with a 15min rest
151 between. All subjects performed all CCFT levels and compensatory strategies were not
152 controlled during the formal test. The CCFT examination was conducted by an assessor
153 blinded to the presence or absence of TrPs.

154 **Electromyography (EMG) acquisition and processing**

155 After gentle skin abrasion using abrasive paste, bipolar surface EMG was recorded
156 with pairs of electrodes positioned 20mm apart (Ambu[®]-Blue Sensor N-50-K/25) and
157 firmly fixed with adhesive tape bilaterally over the following cervical muscles: 1, the

158 sternal head of SCM muscle, over the muscle belly at 1/3 of the distance from the sternal
159 notch to the mastoid process;²⁵ 2, anterior scalene (AS): over the muscle belly parallel to
160 the clavicular head of the SCM;²⁵ and, 3, SC muscle: over the muscle belly at C2-C3
161 level between the uppermost parts of the SCM and UT muscles.²⁶ The reference
162 electrode was placed on the wrist of the participants. Myoelectric signals from SCM,
163 AS, SC and UT muscles were amplified by 5000 (EMG16, 16-channel amplifier,
164 LISiN-OT Bioelettronica[®], Torino, Italy), filtered (-3dB bandwidth, 10-450 Hz),
165 sampled at 2048 Hz, and converted to 12-bit digital samples.

166 Customized MATLAB code (The Mathworks[™], Natick, MA, USA) was used
167 for data processing. EMG raw signals were band-filtered at a 20-400Hz (4th order
168 Butterworth) and the average Root Mean Square (RMS) was calculated from each 10 s
169 contraction. Neck flexor and extensor RMS values were normalized and expressed as a
170 percentage of the maximum RMS value during a reference voluntary contraction. The
171 reference activity for superficial neck flexors was a head lift task, and for superficial
172 neck extensors was head extension against the table in the supine position. For analysis
173 purposes, the mean RMS values were averaged over the two repetitions for each CCFT
174 stage. Finally, the mean of both sides right and left, for each muscle were considered in
175 the analysis for all CCFT stages.

176 **Trigger Point Identification**

177 Screening for TrPs was performed by an assessor with 6 years of experience in
178 TrP diagnosis. The SCM, SC and UT muscles were assessed bilaterally since TrPs in
179 these muscles referred pain to the head mimicking migraine.^{18,19} TrP diagnosis was
180 performed according the following criteria:¹⁷ 1, presence of a palpable taut band in the
181 muscle; 2, presence of a painful spot in the taut band; 3, local twitch response on
182 snapping palpation of the taut band; and, 4, reproduction of referred pain during manual

183 examination. TrP diagnosis was conducted using snapping palpation (first to locate the
184 taut band, and then moving the thumb tip back and forth to roll the underlying fibers) to
185 induce a local twitch response, and flat palpation (placing the padded aspect of the
186 thumb on the painful spot and applying pressure against the underlying tissue or bone)
187 to induce the referred pain.

188 Participants were evaluated during interictal migraine states and pain-free states,
189 and when at least one week had elapsed since the last migraine attack to avoid migraine
190 related allodynia. TrPs were considered active when the referred pain elicited during
191 manual examination reproduced the migraine attack features that the subject usually
192 suffered from, and, therefore, the pain was recognized as a familiar pain.¹⁷ Patients were
193 classified as having active TrPs when they had TrPs reproducing their migraine attack
194 in at least one muscle, either left or right side.

195 **Statistical analysis**

196 Data were analyzed with SPSS software version 20.0 (SPSS Inc©, Chicago, IL).
197 Means and 95% confidence intervals (95%CI) were calculated for the clinical variables.
198 Patients were stratified according to the presence of active TrPs in the SCM, SC, and
199 UT muscles separately. The comparison for the normalized RMS values was conducted
200 with a 2x5 analysis of co-variance (ANCOVA) with CCFT stage (22 mmHg, 24 mmHg,
201 26 mmHg, 28 mmHg, and 30 mmHg) as the within-subject variable, and stratification
202 (presence or absence of active TrPs) as the between-subject variable and the presence of
203 neck pain as co-variate. Separates ANCOVAs were conducted depending on the muscle
204 affected by active TrPs (SCM, SC and UT). The statistical analysis was conducted at
205 95% confidence level. A P value < 0.05 was considered statistically significant.

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207

208 **RESULTS**

209 **Clinical features of the sample**

210 From 100 eligible subjects with migraine who accepted to participated, 30 were
211 excluded for the following reasons: other co-morbid headaches (n=15), receiving
212 anesthetic block (n=6) or botulinum toxin A (n=6) in the past 3 months, and reporting
213 previous head or neck trauma (n=3). Finally, 70 women, mean age: 42±12 years old,
214 with episodic migraine without aura were included. A total of 58 women (83%) self-
215 reported neck pain. All women exhibited active TrPs reproducing their migraine attacks.
216 The mean ± SD number of active TrPs for each patient with migraine was 3.0± 1.5. The
217 UT muscle was the most affected by active TrPs in our sample (n=41, 59%). **Table 1**
218 summarizes demographic and clinical data of the total sample. The clinical status of
219 patients was not dependent on the presence of TrPs in each cervical muscle (**Table 2**).

220 **Neck flexor activity and TrPs**

221 Normalized RMS values for SCM and AS muscles during the five stages of the
222 CCFT in those patients with active TrPs in the sternocleidomastoid, upper trapezius and
223 splenius capitis are shown in **Figs. 2-4**. There was an increase in EMG amplitude of the
224 SCM and AS with the progressive stages of the test independently of the presence of
225 active TrPs in the SCM muscle (SCM: F=16.57; P<0.001, AS: F=15.35; P<0.001),
226 upper trapezius (SCM: F=12.59; P<0.001, AS: F=16.54; P<0.001), or SC muscle (SCM:
227 F=16.15; P<0.001, AS: F=10.18; P<0.001). Women with migraine exhibiting active
228 TrPs in the SCM muscle (SCM: F=10.307; P=0.002, AS: F=7.169; P=0.009), UT
229 muscle (SCM: F=5.19; P=0.026, AS: F=4.491; P=0.044) or SC muscle (SCM: F=7.852;
230 P=0.007, AS: F=6.437; P=0.018) showed lower normalized RMS values of their
231 superficial neck flexors than those without active TrPs in the same muscles (**Figs. 2-4**).
232 The presence of neck pain did not influence the results (SCM: P>0.253, AS: P>0.356).

233 **Neck extensor activity and TrPs**

234 Normalized RMS values for SC muscle during the five stages of the CCFT in
235 those patients with active TrPs in the SCM, UT and SC are shown in **Fig. 5**. There was
236 also an increase in EMG amplitude of the SC with the progressive stages of the test
237 independently of the presence of active TrPs in either the SCM ($F=4.41$; $P=0.039$), UT
238 ($F=4.591$; $P=0.045$), or SC ($F=11.176$; $P<0.001$) muscles. In contrast to the results for
239 the flexor muscles, the results revealed higher normalized RMS values in the SC muscle
240 in women with migraine exhibiting active TrPs in the SC ($F=4.05$; $P=0.046$) and UT
241 ($F=4.014$; $P=0.046$) muscles (**Fig. 5**) compared to those without active TrPs in the same
242 muscles. No significant differences were observed for normalized RMS values in the
243 SC in those patients with active TrPs in the SCM muscle ($F=0.290$; $P=0.592$, **Fig. 5**).
244 The presence of neck pain (SC: $P>0.213$; UT: $P>0.293$) did not influence the results.

245

246 **DISCUSSION**

247 Women with migraine exhibiting active TrPs in the SCM, SC and UT muscles
248 had lower activation of their superficial neck flexors, i.e., SCM and AS, during low-
249 load CCF contractions. In addition, the presence of active TrPs in the superficial neck
250 extensors, i.e., SC and UT, determined increased activation of the SC muscle during
251 cranio-cervical flexion contractions.

252 It is well known that noxious stimulation of a muscle, e.g. with experimental muscle
253 pain via intramuscular injection of hypertonic saline, induces a temporary decrease of
254 EMG amplitude of the painful muscle together with compensatory strategies within the
255 same muscle^{27,28} or across synergistic muscles.²⁹⁻³¹ It may be speculated that a long-
256 lasting nociceptive irritant, such as an active TrP, also induces inhibition of the painful
257 muscle when activated. This knowledge may explain the reduced activation of the SCM

258 and AS muscles in individuals with active TrPs in the same musculature. Interestingly,
259 reduced activation of the SCM and AS was also noted in women with active TrPs in the
260 SC or UT muscles which implies that the altered muscle strategy is not necessarily due
261 to pain induced inhibition locally within the muscle.

262 The observation of reduced activation of the SCM and AS during the CCFT in the
263 women with migraine and active TrPs is in contrast to observations in people with
264 primary neck disorders, including cervicogenic headache.^{15,16} Rather, people with
265 cervical spine disorders show higher activity of the SCM and AS muscles during the
266 CCFT which has been shown to be an indicator of poor performance of the deep neck
267 flexor muscles, i.e., longus colli and longus capitis.^{32,33} However, migraine is a primary
268 headache mainly associated to brain dysfunction with deficient regulation of excitatory-
269 inhibitory balance during cortical activity leading to trigemino-vascular sensitization.
270 Thus, although individuals with migraine usually suffer from concomitant neck pain,⁵
271 they do not have a primary neck pain disorder which would explain these contrasting
272 results. Nevertheless, we observed that the presence of active TrPs within the cervical
273 musculature implies different activation of the neck flexor muscles compared to those
274 without active TrPs in the same muscles. Interestingly, differences in muscle activation
275 was associated to the presence or absence of active TrPs, but not related to the presence
276 of neck pain in our study.

277 During tasks with low mechanical demands, performance can be maintained despite
278 pain, also via modification of antagonist musculature activity.^{30,34} Indeed, one theory of
279 the motor adaption to pain indicates that muscle pain induces reorganization of the
280 motor strategy characterized by reduced activity of agonist muscles and increased
281 activity of antagonist muscles (pain adaptation theory).³⁵ The current work supports the
282 observation of increased antagonist muscle activity since increased SC muscle activity

283 was noted when active TrPs were present within the SC or UT muscles. In support of
284 the current findings, individuals with chronic, but not episodic, migraine exhibit higher
285 activity of their superficial neck extensors (i.e., SC muscle) during low-load, isometric
286 cranio-cervical flexion contractions compared to non-headache individuals (unpublished
287 observations) and women with chronic tension type headache also show greater co-
288 activation of antagonist muscles (i.e. the SC muscle) during isometric neck flexion
289 contractions compared with headache-free subjects.³⁶ Thus, increase co-activation of
290 antagonist musculature appears to be a common feature in people with headache. The
291 results from the current study suggest that increased antagonist muscle co-activation is
292 even more likely in those with active TrPs.

293 Overall, the observation that TrPs are associated with changes in the activation of
294 agonist and antagonist muscles is consistent with earlier findings. Ibarra et al observed
295 increased muscle activity at latent TrPs in an antagonist muscle (i.e., posterior deltoid
296 muscle) during shoulder flexion task.²² Lucas et al³⁷ found that the presence of latent
297 TrPs impaired recruitment or timing of muscle activation when performing active joint
298 movement and Ge et al²⁰ found that the presence of latent TrPs induced incoherent
299 muscle activation patterns in synergist musculature during muscle contractions.
300 However, these studies included latent TrPs, but not active TrPs, which limit the clinical
301 relevance of their data since latent TrPs are not related to clinical pain complaints. Our
302 study is the first one showing that the presence of active TrPs was associated with a
303 different pattern of agonist and antagonist muscle activation in patients with headache.
304 Our finding has potential implication for clinical practice. Since the presence of active
305 TrPs in the cervical musculature is related to altered pattern of neck muscle activation, it
306 would be recommended that clinicians first treat these TrPs before start any therapeutic

307 exercise program targeting at normalizing motor control disturbances observed in these
308 patients.

309 Although the study expands current knowledge on changes in muscle behavior
310 in individuals with migraine, potential limitations should be recognized. First, we only
311 included women with migraine, therefore, we do not know if the same results would be
312 observed in men. Second, we included a single low-load cranio-cervical flexion task for
313 investigating muscular activity, but this task does not necessarily represent muscle
314 demands during daily life activities. Third, psychological features, e.g. fear of
315 movement, were not measured and may have proven useful in understanding the
316 mechanisms underlying the observed altered muscle behavior in people with migraine.
317 Further, a control group of headache-free individuals was not included; thus, although
318 we can confirm differences in the activation of the neck musculature between women
319 with and without active TrPs in their cervical muscles, we cannot confirm that the
320 changed pattern of activation within the migraine group with active TrPs would be
321 significantly different to asymptomatic people.

322

323 **CONCLUSION**

324 In the current study, all women with migraine exhibited active TrPs in the neck
325 muscles reproducing their migraine attack. Women with migraine who have active TrPs
326 in the cervical musculature show an altered pattern of neck muscle activation during a
327 low-load cranio-cervical contraction compared to those without active TrPs in the
328 evaluated muscle. Alterations of afferent input (i.e., painful stimulus induced by active
329 TrPs) appear to influence muscle activation at a multi-muscular level.

330

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333

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Legend of Figures

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457 **Figure 1:** Pressure biofeedback unit (Stabilizer[®], Chattanooga Group Inc. South Pacific,
458 USA) used during the cranio-cervical flexion test (CCFT)

459 **Figure 2:** The normalized root mean square (RMS) values for the sternocleidomastoid
460 and anterior scalene muscles for the five stages of the cranio-cervical flexion test
461 depending on the presence or absence of active trigger points (TrPs) in the
462 sternocleidomastoid muscle (SCM - yes, n=36 / no, n=41). Values for the left and right
463 muscles have been averaged. Data are expressed as means and SEM. * P<0.05; **
464 P<0.01

465 **Figure 3:** The normalized root mean square (RMS) values for the sternocleidomastoid
466 and anterior scalene muscles for the five stages of the cranio-cervical flexion test
467 depending on the presence or absence of active trigger points (TrPs) in the upper
468 trapezius muscle (UT- yes, n=41 / no, n=29). Values for the left and right muscles have
469 been averaged. Data are expressed as means and SEM. * P<0.05; ** P<0.01

470 **Figure 4:** The normalized root mean square (RMS) values for the splenius capitis
471 muscle for the five stages of the cranio-cervical flexion test depending on the presence
472 or absence of active trigger points (TrPs) in the splenius capitis muscle (SC - yes, n=29 /
473 no, n=41). Values for the left and right muscles have been averaged. Data are expressed
474 as means and SEM. * P<0.05; ** P<0.01

475 **Figure 5:** The normalized root mean square (RMS) values for the splenius capitis
476 muscle for the five stages of the cranio-cervical flexion test depending on the presence
477 or absence of active trigger points (TrPs) in the sternocleidomastoid (SCM - yes, n=36 /
478 no, n=41), upper trapezius (UT- yes, n=41 / no, n=29), or splenius capitis (SC - yes,
479 n=29 / no, n=41) muscles. Values for the left and right muscles have been averaged.
480 Data are expressed as means and SEM. * P<0.05; ** P<0.01