Headache
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Published by Blackwell Publishing

ISSN 0017-8748
doi: 10.1111/j.1526-4610.2006.00632.x

Research Submission

Myofascial Trigger Points, Neck Mobility, and Forward Head Posture in Episodic Tension-Type Headache

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Objective.—To assess the differences in the presence of trigger points (TrPs) in head and neck muscles, forward head posture (FHP) and neck mobility between episodic tension-type headache (ETTH) subjects and healthy controls. In addition, we assess the relationship between these muscle TrPs, FHP, neck mobility, and several clinical variables concerning the intensity and the temporal profile of headache.

Background.—TTH is a headache in which musculoskeletal disorders of the craniocervical region might play an important role in its pathogenesis.

Design.—A blinded, controlled pilot study.

Methods.—Fifteen ETTH subjects and 15 matched controls without headache were studied. TrPs in both upper trapezius, both sternocleidomastoids, and both temporalis muscles were identified according to Simons and Gerwin diagnostic criteria (tenderness in a hypersensible spot within a palpable taut band, local twitch response elicited by snapping palpation, and elicited referred pain with palpation). Side-view pictures of each subject were taken in both sitting and standing positions, in order to assess FHP by measuring the craniovertebral angle. A cervical goniometer was employed to measure neck mobility. All measures were taken by a blinded assessor. A headache diary was kept for 4 weeks in order to assess headache intensity, frequency, and duration.

Results.—The mean number of TrPs for each ETTH subject was 3.7 (SD: 1.3), of which 1.9 (SD: 0.9) were active, and 1.8 (SD: 0.9) were latent. Control subjects only had latent TrPs (mean: 1.5; SD: 1). TrP occurrence between the 2 groups was significantly different for active TrPs ($P < .001$), but not for latent TrPs ($P > .05$). Differences in the distribution of TrPs were significant for the right upper trapezius muscles ($P = .04$), the left sternocleidomastoid ($P = .03$), and both temporalis muscles ($P < .001$). Within the ETTH group, headache intensity, frequency, and duration outcomes did not differ depending on TrP activity, whether the TrP was active or latent. The craniovertebral angle was smaller, ie, there was a greater FHP, in ETTH patients than in healthy controls for both sitting and standing positions ($P < .05$). ETTH subjects with active TrPs in the analyzed muscles had a greater FHP than those with latent TrPs in both sitting and standing positions, though differences were only significant for certain muscles. Finally, ETTH patients also showed lesser neck mobility than healthy controls in the total range of motion as well as in half-cycles (except for cervical extension), although neck mobility did not seem to influence headache parameters.

Conclusions.—Active TrPs in the upper trapezius, sternocleidomastoid, and temporalis muscles were more common in ETTH subjects than in healthy controls, although TrP activity was not related to any clinical variable concerning the intensity and the temporal profile of headache. ETTH patients showed greater FHP and lesser neck mobility than healthy controls, although both disorders were not correlated with headache parameters.

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Accepted for publication July 25, 2006.

Key words: tension-type headache, myofascial trigger points, upper trapezius muscle, sternocleidomastoid muscle, temporalis muscle, neck mobility, forward head posture

(*Headache* 2007;47:662-672)

Headache disorders are one of the most common problems seen in medical practice. Among the many types of headache disorders, tension-type headache (TTH) is the most frequent in adults. Population-based studies suggest 1-year prevalence rates of 38.3% for episodic TTH (ETTH), and 2.2% for chronic TTH (CTTH).[1] Despite some advances, the pathogenesis of TTH is not clearly understood.

Bendtsen postulated a pain model for TTH where nociceptive inputs from tender muscles can lead to central sensitization seen in CTTH.[2] Other authors have claimed that TTH is caused by referred pain evoked from several head, shoulder, and neck muscles.[3,4] In their comprehensive text, Simons et al described the referred pain patterns from different myofascial trigger points (TrPs) in head and neck muscles.[4] Since TTH is characterized by bilateral, pressing and tightening pain; mild to moderate of intensity; some pain features of TTH, such as pressure or band-like tightness,[5] or increased tenderness on palpation of neck and shoulder muscles,[6,7] resemble the descriptions of referred pain originating in TrPs.[4]

Simons et al define a TrP as a hyperirritable spot associated with a taut band of a skeletal muscle that is painful on compression, palpation, and/or stretch, and that usually gives rise to a typical referred pain pattern.[4] Active TrPs are cause of clinical symptoms and their evoked referred pain is responsible for the patients' pain. Latent TrPs may not be an immediate source of pain, but might produce other muscle dysfunctions, ie, fatigue, restricted range of motion, and referred pain with muscle contraction or compression.[4] This clinical distinction has been strongly substantiated by histochemical findings at the TrP, since higher levels of concentration of protons, bradykinin, calcitonin gene-related peptide, substance P, tumor necrosis factor-α, interleukin-1β, serotonin, and norepinephrine have been recently found in active TrPs.[8]

Marcus et al found in their nonblinded study that TTH patients showed a greater number of either active or latent TrPs in different muscles than healthy subjects.[9] Fernández-de-las-Peñas et al have recently demonstrated, in blinded controlled studies, that CTTH was associated with active TrPs in the suboccipital muscles,[10] and in the upper trapezius, sternocleidomastoid, and temporalis muscles.[11] Further, we also found that those CTTH subjects with active TrPs had greater headache intensity and frequency than those with latent TrPs.[10,11]

Cervical musculoskeletal abnormalities have been traditionally linked to different headaches.[12,13] An excessive forward head position, or forward head posture (FHP) has already been related to cervicogenic headache (CeH),[14] and we have recently found FHP in association with CTTH.[15] Conversely, restricted neck mobility has been found in CeH[16] and CTTH,[15] but not in unilateral migraine patients.[17]

After such early observations, we have extended our studies in CTTH and migraine to the episodic form of TTH. This article describes the differences in the presence of TrPs in head and neck muscles, FHP, and neck mobility between ETTH subjects and healthy controls. In addition, we assess the relationship between these muscle TrPs, FHP, neck mobility, and several clinical variables concerning the intensity and the temporal profile of headache.

MATERIAL AND METHODS

Subjects.—Fifteen subjects presenting with ETTH and 15 healthy age- and sex-matched subjects without headache during the previous year participated in this study. Patients were recruited form the Neurology Department of the Fundación Hospital Alcorcón, whereas controls were recruited from the staff personal of the Hospital. Patients with ETTH were diagnosed according to the criteria of the International Headache Society by an experienced neurologist.[5] ETTH patients had to have headache less than 15 days per month. The patients were not allowed to take analgesics or muscle relaxants 24 hours prior to the examination. A headache diary was kept for 4 weeks

in order to confirm the diagnosis.[18] The health status of all participants was clinically stable, without current symptoms of any other concomitant disease. This study was supervised by the Departments of Physical Therapy and Neurology of Rey Juan Carlos University and Fundación Hospital Alcorcón, and it was also approved by the local Human Research Committee. All subjects signed an informed consent prior to their inclusion.

Myofascial TrP Examination.—The upper trapezius, both sternocleidomastoids, and both temporalis muscles were evaluated for myofascial TrPs by an assessor who had more than 5 years' experience in TrPs diagnosis, and who was blinded to the subjects' diagnosis. TrP diagnosis was performed following the diagnostic criteria described by Simons et al[4] and by Gerwin et al:[19] (1) presence of a palpable taut band in a skeletal muscle; (2) presence of a hypersensitive tender spot in the taut band; (3) local twitch response elicited by the snapping palpation of the taut band; and (4) reproduction of the typical referred pain pattern of the TrP in response to compression. A TrP was considered active if the referred pain evoked by its compression reproduced the same subject's head pain, whereas a TrP was considered latent if the evoked referred pain did not reproduce an usual or familiar pain.[4,19] Figure 1 details the location and the referred pain patterns evoked by TrPs in the examined muscles based on the comprehensive text of Simons et al.[4]

The TrP examination was performed in a blinded fashion. After TrP assessment, the subject was asked if the TrP reproduced a familiar pain or their usual headache. Since control subjects could have had some head pain but not headache, the assessor remained blinded through the end of the examination.

FHP Assessment.—A picture of the lateral view of each subject was taken to objectively assess FHP. The base of the camera was set at the height of the subject's shoulder. The tragus of the ear was clearly marked and a plastic pointer was taped to the skin overlying the spinous process of the 7th cervical vertebra (C7). Once the picture was obtained, it was used to measure the craniovertebral angle: the angle between the horizontal line passing through C7 and a line extending from the tragus of the ear to C7 (Fig. 2).[20] A smaller craniovertebral angle is associated with a greater FHP. A previous article supported the high reliability of this procedure (intra-class correlation coefficient [ICC] = 0.88).[21]

FHP was assessed in 2 different positions: a relaxed sitting position and a relaxed standing position, in a standard protocol. Details of this protocol can be found elsewhere.[10,15] A picture of the lateral view of each subject was taken in both positions. These measurements were acquired by an assessor blinded to the subjects' diagnosis.

Neck Mobility Assessment.—Active neck mobility was assessed with a cervical goniometric (Fig. 3) device manufactured by Performance Attainment Associates (St. Paul, MN, USA), which has proved to have a good intra- and intertester reliability.[22,23] Neck mobility was recorded as the total range of motion for different types of movement, ie, flexion/extension, lateral flexion, and rotation, as well as for half-cycles, namely movements in a single direction, ie, flexion and extension, and lateral flexion and rotation to each side. Neck mobility was measured in a relaxed sitting position following a protocol described elsewhere.[15] Briefly, all subjects were asked to sit comfortably on the chair. Then, the goniometer was placed at the top of their head. Once the goniometer was set in neutral position, they were asked to move the head actively as far as possible in a standard protocol: forward (flexion), backward (extension), right lateral flexion, left lateral flexion, right rotation, and left rotation.[15] Two measurements were recorded for each type of movement. Since nonsignificant differences were found between both measurements (paired Student's *t*-test), data for further analysis were derived from the average of both values.

Study Protocol.—All subjects had 2 appointments within a 4-week period. All subjects had to be headache free on the day of the examination. In the first visit, assessor 1 gave a headache diary to ETTH subjects. Patients had to register on this diary the daily headache intensity, on a 10-cm horizontal visual analog scale (VAS; range: 0 = no pain to 10 = maximum pain),[24] the headache duration (in hours per day), and the days with headache. This headache diary was kept for 4 weeks. Assessor 1 also informed control healthy subjects about physical therapy and headache, but did not give them a headache diary. Assessor 2, blinded to

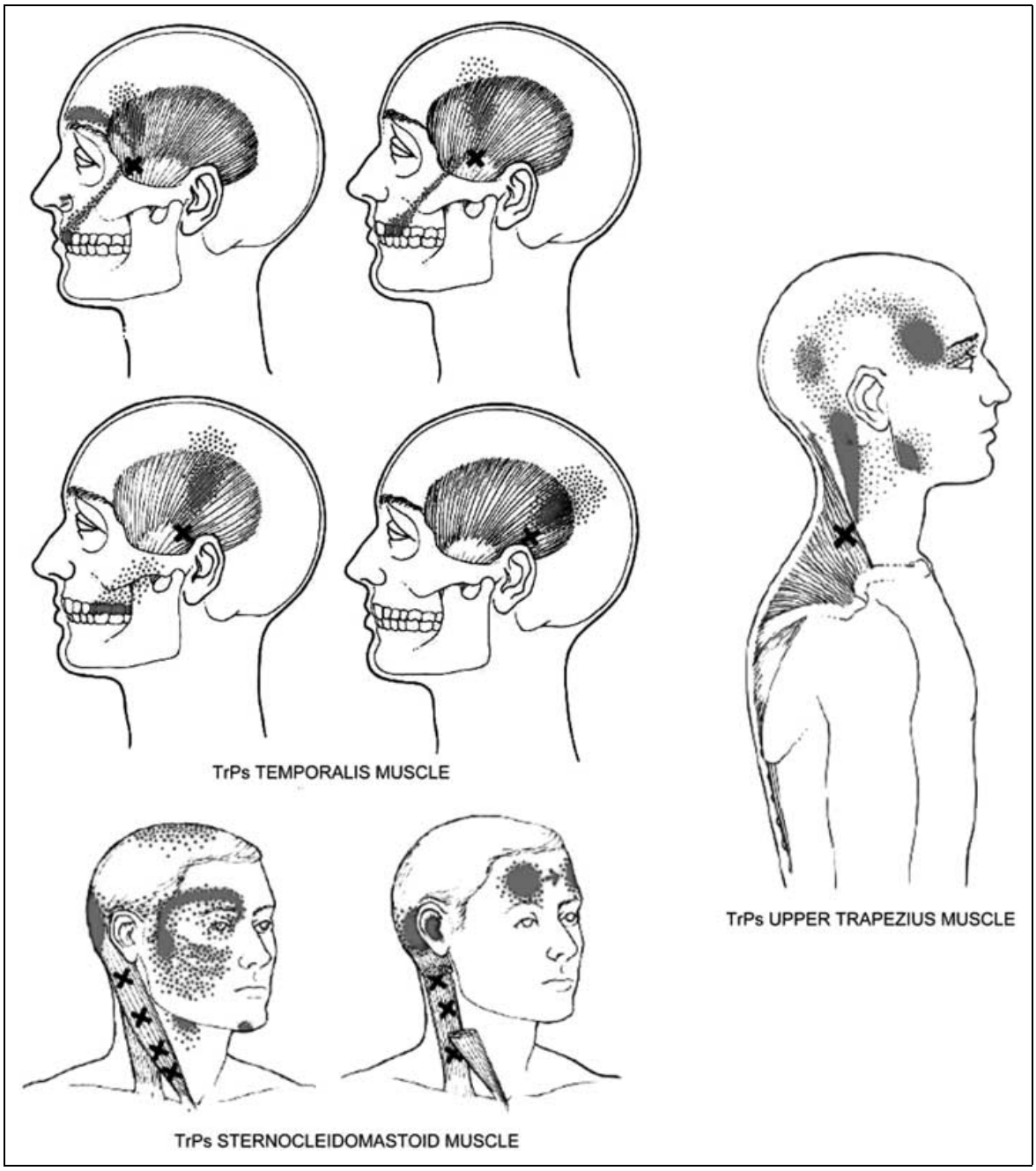

Fig 1.—Referred pain pattern from myofascial trigger points in the upper trapezius, sternocleimastoid, and temporalis muscles. Reprinted with permission from Ref. 4.

the subjects' condition, took 2 pictures of each subject, 1 in sitting, and 1 in standing position. Finally, active neck mobility was obtained by the same assessor.

At the second visit 4 weeks later, assessor 2 repeated the same head posture assessment and examined the aforementioned head and neck muscles for the presence of TrPs. ETTH subjects returned the headache diary to the first assessor who calculated the following variables: (1) headache intensity, which was calculated from the mean of the VAS of the days with headache; (2) headache frequency, which was calculated dividing the number of days with headache by 4 weeks (days per week); and (3) headache duration (hours per day), which was calculated dividing the sum of the total hours of headache by the number of days with headache (hours per day).

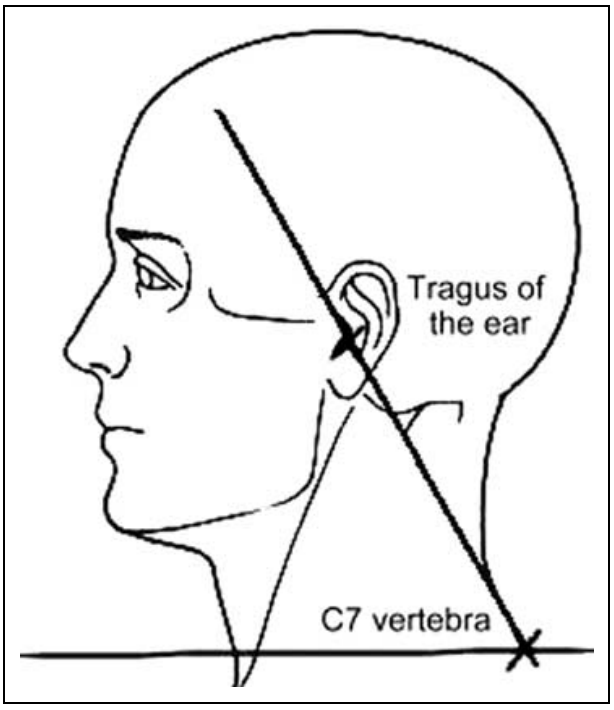

Fig 2.—Measurement of the craniovertebral angle. The angle was assessed directly from a side-view picture using a protractor image and a straight edge.

Statistical Analysis.—Data were analyzed with the SPSS statistical package (12.0 Version, Chicago, IL, USA). Mean values of the craniovertebral angle and all cervical motions, and the number of active and latent TrPs were calculated. A normal distribution of quantitative data was assessed by means of the Kolmogorov-Smirnov test. Quantitative data without a normal distribution (ie, number of active TrPs) were analyzed with nonparametric tests, whereas quantitative data with a normal distribution (ie, headache intensity, duration and frequency, number of latent TrPs, craniovertebral angle in both positions, and neck mobility) were analyzed with parametric tests. Differences in the number of active TrPs between both study groups were assessed with the Mann-Whitney *U*-test; whereas differences in the number of latent TrPs were assessed with the unpaired Student's *t*-test. The χ^2 test was used to assess the differences in the distribution of either latent or active TrPs within each muscle between both study groups. Differences in both FHP and neck mobility between both groups were assessed with the unpaired Student's *t*-test. The unpaired Student's *t*-test was achieved to assess possible gender differences in both neck mobility and FHP. The unpaired Student's *t*-test was also used to analyze both the differences in the clinical variables relating to headache (headache intensity, frequency, and/or duration) and the differences in FHP between ETTH subjects with either latent or active TrPs within each muscle. The Pearson's correlation test (*r*) was used to analyze the association between the craniovertebral angle (FHP) or neck mobility and the clinical variables relating to headache (headache intensity, frequency, and/or duration) in ETTH patients. In general, a *P* value less than .05 was considered statistically significant; however, when 2 related comparisons were performed (ie, cervical flexion and extension, right and left lateral flexions, and right and left rotations) a corrected *P* value of less than .025 was taken (Bonferroni correction).

RESULTS

A total of 15 ETTH subjects, 3 men and 12 women, aged 20 to 70 years, and 15 healthy volunteers, 4 men and 11 women, aged 21 to 70 years, were studied. No significant differences were found for gender or age between both study groups. ETTH subjects were headache free on the day of the evaluation. Demographic and clinical data of each group are given in Table 1.

The mean number of TrPs for each ETTH subject was 3.7 (SD: 1.3), of which 1.9 (SD: 0.9) were active, and 1.8 (SD: 0.9) were latent. Control subjects only had latent TrPs (mean: 1.5; SD: 1). TrP occurrence between the 2 groups was significantly different for active TrPs ($P < .001$), but not for latent TrPs ($P > .05$).

Within the ETTH group, TrPs in the right upper trapezius muscles and in both temporalis muscles were the most prevalent (n = 11; 74%), followed by TrPs in the left sternocleidomastoid (n = 9; 60%). Within the control group, the most prevalent TrPs were located in the right (n = 7; 47%) and in the left (n = 4; 27%) upper trapezius muscles. Differences in the distribution of both active and latent TrPs were significant for the right upper trapezius muscles ($P = .04$), the left sternocleidomastoid ($P = .03$), and both temporalis muscles ($P < 0.001$). Table 2 details the distribution of either latent or active TrPs on each study group.

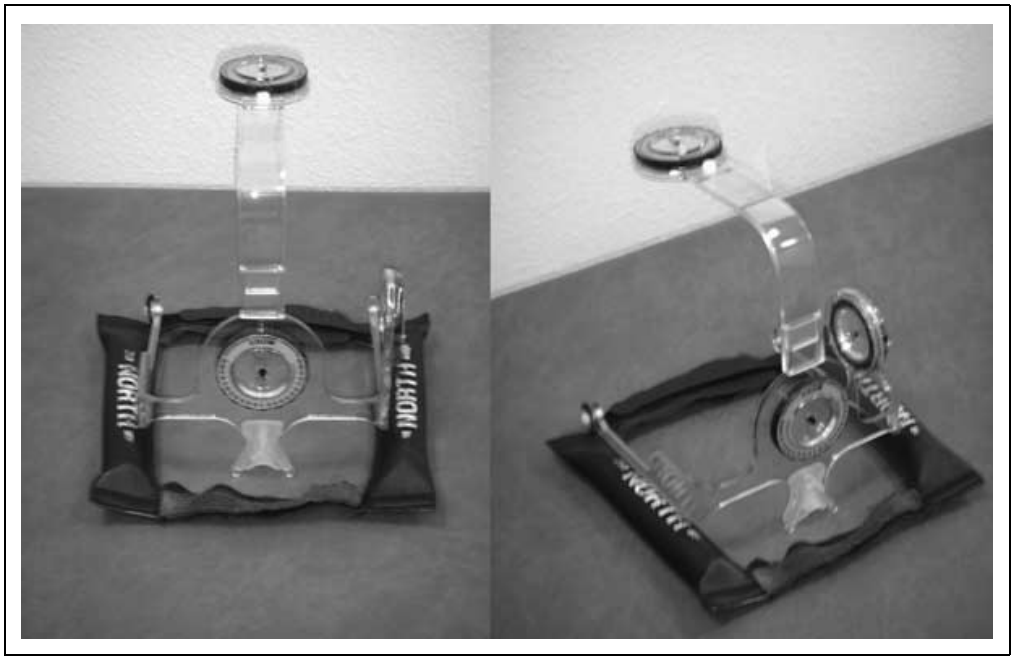

Fig 3.—Cervical goniometer used to assess neck mobility (Performance Attainment Associates, St. Paul, MN, USA).

Within the ETTH group, headache intensity, frequency, and duration outcomes did not differ depending on TrP activity, whether the TrP was active or latent (Table 3). Otherwise, ETTH subjects with active TrPs in the analyzed muscles tended to have a greater FHP than those patients with latent TrPs in both sitting and standing positions. Differences were significant for TrPs in the right sternocleidomastoid and FHP in the standing position ($P = .03$); TrPs in the left sternocleidomastoid and FHP in the sitting position ($P = .04$), and TrPs in the left temporalis muscle and FHP in both sitting ($P = .04$) and standing ($P = .03$) positions. The relationship of FHP to TrP activity on each muscle is shown in Table 4.

In order to verify the reliability of FHP measurements and to check if the head posture remained stable during the study, 2 separate sets of pictures were taken from each subject with a 4-week interval. No significant differences were found between the 2 measurements (paired Student's *t*-test): seated $P > .5$; CCI $= 0.93$, $P < .001$; standing $P > .6$; CCI $= 0.94$, $P < .001$. Therefore, data for further analysis were derived from the average of the 2 values corresponding to each position.

Table 1.—Demographic and Clinical Data of Both Groups

	ETTH (n = 15)	Controls (n = 15)	*P* Value
Gender (male/female)	3/12	4/11	NS
Age (years)	39 ± 17	37 ± 12	NS
Length of headache history (years)	12 ± 5	–	–
Headache intensity (VAS)	4.6 ± 2	–	–
Headache frequency (days/week)	2.8 ± 0.6	–	–
Headache duration (hours per headache day)	7 ± 4	–	–

Values are expressed as mean ± standard deviation.
ETTH = episodic tension-type headache; NS = nonsignificant; VAS = visual analog scale (0-10).

Table 2.—Distribution of Subjects with Myofascial Trigger Points (Active of Latent) in Both Study Groups

	Upper Trapezius Muscle		Sternocleidomastoid Muscle		Temporalis Muscle	
	Right Side	Left Side	Right Side	Left Side	Right Side	Left Side
Subjects with episodic tension-type headache						
Active TrPs (n)	5	2	3	2	6	7
Latent TrPs (n)	6	6	2	7	5	4
Control healthy subjects						
Active TrPs (n)	0	0	0	0	0	0
Latent TrPs (n)	7	4	3	2	1	3
P value	.04	NS	NS	.02	.001	.004

P values express differences between active TrPs.
Differences between latent TrPs were not significant.
n = number of subjects; TrP = myofascial trigger point.

The craniovertebral angle was smaller, ie, there was a greater FHP, in ETTH patients than in healthy controls for both sitting and standing positions ($P < .05$). ETTH patients also showed lesser neck mobility than healthy controls in the total range of motion as well as in half-cycles (except for cervical extension). There were no significant differences in both neck mobility and FHP between males and females in either group. Finally, both neck mobility and FHP did not correlate with headache parameters. Table 5 summarizes both neck mobility and FHP parameters of each study group.

COMMENTS

This article is the first blinded, controlled study providing evidence that active myofascial TrPs in the upper trapezius, sternocleidomastoid, and temporalis muscles are more common in ETTH patients

Table 3.—Headache Intensity, Frequency, and Duration Depending on the Type of Myofascial Trigger Point of Each Muscle within the Episodic Tension-Type Headache Group

		Headache Intensity (VAS)	Headache Frequency (Days/Week)	Headache Duration (Hours/Day)
Right upper trapezius	Active TrPs (n = 5)	5.1 (1.4)	2.7 (0.4)	6.3 (0.7)
	Latent TrPs (n = 6)	4.2 (1.7)	2.7 (0.7)	4.7 (3.2)
Left upper trapezius	Active TrPs (n = 2)	5.1 (0.2)	3 (0.5)	6.4 (0.6)
	Latent TrPs (n = 6)	4.6 (1.8)	2.5 (0.7)	8.6 (2.1)
Right sternocleidomastoid	Active TrPs (n = 3)	5.2 (2)	2.8 (0.8)	6.8 (1.7)
	Latent TrPs (n = 2)	4.3 (0.3)	2.3 (1)	3.5 (3.5)
Left sternocleidomastoid	Active TrPs (n = 2)	3.9 (0.9)	2.5 (0.5)	5.7 (0.4)
	Latent TrPs (n = 7)	4.5 (1.3)	2.5 (0.7)	6.1 (3.8)
Right temporalis	Active TrPs (n = 6)	5 (1.4)	2.7 (0.5)	5.5 (3.1)
	Latent TrPs (n = 5)	4.5 (2)	3.1 (0.6)	7.3 (4.1)
Left temporalis	Active TrPs (n = 7)	4 (1.5)	2.8 (0.6)	7.3 (4)
	Latent TrPs (n = 4)	5.6 (1.9)	2.7 (0.7)	7 (1.4)

Values are expressed as means (standard deviations).
None of the comparison showed statistical significance (unpaired Student's *t*-test, $P > .05$).
TrPs = myofascial trigger points; VAS = visual analog scale (0-10).

Table 4.—Forward Head Posture Depending on the Type of Myofascial Trigger Point on Each Muscle within the Episodic Tension-Type Headache Group

		Craniovertebral Angle Sitting	Craniovertebral Angle Standing
Right upper trapezius	Active TrPs (n = 5)	47.2 (9.6)	48.2 (8.2)
	Latent TrPs (n = 6)	51.7 (4)	52.5 (7.1)
Left upper trapezius	Active TrPs (n = 2)	47 (2.8)	49 (1.5)
	Latent TrPs (n = 6)	48 (4)	50 (3.8)
Right sternocleidomastoid	Active TrPs (n = 3)	45.6 (4.9)	48.3 (6)*
	Latent TrPs (n = 2)	41.5 (9.1)	41 (1.4)
Left sternocleidomastoid	Active TrPs (n = 2)	42.5 (6.6)*	46 (8.4)
	Latent TrPs (n = 7)	51.2 (4.2)	50 (6.8)
Right temporalis	Active TrPs (n = 6)	49.5 (6.1)	47.2 (7.7)
	Latent TrPs (n = 5)	50.2 (6.4)	54.6 (5.5)
Left temporalis	Active TrPs (n = 7)	46.3 (6)*	46.9 (5.8)*
	Latent TrPs (n = 4)	54.7 (4.4)	56.3 (6.4)

*Significant in comparison with the latent TrP subgroup (unpaired Student's *t*-test, $P < .05$).
Values are expressed as means (standard deviations).
A smaller craniovertebral angle indicated a greater FHP.
TrPs = myofascial trigger points.

than in healthy subjects. In addition, ETTH subjects showed greater FHP, that is, a smaller craniovertebral angle, and lesser neck mobility than controls. Finally, headache parameters (ie, intensity, duration, and frequency) were not significantly different between ETTH patients with active TrPs and those with latent TrPs in the same muscles. Neck mobility and FHP did not also correlate with headache characteristics.

Current findings for ETTH subjects complete our previous findings for CTTH subjects, in whom head and neck muscle TrPs were also more common than in healthy controls.[10,11] However, in CTTH subjects, active TrPs were related to greater headache intensity and frequency than latent TrPs.[10,11] These latter results have not been replicated in this study, since none of the clinical variables concerning the intensity and the temporal profile of headache differed between ETTH subjects with active TrPs than those with latent TrPs. If there were a lesser degree of central sensitization in ETTH, because of the intermittent nature of the condition, one would expect fewer active and more latent TrPs in ETTH subjects than in CTTH patients. Our findings do not support this hypothesis, since ETTH patients showed a similar number of either latent or active TrPs than CTTH.[11] Since TrPs, either active or latent, are responsible for the liberation of nociceptive mediators,[8] it seems plausible that TrPs might be a triggering factor for central sensitization. In such way, the presence of TrPs can contribute to the evolution of ETTH to the chronic form. Our results underline the importance of inspection and inactivation of TrPs in head and neck muscles, which are contributing to sensitization of the trigeminal nucleus caudalis in TTH patients.

Animal[25] and human[26,27] studies clearly show the convergence of cervical and trigeminal afferents, constituting the anatomical basis for the referred headache pains from the TrPs in the analyzed muscles. In addition, peripheral and central sensitization and decreased descending inhibition induced by long-term nociceptive stimuli from TrPs may also involve in referred pain to trigeminal region from active TrPs.[28] Olesen proposed that headache is due to an excess of nociceptive inputs from peripheral structures.[29] According to his model, headache intensity is the sum of nociceptive inputs from cranial and extracranial tissues converging on trigeminal nucleus caudalis neurons. Convergence of the nociceptive afferents from the receptive fields of cervical roots C1-C3, which include the upper trapezius and the sternocleidomastoid muscles, and those of the trigeminal nerve, which include the temporalis muscle, occurs in the nucleus

Table 5.—Range of Motion for All Cervical Movements of Each Study Group

	ETTH Group	Control Group	*P* Value
Flexion/Extension			
Flexion	47.2° ± 11.4°	66.8° ± 9.7°	.001
Extension	49.3° ± 11.8°	48.3° ± 14.2°	NS
Total	96.4° ± 18.7°	115.2° ± 28.7°	.002
Lateral flexion			
Right	34.7° ± 7.1°	41.2° ± 6.7°	.01
Left	34.8° ± 7.7°	43.4° ± 5.6°	.001
Total	69.4° ± 12.4°	84.5° ± 12.1°	.01
Rotation			
Right	58.2° ± 9.7°	72.5° ± 5.5°	<.001
Left	59.6° ± 9.5°	74.2° ± 6.9°	
Total	117.7° ± 18.8°	146.5° ± 9.1°	
Craniovertebral angle			
Sitting position	48.8° ± 7°	53.8° ± 4°	.02
Standing position	50° ± 7°	55.9° ± 5.5°	.01

Values are expressed as mean ± standard deviation.
P values come from the unpaired Student's *t*-test (a *P* value less than .025 was considered statistically significant).
ETTH = episodic tension-type headache ; NS = nonsignificant.

caudalis.[25-27] Continuous or prolonged nociceptive afferent input resulting in temporal and spatial summation could lead to central sensitization postulated to occur in the chronic form of TTH. It is possible that nociceptive inputs from head and neck muscle TrPs can produce a continuous afferent bombardment to the *trigeminal nerve nucleus caudalis*. Such repeated nociceptive activation of the *nucleus caudalis* could produce central sensitization. Inactivation of head, neck, and shoulder TrPs in those with TTH would be expected to decrease headache parameters. Since an association between TrPs and ETTH has been found in this study; a therapeutic approach based on TrP management should now be evaluated.

Neck mobility was lesser in ETTH patients than in controls. Our results are in disagreement with those reported by Zwart, who did not find any significant difference between TTH subjects and controls.[16] Yet, we have to consider that neck mobility reported by Zwart for TTH patients[16] (127° ± 19.6° in flexion/extension; 91° ± 12.8° in lateral-flexion; 168° ± 17.2° in rotation) and for healthy subjects (129° ± 17.9° in flexion/extension; 94° ± 17.9° in lateral-flexion; 170° ± 22.1° in rotation) was greater than that in our study (see Table 5). Chen et al have reported that neck mobility depends on the technology or the method of assessment, ie, goniometer, inclinometer, potentiometer, etc.[30] In their meta-analysis, Chen et al established the following normative values: flexion/extension, 150° to 116°; flexion, 69° to 48°; extension, 93° to 61°; whole lateral bending, 108° to 76°; right- or left-side bending, 49° to 38°; whole rotation, 186° to 136°, and right or left rotation, 93° to 70°.[30] Based on these results, neck mobility of our nonheadache group falls within the normative range, whereas neck mobility of our ETTH patients falls below normative values. Therefore, we may conclude that our control healthy group had normal neck mobility, and that our ETTH group presented a decrease in neck mobility. Further, neck mobility did not correlate with any clinical variable concerning the intensity and the temporal profile of headache. Our results are in agreement with those reported by Griegel-Morris et al,[31] who did not find an association between the severity of postural abnormalities and the severity and frequency of pain in patients presenting with neck pain, and with Fernández-de-las-Peñas et al,[15] who also did not find an association between headache pain parameters and neck mobility in CTTH subjects. Based on these findings, it seems that differences in neck mobility can be most likely a consequence of the abnormal head posture, the pain, or both, rather than a causative factor for headache.

Our results also demonstrated that patients with ETTH had a greater FHP, ie, a smaller craniovertebral angle, than control subjects in both sitting and standing positions. FHP has been previously related to other headache disorders. For instance, Watson and Trott found that CeH patients showed a lesser craniovertebral angle than controls (44.5° ± 5.5° vs 49.1° ± 2.9°; $P < .001$).[14] Fernández-de-las-Peñas et al demonstrated that CTTH patients also show a lesser craniovertebral angle than normal controls (45.3° ± 7.6° vs 54.1° ± 6.3°; $P < .001$).[15] In addition, Fernández-de-las-Peñas et al have also found that unilateral migraine showed greater FHP than controls in both sitting and standing position ($P < .001$).[17] In contrast to our findings, Zito et al have recently reported no significant differences in FHP between CeH, migraine, and healthy subjects.[32] The craniovertebral angle in our ETTH group was similar to that found in CeH,[14] CTTH,[15]

and unilateral migraine subjects,[17] whereas the mean craniovertebral angle in our nonheadache group was similar than that found in healthy subjects in the aforementioned studies. Therefore, we can regard our control healthy group as having a normal posture, while our ETTH group presented an altered posture. Based on previous and current findings, FHP appears to be a feature common to several headache syndromes.

ETTH subjects with active TrPs in the analyzed muscles tended to have a smaller craniovertebral angle than those with latent TrPs in both sitting and standing positions. Although FHP is usually associated with shortening of the posterior cervical extensor muscles (suboccipital, semispinalis, splenii, and upper trapezius muscles) as well as shortening of the sternocleidomastoid muscle, it is also possible that FHP might be a consequence of pain, ie, an antalgic posture, to try to reduce pain. Nevertheless, our studies suggest that shortened, contracted head and neck muscles associated with FHP may contribute to development or perpetuation of TTH. However, we should recognize that FHP was not really significantly associated with increased TrP activity in all the analyzed muscles. Whether FHP contributes to the origin or the perpetuation of headaches must be verified by future research. Determination of the clinical significance of FHP in different headaches would require the development and testing of specific physical therapy programs.

There are some limitations to our studies. First, only subjects with TTH or unilateral migraine have been evaluated. Hence, our results cannot be extrapolated to other headache disorders, such as CeH, nummular headache, or cluster headache. It would be interesting to repeat the same procedure with patients suffering from other disorders in order to explore the relevance of head and neck muscle TrPs in headache. The second limitation was the small sample size. To our knowledge, our studies are the first ones to analyze the relationship between craniocervical muscle TrPs, FHP, neck mobility, and clinical features in TTH. However, it would be necessary to repeat the same procedure with a greater number of subjects to confirm our findings in both ETTH and CTTH patients. Further research is needed to clearly define the role of head and neck muscle TrPs.

In conclusion, active TrPs in the upper trapezius, sternocleidomastoid, and temporalis muscles were more common in ETTH subjects than in healthy controls, although TrP activity was not related to any clinical variable concerning the intensity and the temporal profile of headache. ETTH patients showed greater FHP and lesser neck mobility than healthy controls, although both disorders were not correlated with headache parameters. ETTH subjects with active TrPs tend to have a greater FHP than those patients with latent TrPs. Since TrPs might contribute to the origin and/or maintenance of headache, a comprehensive knowledge of the role of these muscles in the appearance of headache awaits further research.

Acknowledgment: We would like to thank Dr. David Simons and Dr. Robert D. Gerwin for their kind encouragement and support.

Conflict of Interest: None

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