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Pericranial tenderness in females with episodic cervical headache versus asymptomatic controls

Sarah Mingels^{1*}, Marita Granitzer^{1‡}

1. REVAL Rehabilitation Research Centre, Biomedical Research Institute, Faculty of Medicine and Life Sciences, Hasselt University, 3500, Hasselt, Belgium

*Corresponding author: Hasselt University, Faculty of Medicine and Life Sciences, Agoralaan, 3590, Diepenbeek, Belgium.

Tel.: +32-11-268509

Email: sarah.mingels@uhasselt.be

‡Hasselt University, Faculty of Medicine and Life Sciences, Agoralaan, 3590, Diepenbeek, Belgium.

Tel.: +32-11-269344

Email: marita.granitzer@uhasselt.be

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Abstract

Objectives. To compare pericranial tenderness of females with episodic cervical headache versus matched asymptomatic controls.

Methods and Material. Through a single-blind cross-sectional study pericranial tenderness was compared between 20 females with episodic cervical headache (29.4 ± 13.2 years) and 20 age-and gender-matched asymptomatic controls (30.1 ± 13.7 years). Pericranial tenderness was bilaterally measured in a headache free period with the 'Total Tenderness Score' in the suboccipital, temporal, frontal, masseter, upper trapezius, levator scapula and sternocleidomastoid muscle insertions. Passive cervical mobility, headache intensity, frequency and duration were secondary outcomes. Analysis was done with a 95% confidence level (SPSS version 22). The Mann-Whitney U-test was used to compare pericranial, cephalic, cervical and muscle specific tenderness between groups. Correlations between 1) passive cervical mobility and 2) headache characteristics and the total tenderness score were estimated with Spearman's rho.

Results. The Headache-group (1.25 ± 0.89) showed a two time higher ($p < 0.05$) pericranial total tenderness score compared to the Control-group (0.62 ± 0.70). Higher ($p < 0.05$) scores were observed for the left suboccipital, temporal, masseter, upper trapezius, levator scapula and sternocleidomastoid muscles and the right suboccipital, frontal, upper trapezius and levator scapula muscles. Grouping the tenderness scores into cervical (suboccipital, upper trapezius, levator scapula, sternocleidomastoid) and cephalic (frontal, temporal, masseter) regions revealed also greater scores ($p < 0.05$) in the Headache-group. In the latter the total tenderness score was significantly positively correlated with passive cervical extension ($\rho = 0.78$).

Conclusion. Consistent higher tenderness scores suggest involvement of sensitization in patients with episodic cervical headache. A positive correlation was seen between passive

cervical extension and sensitivity.

Keywords. Headache, episodic, sensitivity, women, posture

Introduction

Headache is one of the most frequently reported complaints in working women for which primary care physicians and physiotherapists are consulted.¹ Some of these headaches can be provoked by poor sitting postures.^{2,3,4}

In Europe, people spend five to six hours a day on sitting activities.⁵ Higher prevalence of musculoskeletal complaints were nevertheless reported when daily use of the computer exceeded three hours.⁶ Risks of developing such complaints are positively correlated not only to work hours, but also to female gender.⁷ A cross-sectional study by Malinska and Bugajska (2010) revealed that headache was the most important complaint in 55% of female employees who regularly used portables while working.⁸

Another remarkable fact is that sitting behavior during the use of mobile computing technologies such as a laptop, desktop, smartphone or tablet is often characterized by an increased forward head position (FHP).⁹⁻¹³ In particular cervical headaches can be provoked and worsened by a pronounced FHP. Such a habitual posture can create abnormal loading on cervical structures and thereby affect the cervical range of motion (CROM).¹⁴⁻¹⁷

The CROM is an important feature and diagnostic criterion in the examination of patients with headache.^{18,19} A restricted CROM has implications on proprioceptive mechanisms of the cervical spine. Proprioceptive failure can reduce postural control and increase the load on spinal tissue.¹⁶ An augmented CROM on the other hand, can cause tissue deformation via creep and enlarge the neutral zone.²⁰ A dysfunctional CROM can alter spinal posture, change the habitual posture, eventually be harmful and lead to activation of nociceptors.^{21,22} Through repetitive

nociceptive stimuli (wind-up), second-order neurons in the dorsal root become sensitized and even induce neuroplastic changes.²³ In patients with posture-related headache, nociceptive cervical stimuli might first sensitize the trigemino-cervical complex whereas in time repeated noxious input can cause central sensitization.^{2,24} The latter has been mooted as an underlying mechanism in chronic tension-type headache. These patients present with an increased pain sensitivity in cephalic and extra-cephalic muscles.²⁴ Hence, sensitization of nociceptive pain pathways in the central nervous system, due to prolonged nociceptive stimuli, seems a plausible explanation for the conversion of episodic into chronic pain. The most accepted theory is that episodic headache is more related to peripheral and chronic headache to central mechanisms.^{24,25} These findings indicate a generalized increased pain sensitivity and support a central sensitization hypothesis.²⁶ Yet, the International Headache Society emphasizes that an increased pericranial tenderness is a feature in both episodic and chronic tension-type headache. The latter was confirmed by a recent study by Palacios Ceña et al. (2017) in which similar local and widespread pressure hyperalgesia was found for episodic and chronic tension-type headache. These results could indicate involvement of peripheral and central mechanisms in both forms of headache.²⁷

The above mentioned inconsistencies and chronification in 3 to 5% of all patients with episodic headache, plead for more in-depth research on episodic headache. Besides, most studies focus on chronic headache.²⁴⁻³⁰ Especially women seem at risk for the development of chronic pain because of a lower pain threshold for mechanical stimuli.³¹

Since a dysfunctional CROM is considered to be a potential source of spinal musculoskeletal symptoms, neck mobility and muscle tenderness seem to be related.^{3,16,20-22,32}

Within this hypothesis, pericranial tenderness ('Total Tenderness Score')^{28,33,34}, passive CROM and their inter-relation will be compared between a cervical Headache-group and a asymptomatic control group (C). Patients with episodic headache were targeted since

indications of centralisation exist.²⁷

Methods

Design

A single-blind cross-sectional comparison of pericranial tenderness between females with episodic cervical headache in a headache free period versus matched asymptomatic controls.

Participants

Sixty four potential candidates for the Headache-and C-group responded to a general call which was launched at the Hasselt University. Using an informative questionnaire, containing the in- and exclusion criteria (based on the International Headache Society, 2013), 62 female participants were selected. Twenty participants met the criteria for the Headache-group (Table 1). Twenty asymptomatic participants were matched for age and gender to compose the C-group.

Selection of the participants for the Headache-group took place through an examination and interview by a manual therapist and a physician. Inclusion criteria for the Headache-group were: females, between 18 and 58 years, meeting specific headache-criteria (Table 1). Exclusion criteria: pregnancy, physiotherapy for head or neck problems 12 months before the study, serious pathology (neurological: diseases of the central or peripheral nervous system; cardiovascular: blood pressure related pathology; endocrine: e.g. diabetes; musculoskeletal: pathology or deformities affecting the spine), pain radiation in the upper extremities and a history of neck/head trauma.

Inclusion criteria for the C-group were: asymptomatic females, between 18 and 58 years.

Exclusion criteria: pregnancy, history of neck/head trauma or pain.

The study is registered at ‘ClinicalTrilas.gov (ID NCT02887638)’. The Medical Ethical Committee of the ‘Ziekenhuis Oost-Limburg’ granted approval for the study (ref. B371201423025) and all participants signed the written informed consent in which information was given concerning the confidentiality of the data. Included participants were anonymized through a numeral code according to their features (Headache or Control). The researcher (Sarah Mingels) who performed the testing and statistical analysis only had access to encoded data. An independent researcher (AV) provided the encoding. The protection of personal data is legally determined by the law of December 8th 1992 on the protection of privacy according to the Belgian law.

[Table 1]

Outcomes, measurements and instruments

Pericranial tenderness was the primary outcome which was evaluated with the ‘Total Tenderness Score (TTS)’. The TTS ranges from 0 (no sensitivity) to 3 (high sensitivity) and is reliable in healthy adults and patients with tension-type headache.³³⁻³⁵ The TTS is recognized worldwide as both a scale and a tenderness measure used in muscular and headache research.³⁶ Headache intensity³⁷ (100 mm Visual Analogue Scale (VAS)/week), duration (hours/day) and frequency (days/month) were secondary outcomes extracted from the ‘Belgian Headache Society’ diary which was completed by the Headache-group four weeks before the start of the measurements.³⁸ Maximal passive cervical flexion and extension (°) were secondary outcomes assessed by an universal goniometer. The reliability of this apparatus is excellent (Intra-class Correlation Coefficient: passive flexion 0.83, passive extension 0.86).³⁹

Procedure

Maximal passive cervical motion (C1-C7) was measured according to the procedure of Norkin and White (2009)⁴⁰ in a headache free period. Two consecutive passive cervical flexion and extension measurements were performed in the sagittal plane (left side of the face) with the participant in neutral sitting posture, i.e. both feet flat on the floor, 90° flexion in the hips and knees and the spine positioned in neutral from lumbar to thoracic.⁴¹ Measurements were executed by a trained examiner. The researcher was blinded for the different groups and participants were tested randomly. An independent researcher (AV) determined the ad random sequence through lottery. Between each measurement a pause of 1 minute was provided. Afterwards averages were calculated. Next, pericranial TTS were bilaterally determined on marked muscle insertions of the levator scapula, sternocleidomastoid (SCM), upper trapezius (UT), temporal, masseter, frontalis and suboccipitals as described by Langemark and Olesen (1987).³⁵ From the TTS cephalic, cervical and muscle specific tenderness scores (TS) were derived. To determine cephalic and cervical TS the pericranial muscles were grouped in a cephalic (frontal, temporal, masseter muscles) and cervical group (SCM, levator scapula, UT, suboccipital muscles). Pressure was applied by the examiner on the insertion while making small circular movements with the thumb for five seconds.³⁴ The participant's response was recorded on a 4-point scale: 0 = no visible reaction or verbal report of discomfort, 1 = mild mimic reaction but no verbal report of discomfort, 2 = verbal report and mimic reaction of painful tenderness and discomfort and 3 = marked grimacing or withdrawal, verbal report of marked painful tenderness and pain. The measurements were performed three times in a fixed order (as mentioned above) starting on the right side. The maximum TTS was 42 ($7 \times 2 \times 3$ (insertion \times right/left \times maximum score)). Maximum cephalic and cervical TS were 18 and 24 respectively.²⁸ Total scores were averaged and converted to a 0-3 scale.

Statistical analysis

Analysis was done using SPSS version 22 with a 95% confidence level ($p < 0.05$). Equality of groups was tested by the Mann-Whitney U-test (Table 2). Parametric or non-parametric statistics were applied based on the following assumptions: sample size, normality (Shapiro-Wilk) and equivalence (Brown-Forsythe). All assumptions had to be met in order to apply parametric statistics. In case of normal distribution values were expressed by the mean (\pm standard deviation). Pearson's r or Spearman's ρ estimated a possible correlation between variables based on the assumptions (linearity, equal variances and normal distribution). Given the explorative nature of the study no type I(α)-corrections (Bonferroni) were applied.

Results

General

Non-parametric statistics were used because of the small sample size and Brown-Forsythe results ($p < 0.05$). To compare pericranial, cephalic, cervical and muscle specific TS and passive cervical range of motion between groups the Mann-Whitney U-test was used (Table 3). Correlations between 1) headache characteristics and 2) passive cervical mobility versus tenderness were estimated with Spearman's ρ . Confidence intervals (95%) were determined for each measurement (Table 2). A priori analysis, based on the TTS, revealed that in order to obtain a power of 80% (0.05 probability of a type-I error) 16 participants per group are needed.⁴² For the TTS a post-hoc power analysis (power of 80%) was done (98.9%).

Group characteristics

Table 2 provides a summary of the group characteristics. No significant differences were found.

[Table 2]

Primary outcome: Pericranial tenderness

The pericranial TTS was higher in the Headache-group ($p = 0.0001$). Similarly, higher muscle specific TS were seen in the Headache-group for the left suboccipital, temporal, masseter, UT, levator scapula and SCM muscles and the right suboccipital, frontal, UT and levator scapula muscles ($p < 0.05$) (Table 3). Comparison of the cephalic and cervical TS between groups revealed higher scores for both regions in the Headache-group ($p < 0.05$) (Table 3). This was the case for both the left and right side ($p < 0.05$). No significant intra-group left-right differences were seen.

Secondary outcome: Headache-characteristics

A strong correlation was found between headache frequency and the TTS ($\rho = -0.60$). No correlations were detected between headache 1) intensity ($\rho = 0.36$) and 2) duration ($\rho = -0.20$) versus the TTS.

Secondary outcome: Passive cervical flexion and extension

In the Headache-group the following correlations were observed (Figure 1): a strong correlation between passive cervical extension and the TTS ($\rho = 0.78$), a moderate to strong correlation between passive cervical extension and the cervical TS ($\rho = 0.68$) and a strong correlation

between passive cervical extension and cephalic TS ($\rho = 0.74$). No correlations were seen between passive cervical flexion and tenderness.

[Table 3]

[Figure 1]

Discussion

Since sensitization is closely related to chronification of headache the main focus of the current study was to explore sensitization in females with episodic cervical headache in whom headache was provoked by sitting postures (Table 1). Although becoming a growing problem group, sensitisation was never researched. Having more insight would be a help for physiotherapists in the prevention of chronification.

The most relevant results for the Headache-group were: 1) significantly higher scores on the pericranial TTS, cephalic, cervical and muscle specific TS and 2) the association between passive cervical extension and the TTS.

Pericranial tenderness

The significantly higher TTS, cephalic, cervical and muscle specific TS in the Headache-group seem to confirm the hypothesis of sensitization of the trigemino-cervical nucleus. The latter fits the general accepted theory that sensitization occurs due to peripheral nociceptive input.⁴³⁻⁴⁵ Yet, little is known about mechanisms that provoke an increased tenderness.^{2,31,45,46} A possible mechanism could be peripheral sensitization of cervical myofascial nociceptors caused by poor

sitting postures.²⁸ Associations between pain and posture have been reported previously. Pain experienced over the entire trapezius muscle has been assigned to an increased head-flexion and more pain at muscle palpation was related to uncomfortable and prolonged postures.^{47,48} In our study 80% of the patients reported that studying and/or working with the laptop or desktop was the primary provocative source to develop headache (Table 2). These uncomfortable postures increase the load on cervical structures. The repeated character of mechanical stimuli, from tissues innervated by C1-C3, might activate myofascial pericranial nociceptors and cause headache through convergence at the trigemino-cervical complex.² Repetitive nociceptive stimuli are hypothesised to interfere with the endogenous pain modulation and thereby leading to sensitization. A dysfunction in endogenous pain modulation in patients with episodic headache may be a predisposing factor that increases vulnerability for recurrent and eventually chronic headaches.⁴⁸ Preceding studies have identified both peripheral and central sensitization as contributing factors to headache and its chronification.²⁴ Although cephalic and extra-cephalic sensitization are features of chronic headache, a more recent study mooted central sensitization in episodic headache.^{25,26,27} The higher cervical scores in the current study could suggest involvement of central mechanisms in episodic headache.²⁷

No left-right differences in tenderness were detected in the Headache-group. Our measurements however, were taken in a headache free period. In contrast, Aaseth, Grande, Lundqvist and Russell (2014) described such differences when measurements were taken during a headache period.⁴⁹

Headache characteristics

No correlation between headache intensity and duration vs. the TTS in our study could be detected. Similar results have been reported in patients with chronic tension-type headache.⁴⁸⁻

⁵¹ In patients with chronic headache an association seems to exist between the number of active pericranial triggerpoints, a higher pain intensity and headache duration.⁵⁰⁻⁵² Hypothetically, it could be insinuated that higher TTS at several pericranial locations are contributing to or a consequence of chronicity.

Passive cervical flexion and extension

Participants in the Headache-group presented with lesser neck mobility for passive cervical flexion and extension compared to the C-group. Conflicting results exist concerning passive CROM in patients with episodic headache.^{3,54} Since Chen et al. (1999) reported a larger CROM in females in all age groups, comparing results is difficult because in most studies both sexes were included.⁵⁵

Passive cervical mobility in our Headache-group was larger compared to previous studies (flexion 59.22 vs. 47.20° and extension 54.50 vs. 49.30°).^{3,53} Since we solely examined females a possible explanation for the larger cervical mobility could be the general larger joint mobility in women.^{53,55}

Interestingly, in the Headache-group a positive correlation exists between cervical extension range of motion and tenderness. The authors hypothesize that the Headache-group might use cervical extension as an ‘unload-mechanism’ for the increased stress on the cervical region created by most sitting postures.⁴⁰ The resulting enlarged neutral zone or augmented muscular activity could provoke a sensitization process. A prolonged postural cervical hyperextension and an increased cervical mobility are both associated with headache.^{3,56,57} It seems that in patients with headache differences in neck mobility might be the consequence of the posture, rather than a direct cause for headache.³ In addition, dysfunctions in mobility are associated with local increased tenderness.⁵⁸

Conclusion

It can be concluded from our results on the TTS that female participants with episodic cervical headache already have a tendency to progress into a state of central sensitization. The higher cervical and cephalic TS support our postulated hypothesis. Both peripheral and central sensitization are associated with chronification.^{24,46} Therefore, tenderness scores could be used in clinical practice to screen patients with episodic cervical headache who might be at risk of sensitization. Higher scores should be a signal for the physiotherapist to take action to prevent aggravation of a possible ongoing sensitization process. Finally, an increased passive cervical extension range of motion and a higher sensitivity seem to be associated.

Suggestions and limitations

To determine a possible relation between posture and sensitization in episodic cervical headache and whether an increased tenderness in episodic postural-induced headache is prognostic to develop chronic headache further research is needed. In addition, comparing pressure pain hypersensitivity in trigeminal and extra-trigeminal areas in individuals with episodic and chronic headache could assist to a better understanding of underlying mechanisms.²⁵ Results in this study refer to a female sample size. In order to quantify the TTS it would be interesting to compare the TTS between males and females. Finally, more in depth research involving postural variables such as a forward head position and thoracic kyphosis is needed. Although the sample size was small, significant differences could be detected. Future research with a larger sample size is needed to investigate and clarify the correlations in our study.

Declaration of interest statement

We declare that we have no competing interests.

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Tables

Table 1. Inclusion criteria for the Headache-group

Inclusion criteria	
Characteristics	<p>Episodic headache</p> <p>Cervical stiffness</p> <p>Headache worsens with provocative manoeuvres/postures</p> <p>At least two of the following characteristics:</p> <ol style="list-style-type: none"> 1. pressing or tightening (non-pulsating) 2. mild or moderate intensity 3. reduced cervical ROM 4. neck pain related to the headache
Provocation	<p>Headache provoked by at least one of the following:</p> <ol style="list-style-type: none"> 1. Poor cervical posture (e.g. forward head posture) 2. Sitting posture 3. Repetitive cervical movement 4. Prolonged posture
Autonomous	<ol style="list-style-type: none"> 1. no nausea or vomiting 2. no photophobia or phonophobia
Duration	<p>At least 10 episodes of headache occurring on 1- 14 days per month on average for</p> <p>>3 months (≥ 12 and ≤ 180 d/y) and lasting from 30 minutes to 7 days</p>

ROM, Range of Motion; d/y, days a year

Table 2. Mean (SD) group characteristics of the Headache-group and C-group

	Headache (n=20)	C (n=20)	p ^a
Cervical flexion (°) [CI]	59.22 (8.37) [55.31-63.14]	66.10 (12.69) [60.16-72.04]	0.06
Cervical extension (°) [CI]	54.50 (8.93) [50.34-58.70]	57.25 (9.17) [52.96-61.54]	0.28
Age (years)	29.4 (13.21)	30.1 (13.71)	0.81
Intensity (VAS/week)	4.38 (1.30)	N/A	N/A
Frequency (days/week)	3.10 (1.32)	N/A	N/A
Duration (hours/week)	7.22 (4.19)	N/A	N/A
Headache provocation	80% laptop or desktop use 15% watching television% 5% ironing	N/A	N/A

C, asymptomatic controls; SD, Standard Deviation; [CI], 95% Confidence Interval; VAS, Visual Analogue Scale;

N/A, not applicable; n , number of participants.; ^a Statistically significant difference when p<0.05 with the Mann-Whitney U-Test

Table 3. Summary of the mean (SD) total, regional and individual TS for the Headache-group and C-group

	Headache (n=20)	C (n=20)	p^a
Pericranial TTS (SD) [CI]	1.25 (0.89) [0.86-1.64]	0.62 (0.70) [0.31-0.93]	0.0001*
Regional TS (SD) [CI]			
<i>Cephalic</i>	1.18 (0.88) [0.79-1.57]	0.68 (0.74) [0.36-1.00]	0.0001*
<i>Cervical</i>	1.30 (0.90) [0.91-1.69]	0.57 (0.70) [0.26-0.88]	0.0001*
Regional Left TS (SD) [CI]			
Cephalic	1.25 (0.86) [0.87-1.63]	0.75 (0.75) [0.42-1.08]	0.0006*
Cervical	1.29 (0.81) [0.94-1.64]	0.54 (0.64) [0.26-0.82]	<.0001*
Regional Right TS (SD) [CI]			
Cephalic	1.12 (0.92) [0.72-1.52]	0.58 (0.70) [0.27-0.89]	0.0004*
Cervical	1.31 (0.99) [0.88-1.74]	0.60 (0.73) [0.28-0.92]	<0.0001*
Individual Left TS (SD) [CI]			
Levator	1.20 (0.69) [0.90-1.50]	0.45 (0.60) [0.19-0.71]	0.001*
Suboccip	1.20 (0.83) [0.84-1.56]	0.50 (0.61) [0.23-0.77]	0.006*
SCM	1.50 (0.83) [1.14-1.86]	0.80 (0.77) [0.46-1.14]	0.012*
UT	1.25 (0.91) [0.85-1.65]	0.40 (0.50) [0.18-0.62]	0.002*
Temporal	1.60 (0.82) [1.24-1.96]	1.00 (0.73) [0.68-1.32]	0.021*
Masseter	1.35 (0.88) [0.96-1.74]	0.70 (0.92) [0.30-1.10]	0.020*
Frontal	0.80 (0.69) [0.50-1.10]	0.55 (0.51) [0.33-0.77]	0.267
Individual Right TS (SD) [CI]			
Levator	1.00 (0.86) [0.62-1.38]	0.45 (0.60) [0.19-0.71]	0.030*
Suboccip	1.35 (0.99) [0.92-1.78]	0.45 (0.60) [0.19-0.71]	0.002*
SCM	1.40 (1.14) [0.90-1.90]	0.80 (0.69) [0.50-1.10]	0.102

UT	1.40 (0.99) [0.97-1.83]	0.75 (0.91) [0.35-1.15]	0.035*
Temporal	1.30 (0.92) [0.9-1.70]	0.95 (0.83) [0.59-1.31]	0.254
Masseter	1.15 (0.93) [0.74-1.56]	0.70 (0.73) [0.38-1.02]	0.096
Frontal	0.90 (0.85) [0.53-1.27]	0.25 (0.44) [0.06-0.44]	0.006*

C, asymptomatic controls; SD, Standard Deviation; [CI], 95% Confidence Interval; TTS, total tenderness score; TS, tenderness score; Levator, levator scapula; Suboccip, suboccipital; SCM, sternocleidomastoid; UT, upper trapezius; n, number of participants; ^a Statistically significant difference when $p < 0.05$ (*) with the Mann-Whitney U-Test

Figure 1. Correlations in the Headache-group between: Left: Cervical extension and TTS ($\rho=0.78$), Middle: Cervical extension and cephalic TS ($\rho=0.74$) and Right: Cervical extension and cervical TS ($\rho=0.68$) (TTS = total tenderness score, TS = tenderness score, $^{\circ}$ = degree).