Pericranial tenderness in females with episodic cervical headache versus asymptomatic controls

Peer-reviewed author version

MINGELS, Sarah & GRANITZER, Marita (2018) Pericranial tenderness in females with episodic cervical headache versus asymptomatic controls. In: JOURNAL OF MANIPULATIVE AND PHYSIOLOGICAL THERAPEUTICS, 41(6), p. 488-495.

DOI: 10.1016/j.jmpt.2017.11.005
Handle: http://hdl.handle.net/1942/25229

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

Pericranial tenderness in females with episodic cervical headache versus asymptomatic controls

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 are 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 intensity[37] (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 thoracal.[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 × 2 × 3 (insertion × right/left × 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 (± 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 rho. 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 (ρ = 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.

References

1. Rinne M, Garam S, Hakkinen A, Ylinen J, Kukkonen-Harjula K, Nikander R. Therapeutic exercise training to reduce chronic headache in working women: Design of a randomized controlled trial. Phys Ther. 2016;96(5):631-40.

2. Bogduk N. Anatomy and physiology of headache. Biomed Pharmacother. 1995;49(10):435-45.

3. Fernandez-de-Las-Penas C, Cuadrado ML, Pareja JA. Myofascial trigger points, neck mobility, and forward head posture in episodic tension-type headache. Headache. 2006b;47(5):662-72.

4. Knackstedt H, Bansevicius D, Aaseth K, Grande RB, Lundqvist C, Russell MB. Cervicogenic headache in the general population: the Akershus study of chronic headache. Cephalalgia. 2010;30(12):1468-76.

5. Bennie JA, Chau JY, van der Ploeg HP, Stamatakis E, Do A, Bauman A. The prevalence and correlates of sitting in European adults - a comparison of 32 Eurobarometer-participating countries. Int J Behav Nutr Phys Act. 2013;1:107.

6. Kanchanomai S, Janwantanakul P, Pensri P, Jiamjarasrangsi W. Prevalence of and factors associated with musculoskeletal symptoms in the spine attributed to computer use in undergraduate students. Work. 2012;43(4):497-506.

7. Noack-Cooper K, Sommerich CM, Mirka GA. College students and computers: assessment of usage patterns and musculoskeletal discomfort. Work. 2009;32(3):285-98.

8. Malinska M, Bugajska J. The influence of occupational and non-occupational factors on the prevalence of musculoskeletal complaints in users of portable computers. Int J Occup Saf Ergon. 2010;16(3):337-43.

9. Jung SI, Lee NK, Kang KW, Kyoung K, Lee DY. The effect of smartphone usage time on posture and respiratory function. J Phys Ther Sci. 2016;28(1):186-89.

10. Kim SY, Koo SJ. Effect of duration of smartphone use on muscle fatigue and pain caused by forward head posture in adults. J Phys Ther Sci. 2016;28(6):1669-72.

11. Saito S, Miyao M, Kondo T, Sakakibara H, Toyoshima H. Ergonomic evaluation of working posture of VDT operation using personal computer with flat panel display. Ind Health. 1997;35(2):264-70.

12. Straker L, Jones KJ, Miller J. A comparison of the postures assumed when using laptop computers and DT computers. Appl Ergon. 1997;28(4):263-69.

13. Vasavada AN, Nevins DD, Monda SM, Hughes E, Lin DC. Gravitational demand on the neck musculature during tablet computer use. Ergonomics. 2015;58(6):990-1004.

14. Abboud J, Marchand A, Sorra K, Descarreaux M. Musculoskeletal physical outcome measures in individuals with tension-type headache: a scoping review. Cephalalgia. 2013;33(16):1319-36.

15. Fernandez-de-Las-Penas C. Tension-Type and Cervicogenic Headache, Pathophysiology, Diagnosis and Management. 1st ed. Massachusetts (US): Jones and Bartlett Publishers, 2010.

16. Fernandez-de-las-Penas C, Alonso-Blanco C, Cuadrado ML, Pareja JA. Forward head posture and neck mobility in chronic tension-type headache: a blinded, controlled study. Cephalalgia. 2006;26:314-19.

17. Jull G, Grieve GP. Modern Manual Therapy of the Vertebral Column. 1st ed. Edinburgh (UK): Churchill Livingstone, 1986.

18. Luedtke K, Boissonnault W, Caspersen N, Castien R, Chaibi A, Falla D, Fernandez-de-las-

Penas C, Hall T, Hirsvang JR, Horre T, Hurley D, Jull G, Kroll LS, Madsen BK, Mallwitz J, Miller C, Schafer B, Schottker-Koniger T, Starke W, von Piekartz H, Watson D, Westerhuis P, May A. International consensus on the most useful physical examination tests used by physiotherapists for patients with headache: A Delphi study. Man Ther. 2016;23:17-24.

19. Sjaastad O, Fredriksen TA. Cervicogenic headache: criteria, classification and epidemiology. Clin Exp Rheumatol. 2000;18:S3-6.

20. McGill SM, Brown S. Creep response of the lumbar spine to prolonged full flexion. Clin Biomech. 1992;7:43–6.

21. Westgaard RH, Winkel J. Guidelines for occupational musculoskeletal load as a basis for intervention: a critical review. Appl Ergon. 1996;27:79-88.

22. Briggs A, Straker L, Greig A. Upper quadrant postural changes of school children in response to interaction with different information technologies. Ergonomics. 2004;47:790–819.

23. Nijs J, van Wilgen CP. Als pijn chronisch wordt, revalidatie van patiënten met chronische pijn. 1st ed. Antwerpen (BE): Standaard Uitgeverij, 2009.

24. Lai TH, Protsenko E, Cheng YC, Loggia ML, Coppola G, Chen WT. Neural Plasticity in Common Forms of Chronic Headaches. Neural Plast. 2015. doi: 10.1155/2015/205985. PubMed PMID: 26366304; PMCID: PMC4558449.

25. de Tommaso M, Fernandez-de-las-Penas C. Tensiontype headache. Curr Rheumatol Rev. 2016;12:127–39.

26. Ashina A, Babenko L, Jensen R, Ashina M, Mageri W, Bendsten L. Increased muscular and cutaneous pain sensitivity in cephalic region in patients with chronic tension-type headache. Eur J Neurol. 2005;12:543–49.

27. Palacios Ceña M, Castaldo M, Kelun Wang, Torelli P, Pillastrini P, Fernández-de-Las-Peñas C, Arendt-Nielsen L. Widespread pressure pain hypersensitivity is similar in women with

frequent episodic and chronic tension-type headache: A blinded case-control study. Headache. 2017;57(2):217-25.

28. Bendtsen L, Fernandez-de-Las-Penas C. The role of muscles in tension-type headache. Curr Pain Headache Rep. 2001;15(6):451-58.

29. Midgette LA, Scher AI. The epidemiology of chronic daily headache. Curr Pain Headache Rep. 2009;13(1):59-63.

30. Voight A, Gould H. Chronic Daily Headache: Mechanisms and Principles of Management. Curr Pain Headache Rep. 2016;20(2):1-10.

31. Kroner-Herwig B, Gassmann J, Tromsdorf M, Zahrend E. The effects of sex and gender role on responses to pressure pain. Psycho-social medicine. 2012;9:1-10.

32. Banks K. Rehabilitation of movement: theoretical basis of clinical practice. London (UK): Saunders, 1998.

33. Soee AL, Skov L, Kreiner S, Tornoe B, Thomsen LL. Pain sensitivity and pericranial tenderness in children with tension-type headache: a controlled study. J Pain Res. 2013;6:425-34.

34. Bendtsen L, Jensen J, Jensen NK, Olesen J. Pressure-controlled palpation: a new technique which increases the reliability of manual palpation. Cephalalgia. 1995;15(3):205–10.

35. Langemark M, Olesen J. Pericranial tenderness in tension headache. A blind, controlled study. Cephalalgia. 1987;7(4):249-55.

36. Tornøe B, Andersen LL, Skotte JH, Jensen R, Gard G, Skov L, et al. Test-retest repeatability of strength capacity, aerobic power and pericranial tenderness of neck and shoulder muscles in children - relevant for tension-type headache. J Pain Res. 2013;6:643-51.

37. Jensen MP, Turner JA, Romano JM, Fisher LD. Comparative reliability and validity of chronic pain intensity measures. Pain. 1999;83(2):157-62.

38. neuro.be [Internet]. Belgian Headache Society [cited 2015 Dec 25]. available from:

http://www.neuro.be/bhs/

39. Williams MA, McCarthy CJ, Chorti A, Cooke MW, Gates S. A systematic review of reliability and validity studies of methods for measuring active and passive cervical range of motion. J Manipulative Physiol Ther. 2010;33(2):138-55.

40. Norkin CC, White DJ. Measurement of joint movement. A guide to goniometry. 5th ed. Philadelphia (US): Davis Company, 2009.

41. Mingels S, Dankaerts W, van Etten L, Thijs H, Granitzer M. Comparative analysis of head-tilt and forward head position during laptop use between females with postural induced headache and healthy controls. J Bodyw Mov Ther. 2016;20(3):533-41.

42. Lipchik GL, Holroyd KA, Talbot F, Greer M. Pericranial muscle tenderness and exteroceptive suppression of temporalis muscle activity: A blind study of chronic tension-type headache. Headache. 1997;37(6):368-76.

43. Chua NH, van Suijlekom HA, Vissers KC, Arendt-Nielsen L, Wilder-Smith OH. Differences in sensory processing between chronic cervical zygapophysial joint pain patients with and without cervicogenic headache. Cephalalgia. 2011;31(8):953-63.

44. Fernandez-de-Las-Penas C, Courtney CA. Clinical reasoning for manual therapy management of tension type and cervicogenic headache. J Manipulative Physiol Ther. 2014;22(1):44–50.

45. Hylleraas S, Davidsen EM, Benth JS, Gulbrandsen P, Dietrichs E. The usefulness of testing head and neck muscle tenderness and neck mobility in acute headache patients. Funct Neurol. 2010;25(1):27-31.

46. Christensen MB, Bendtsen L, Ashina M, Jensen R. Experimental induction of muscle tenderness and headache in tension-type headache patients. Cephalalgia. 2005;25(11):1061-67.

47. Brink Y, Louw Q, Grimmer K, Jordaan E. The relationship between sitting posture and seated-related upper quadrant musculoskeletal pain in computing South African adolescents: A

prospective study. Man Ther. 2015;20(6):820-26.

48. Drummond P, Knudsen L. Central pain modulation and scalp tenderness in frequent episodic tension-type headache. Headache. 2011;51(3):375-83.

49. Aaseth K, Grande R, Lundqvist C, Russell MB. Pericranial tenderness in chronic tension-type headache: the Akershus population-based study of chronic headache. J Headache Pain. 2014;15:58.

50. Fernandez-de-Las-Penas C, Cuadrado ML, Arendt-Nielsen L, Ge H, Pareja JA. Increased pericranial tenderness, decreased pressure pain threshold, and headache clinical parameters in chronic tension-type headache patients. Clin J Pain 2007;23(4):346-52.

51. Palacios-Cena M, Fernandez-Munoz JJ, Cigaran-Mendez M, Moron-Verdasco A, Fernandez-de-Las-Penas C. Association between the frequency and duration, but not the intensity, of headache with mechanical hypersensitivity and the health of patients with tension type headache. Rev Neurol. 2015;60(6):241-48.

52. Fernandez-de-Las-Penas C, Alonso-Blanco C, Cuadrado ML, Gerwin RD, Pareja JA. Myofascial trigger points and their relationship to headache clinical parameters in chronic tension-type headache. Headache. 2006a;46(8):1264-72.

53. Russek LN, Errico DM. Prevalence, injury rate and, symptom frequency in generalized joint laxity and joint hypermobility syndrome in a "healthy" college population. Clin Rheumatol. 2016;35(4):1029-39.

54. Sohn JH, Choi HC, Lee SM, Jun AY. Differences in cervical musculoskeletal impairment between episodic and chronic tension-type headache. Cephalalgia. 2010;30(12):1514-23.

55. Chen J, Solinger AB, Poncet JF, Lantz CA. Meta-analysis of normative cervical motion. Spine (Phila Pa 1976). 1999;24:1571-78.

56. LaBan MM, Meerschaert JR. Computer-generated headache. Brachiocephalgia at first byte. Phys Med Rehabil Clin. 1989;68(4):183-85.

57. Rozen TD, Roth JM, Denenberg N. Cervical spine joint hypermobility: a possible predisposing factor for new daily persistent headache. Cephalalgia. 2006;26(10):1182-85.
58. Tuttle N, Barrett R, Laakso L. Posteroanterior movements in tender and less tender locations of the cervical spine. Man Ther. 2009;14(1):28-35.

Tables

Table 1. Inclusion criteria for the Headache-group

Inclusion criteria	
Characteristics	Episodic headache Cervical stiffness Headache worsens with provocative manoeuvres/postures At least two of the following characteristics: 1. pressing or tightening (non-pulsating) 2. mild or moderate intensity 3. reduced cervical ROM 4. neck pain related to the headache
Provocation	Headache provoked by at least one of the following: 1. Poor cervical posture (e.g. forward head posture) 2. Sitting posture 3. Repetitive cervical movement 4. Prolonged posture
Autonomous	1. no nausea or vomiting 2. no photophobia or phonophobia
Duration	At least 10 episodes of headache occurring on 1- 14 days per month on average for >3 months (≥ 12 and ≤ 180 d/y) and lasting from 30 minutes to 7 days

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]			
Cephalic	1.18 (0.88) [0.79-1.57]	0.68 (0.74) [0.36-1.00]	0.0001*
Cervical	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 (ρ=0.78), Middle: Cervical extension and cephalic TS (ρ=0.74) and Right: Cervical extension and cervical TS (ρ=0.68) (TTS = total tenderness score, TS = tenderness score, ° = degree).