Pain-Pressure Threshold in the Head and Neck Region of Episodic Tension-type Headache Patients

Hyung-Suk Kim, DDS
Graduate Student
Department of Oral Medicine and Oral Diagnosis

Sung-Chang Chung, DDS, PhD
Professor
Department of Oral Medicine and Oral Diagnosis

Young-Ku Kim, DDS, PhD
Professor
Department of Oral Medicine and Oral Diagnosis

Sung-Woo Lee, DDS, PhD
Professor
Department of Oral Medicine and Oral Diagnosis

Seoul National University
College of Dentistry
Seoul, Korea

Correspondence to:
Dr Sung-Chang Chung
Orofacial Pain Clinic
Department of Oral Medicine and Oral Diagnosis
Seoul National University
College of Dentistry
28-2 Yunkeun-Dong, Chongro-Ku
Seoul, 110-744, Korea

Pain-pressure thresholds of the head and neck region of 31 female patients (aged 13 to 50 years; mean, 28.4 ± 9.6 years) suffering from episodic tension-type headache and 32 female control subjects (aged 15 to 46 years; mean, 26.6 ± 8.6 years) were recorded with an electronic algometer by the same blinded observer. The multivariate analysis of variance revealed that the algometer values obtained from different age groups of patients and control subjects were statistically different, but the values for the right-side muscles were not statistically different from the corresponding values for the left-side muscles. The pain-pressure thresholds of the patient group were lower than those of the control group for the superior sternocleidomastoid muscles, middle sternocleidomastoid muscles, and trapezius insertion muscles ($P < .01$) but were not statistically different for the anterior temporal, middle temporal, posterior temporal, deep masseter, anterior masseter, inferior masseter, medial pterygoid, posterior digastric, splenius capitis, and upper trapezius muscles ($P > .01$). The results may indicate that pain-pressure thresholds of the head and neck region should be considered in the diagnosis of episodic tension-type headache. The results may also propose that the increased pain sensitivity of the head and, especially, the neck region, may be included in the pathogenetic mechanism in episodic tension-type headache.
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key words: pain-pressure threshold, tension-type headache, algometer, head and neck muscles

It has been estimated that one in three people suffers from severe headaches at some stage of his/her life. More than 30 million pounds of aspirin—of which at least a major part is taken for the relief of headache—is consumed annually.[1] Only a minority of sufferers, such as those with migraine or cluster headache, display a very distinct symptomatology. The great majority have a constant, dull, aching pain unassociated with other symptoms. In the general population, these headaches are often called *tension headaches, tension* probably alluding to tension from stress as well as tension in the pericranial muscles.[2]

There remains much controversy surrounding the pathogenetic mechanism in tension-type headache. Contraction of pericranial muscles and/or increased pain sensitivity has been thought to play a role in the pathogenesis of tension-type headache.[3,4] Sustained contraction of muscles is mentioned as a main feature, but no guidelines are given for the verification of sustained muscular contraction in an individual.[5] Because many investigators have found increases in tenderness of the pericranial muscle or of pericranial

electromyographic resting levels in only a fraction of patients suffering from tension-type headache,[5–7] they doubted the credibility of the concept of marked tonic muscle contraction as the direct cause of headaches.[2] However, the pericranial muscles examined in the above studies[5–7] were selected based on their presumed relationship to the pain without scientific evidence. The alternative mechanisms for tension-type headache, such as central mechanisms described by Schoenen et al,[8] cannot explain the pain alone.

Furthermore, tension-type headache can be classified more specifically as chronic or episodic. Because these two forms have different characteristics,[9] they might also differ from a pathophysiologic point of view. Therefore, the mechanism of tension-type headache is still unclear and should be studied separately in its chronic and episodic forms.

A simple and obvious approach to the evaluation of muscles involved in tension-type headache is palpation of the muscles with demonstration of abnormal muscle tenderness. However, the process of palpation is very subjective. For this reason, different algometers have been used to determine pain-pressure thresholds (PPTs) in clinical situations, and the reliability and validity of these algometers when used on the human stomatognathic system has been reported to be acceptable.[10,11]

The aim of this study was to measure the PPTs in the head and neck region of episodic tension-type headache sufferers and in headache-free control subjects to help determine which muscular areas should be the focus of research on the pathogenetic mechanisms underlying episodic tension-type headache.

Materials and Methods

Subjects

Thirty-one subjects (females aged 13 to 50 years; mean, 28.4 years; standard deviation, 9.6 years) who had episodic tension-type headaches and 32 matched headache-free control subjects (females aged 15 to 46 years; mean, 26.6 years; standard deviation, 8.6 years) were included in the study. Criteria for control subjects included: *(1)* self-report evidence that they had never considered themselves to be headache sufferers; and *(2)* self-report evidence that they experienced, at most, six mild headaches per year.[12] A questionnaire and a detailed interview were used to select headache subjects who met diagnostic criteria for episodic tension-type headaches.[13] The subjects with episodic tension-type headaches were tested during intervals between headache attacks. All subjects were free from any major medical or psychiatric diagnoses. Headache and control subjects did not differ significantly in age distribution ($P > .4$).

Apparatus

The electronic algometer type I (Somedic, Stockholm, Sweden) was used to record the PPTs of the subjects. When the subject first felt pain, she pushed the button on the patient-operated switch to indicate the first point at which pain occurred. The digital display of the PPT stopped immediately for about 5 seconds and a red light turned on so that the operator could record the value easily (Figs 1a and 1b).

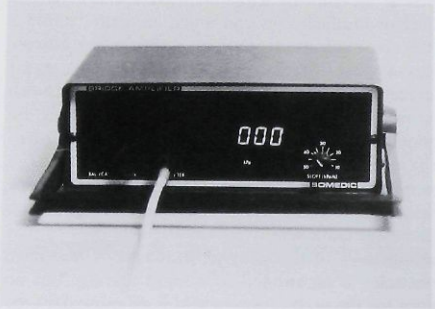

Fig 1a Electronic algometer used in the study.

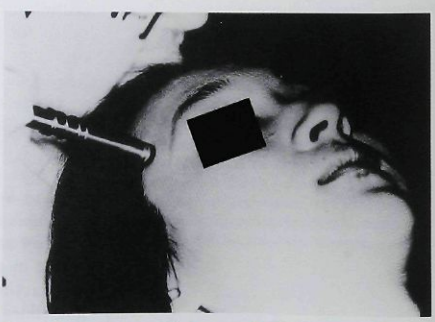

Fig 1b Recording of the PPTs with the electronic algometer.

Recording Procedure

Before the examination, the preliminary application of the pressure algometer to the frontal muscle for the reference point was performed one or two times for each subject. This allowed the subject to be familiar with the procedure. No information about the subjects' headache history was available to the observer, and all subjects were unknown to the observer.

The palpated points recommended by previous researchers[14–16] were modified and used for the present study. Following are the muscles (Fig 2) and the methods used in the current study for palpation with the electronic algometer:

1. Anterior temporal muscle: The subject is asked to clench and relax to help identify the muscle. The fibers above the infratemporal fossa and immediately above the zygomatic process are palpated.
2. Middle temporal muscle: Fibers in the depression about 2 cm lateral to the lateral border of the eyebrow are palpated.
3. Posterior temporal muscle: The subject is asked to clench and relax to help identify the muscle. The superior fibers behind the ears to directly above the ears are palpated.
4. Deep masseter muscle: Superior fibers immediately below the notch in the zygomatic arch are palpated with the subject's mouth closed.
5. Anterior masseter muscle: The subject is asked to clench while the masseter is observed for the location. Fibers of the anterior border are palpated immediately below the zygomatic arch.
6. Inferior masseter muscle: The area 1 cm superior and anterior to the angle of the mandible is palpated.
7. Medial pterygoid muscle: The area under the mandible at a point 2 cm anterior to the angle of the mandible is palpated superiorly. If this area is not easily accessible, the subject's jaw should be in laterotrusion to the ipsilateral side when palpated.
8. Posterior digastric muscle: The area immediately behind the mandible at a point 2 cm superior to the angle of the mandible is palpated.
9. Superior sternocleidomastoid muscle (SCM): The fibers immediately below the mastoid process are palpated.
10. Middle SCM: The subject is asked to rotate the neck to the contralateral side to help identify the muscle. Midpoint fibers between the superior SCM point and the insertion point of the SCM are palpated.
11. Splenius muscle of the head: The midpoint between the superior SCM point and the trapezius muscle insertion point is palpated.
12. Trapezius insertion muscle (TIM): The superior insertion of the trapezius muscle immediately below the occipital bone is palpated.
13. Upper trapezius muscle: Midpoint fibers overlying the upper border of the muscle between the shoulder angle and the midline are palpated.

Both right and left muscles were palpated, making a total of 26 points in all subjects.

To avoid experimental bias, the 26 points were

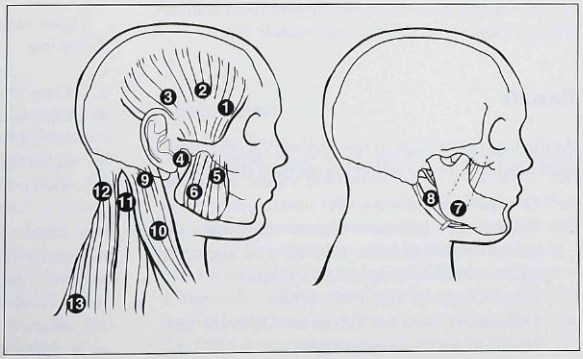

Fig 2 Location of the areas palpated. (1 = anterior temporal muscle; 2 = middle temporal muscle; 3 = posterior temporal muscle; 4 = deep masseter muscle; 5 = anterior masseter muscle; 6 = inferior masseter muscle; 7 = medial pterygoid muscle; 8 = posterior digastric muscle; 9 = superior SCM; 10 = middle SCM; 11 = splenius muscle of the head; 12 = TIM; 13 = upper trapezius muscle).

Table 1 Result of MANOVA by the Initial Model

Hypothesis of no statistical differences between subject groups (headache versus control)					
Statistic*	Value	F	Num DF	Den DF	$P > F$
Wilks' lambda	0.48405946	8.9368	13	109	.0001
Pillai's trace	0.51594054	8.9368	13	109	.0001
Hotelling-Lawley trace	1.06586189	8.9368	13	109	.0001
Roy's greatest root	1.06586189	8.9368	13	109	.0001
Hypothesis of no statistical differences among age groups					
Statistic*	Value	F	Num DF	Den DF	$P > F$
Wilks' lambda	0.59137399	2.5185	26	218	.0002
Pillai's trace	0.44425926	2.4163	26	220	.0003
Hotelling-Lawley trace	0.63072230	2.6199	26	216	.0001
Roy's greatest root	0.51334507	4.3437	13	110	.0001
Hypothesis of no statistical differences between locations (left versus right)					
Statistic*	Value	F	Num DF	Den DF	$P > F$
Wilks' lambda	0.91188310	0.8102	13	109	.6483
Pillai's trace	0.08811690	0.8102	13	109	.6483
Hotelling-Lawley trace	0.09663179	0.8102	13	109	.6483
Roy's greatest root	0.09663179	0.8102	13	109	.6483

*The F statistic for Roy's greatest root is an upper bound. The F statistic for Wilk's lambda is exact.
Num DF = numerator's degree of freedom; Den DF = denominator's degree of freedom.

palpated in random order and the subjects could not see the digital display. The palpation was conducted bilaterally with a standard application rate of 40 kPa/sec.

Statistical Analysis

For the comparison of the PPTs in control subjects versus headache subjects, the multivariate analysis of variance (MANOVA) in the Statistical Analysis System (SAS) (1988) was used. After finding the statistically significant differences between the headache and control subjects, the MEANS in SAS (1988) was used to obtain the simultaneous individual confidence intervals for each muscle type.

Results

At the beginning stage of the MANOVA, the model used consisted of the following parts of the PPT:

1. Overall effect (2 + 3 + 4)
2. Differences between algometer values obtained from headache subjects and algometer values obtained from control subjects
3. Effect of age on algometer values
4. Differences between values for different muscle locations (left versus right)

There were 13 PPT responses corresponding to 13 individual muscles on the left, and 13 PPT responses corresponding to 13 individual muscles on the right. Each subjects was put into one of three groups: 13 to 19 years old; 20 to 29 years old; and 30 years and older. The results of the analysis under this model are summarized in Table 1, which revealed that *(1)* values for the right-side muscles were not statistically different from the corresponding values for the left-side muscles ($P > .6$); *(2)* values from headache subjects were statistically different from those of control subjects; and *(3)* values from different age groups were statistically different.

Those results led us to adopt a model with the following:

1. Overall effect (2 + 3)
2. Differences between algometer values obtained from headache subjects and algometer values obtained from control subjects
3. Effect of age on algometer values

The results of the analysis under this model are summarized in Table 2, which shows that *(1)* values from headache subjects were statistically different from those of control subjects ($P < .01$); and *(2)* values from different age groups were statistically different ($P < .01$).

Table 2 Result of MANOVA by the Final Model

Hypothesis of no statistical differences between subject groups (headache versus control)					
Statistic*	Value	F	Num DF	Den DF	$P > F$
Wilks' lambda	0.48411150	9.0170	13	110	.0001
Pillai's trace	0.51588850	9.0170	13	110	.0001
Hotelling-Lawley trace	1.06563986	9.0170	13	110	.0001
Roy's greatest root	1.06563986	9.0170	13	110	.0001
Hypothesis of no statistical differences among age groups					
Statistic*	Value	F	Num DF	Den DF	$P > F$
Wilks' lambda	0.59440491	2.5136	26	220	.0002
Pillai's trace	0.44086124	2.4143	26	222	.0003
Hotelling-Lawley trace	0.62302472	2.6199	26	218	.0001
Roy's greatest root	0.50570243	4.3179	13	111	.0001

*The F statistic for Roy's greatest root is an upper bound. The F statistic for Wilk's lambda is exact.
Num DF = numerator's degree of freedom; Den DF = denominator's degree of freedom.

Table 3 Simultaneous Confience Intervals (99%) for PPTs Measured With an Algometer

	Subject		
	Headache (n = 31)	Control (n = 32)	Significance
Anterior temporal	289.77 ± 26.23	294.61 ± 25.82	NS
Middle temporal	327.44 ± 28.37	318.78 ± 27.82	NS
Posterior temoral	370.31 ± 35.16	347.86 ± 34.60	NS
Deep masseter	246.94 ± 23.16	228.69 ± 22.80	NS
Anterior masseter	223.26 ± 19.30	243.91 ± 19.00	NS
Inferior masseter	206.63 ± 18.28	214.88 ± 18.00	NS
Medial pterygoid	156.60 ± 19.31	176.84 ± 19.00	NS
Posterior digastric	164.77 ± 16.88	183.48 ± 16.61	NS
Superior SCM	185.32 ± 18.97	246.33 ± 18.68	*
Middle SCM	157.94 ± 17.95	215.55 ± 17.67	*
Splenius muscle of the head	219.40 ± 21.64	212.44 ± 21.31	NS
Trapezius insertion	231.87 ± 21.74	295.91 ± 21.40	*
Upper trapezius	284.11 ± 30.52	291.45 ± 30.04	NS

SCM = Sternocleidomastoid muscle.
NS = not significant ($P > .01$).
*$P < .01$.

The MEANS procedure has been done as a post hoc analysis to gain insight into which muscle types contribute to the statistically significant differences. The results are summarized in Table 3 in terms of 99% simultaneous confidence intervals. The PPTs were significantly lower in subjects suffering from episodic tension-type headaches than in control subjects at only three cervical sites (superior SCM, middle SCM, and TIM) ($P < .01$). The differences among PPTs at the other 10 sites were not statistically significant ($P > .01$).

Discussion

The present study suggests that cervical muscles are more tender than masticatory muscles in subjects with tension-type headaches. In the evaluation of muscle tenderness, either apparatus or simple digital palpation can be applied. Using a pressure algometer to obtain PPT measurements is a reproducible way of studying pain sensitivity,[17–19] and the reliability of the device used here has been found acceptable in previous studies.[10,11]

Although the reliability for the medial pterygoid muscle and posterior digastric muscle sites were acceptable, some researchers doubt the reliability for these muscles due to the difficult anatomic accessibility. Therefore, the PPTs of these muscles should be interpreted with caution.

According to many earlier studies using the algometer or digital palpation, tension-type headache patients have increased tenderness of pericranial muscles, especially in the head region.[5,7,8,19–22] However, one study[14] did show that there were no statistically significant differences in PPTs between control subjects and tension-type headache patients. In this study,[14] the PPTs were not statistically different in all pericranial muscles but rather in only three neck muscle sites (superior SCM, middle SCM, TIM). The different results from Bovim's study[14] might be the result of differences in diagnostic criteria or characterization of subjects. For other studies, many methodologic differences could be found in comparison with the present study.

The present study seems to show that decreased PPT of pericranial muscles in the neck is related to the episodic tension-type headache. This finding differs from other studies[5–8] that show statistically significant differences in temporal and other masticatory muscles between control subjects and tension-type headache subjects. The varied results might be caused by various factors such as the area of pain, diagnostic criteria, the size of contact area, and the application rate of the algometer, or other methodologic issues.[10] Although we did not record specific areas of pain, previous research supports that specific muscles cause pain in specific areas. Our subjects had more occipital and frontal pain, common to tenderness in neck muscles.

In this study, no statistically significant difference between the PPTs in control subjects and headache subjects was found in 10 pericranial masticatory muscles and in some cervical muscles. Because of this specificity of tenderness, the results of this study do not support the hypothesis that there is a diffuse disruption of the central pain modulating system as one of the pathophysiologic hallmarks of tension-type headache.[23] In contrast, the presence of more localized pericranial disruption of muscular nociception is supported.

In addition, some subjects with tension-type headaches did not apparently have decreased PPTs in any muscle of the head or neck region, and some subjects did show markedly decreased PPTs but did not suffer from headaches. This result seems to suggest that there is no simple direct causative relationship between the increased pain sensitivity of the head and neck region and episodic tension-type headache. Or, it could be hypothesized that the generation of headaches takes place in the brain itself, and the effect is a pericranial localized tenderness in the muscles.[4] Despite theoretical implications, the present study does support the hypothesis that nociception is primarily myofascial in origin, but supraspinal facilitation may play a large role in tension-type headache.[23] Furthermore, the results of this study support the localization of nociception from SCM and TIM areas, and this may be the primary characteristic of episodic tension-type headaches of the headache subjects of this study.

Because a number of factors such as the size of contact area and the rate of application[10] may influence the results obtained, the simple comparison with other studies has little meaning. It is suggested that a more extensive study include a much larger control group for the assessment of normal statistical distribution of the PPTs. In addition, more study on muscle tenderness and pain is needed. Further studies using other methods such as electromyography, nerve blocking, and exteroceptive suppression in the neck region are needed to clarify pathogenetic factors concerning episodic tension-type headache.

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Resumen

Umbral de presión-dolor en la región de la cabeza y cuello de pacientes que sufren de cefaleas episódicas relacionadas a la tensión

Se registraron los umbrales de presión-dolor de la región de la cabeza y cuello de 31 pacientes del sexo femenino (cuyas edades variaban entre los 13–50 años; media 28,4 ± 9,6 años) y quienes sufrían de cefaleas episódicas relacionadas a la tensión y 32 mujeres pertenecientes al grupo de control (cuyas edades variaban entre los 15–46 años; media 26,6 ± 8,6 años). Tales registros fueron efectuados con un algómetro electrónico por el mismo observador "ciego." El análisis de varianza multivariada reveló que los valores del algómetro obtenidos de diferentes grupos de edad en sujetos del grupo experimental y del grupo de control fueron estadísticamente diferentes, pero los valores para los músculos del lado derecho no fueron estadísticamente diferentes de los valores correspondientes a los músculos del lado izquierdo. Los umbrales de presión-dolor del grupo experimental fueron menores que aquellos del grupo de control en el caso de los músculos esternocleidomastoideos, esternocleidomastoideos medios, y las inserciones del trapecio ($P > 0,01$), pero no fueron estadísticamente diferentes en el caso de los músculos temporal anterior, temporal medio, temporal posterior, masetero profundo, masetero anterior, masetero inferior, pterigoideo medio, digástrico posterior, esplenio craneal, y trapecio superior ($P < 0,01$). Los resultados pueden indicar que los umbrales de presión-dolor de la región de la cabeza y el cuello deberían ser considerados en el diagnóstico de las cefaleas episódicas relacionadas a la tensión. Los resultados también pueden indicar que la mayor sensibilidad al dolor en la cabeza y, especialmente en la región del cuello, puede ser incluida en el mecanismo patogenético de las cefaleas episódicas relacionadas a la tensión.

Zusammenfassung

Die Druckschmerz-Schwelle in der Kopf- und Nackenregion von Patienten mit episodischem Spannungstyp Kopfweh

Die Druckschmerz-Schwelle der Kopf- und Nackenregion von 31 Frauen (Alter 13-50 Jahre, 28,4 ± 9,6 Jahre) mit episodischem Spannungstypkopfweh und von 32 weiblichen Kontrollsubjekten (Alter 15–46 Jahre, 26,6 ± 8,6 Jahre) wurde mit einem elektronischen Algometer durch denselben Untersucher blind untersucht. Die Varianzanalyse ergab, dass die Werte aus verschiedenen Altersgruppen von Patienten und Kontrollsubjekten statistisch verschieden waren, dass aber die Werte der Muskeln auf der rechten Seite statistisch nicht von der entsprechenden Werten der andern Seite abwichen. Für die oberen und mittleren Anteile des M. sternocleidomastoideus und den Ansatz des M. trapezius war die Druckschmerzschwelle der Patientengruppe tiefer als diejenige der Kontrollgruppe ($P < 0,01$), aber sie war statistisch nicht signifikant verschieden für die anterioren, mittleren und posterioren Anteile des M. temporalis, die tiefen, anterioren und inferioren Portionen des M. masseter, den M. pterygoideus medialis, den posterioren Anteil des M. digastricus, den splenius capitis und die oberen Anteile des M. trapezius ($P > 0,01$). Diese Resultate können bedeuten, dass die Druckschmerz-Schwelle der Kopf- und Nackenregion in die Diagnose des episodischen Spannungstyp Kopfweh einbezogen werden muss. Die Resultate lassen ebenso annehmen, dass eine erhöhte Schmerzsensitivität des Kopfes und speziell des Nackens ihren Einfluss haben könnte im pathogenetischen Mechanismus des episodischen Spannungstyp Kopfweh.