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Central and peripheral mechanisms in chronic tension-type headache

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Summary

The second exteroceptive suppression of masseter muscle activity (ES2) and tenderness in pericranial muscles were evaluated in 112 young adults who met IHS criteria in the following diagnostic classifications: 31 chronic tension headache, 31 episodic tension headache, 33 migraine without aura and 17 migraine with aura. An additional 31 subjects served as controls. Pericranial muscle tenderness better distinguished diagnostic subgroups and better distinguished recurrent headache sufferers from controls than did masseter ES2. Chronic tension headache sufferers exhibited the highest pericranial muscle tenderness, and controls exhibited the lowest tenderness ($P < 0.01$). All chronic tension headache sufferers exhibited muscle tenderness in at least one of the pericranial muscles evaluated, while tenderness was exhibited by 52% of controls. The association between pericranial muscle tenderness and chronic tension headache was independent of the intensity, frequency, or chronicity of headaches. Our findings raise the possibility that pericranial muscle tenderness is present early in the development of tension headache, while ES2 suppression only emerges later in the evolution of the disorder.

Keywords

Headache; Migraine; Exteroceptive suppression; Masseter; Muscle tenderness

Introduction

The pathophysiology of chronic tension headache is not well understood. At one time, abnormal and sustained contraction of pericranial muscles was assumed to be the primary cause of pain (Ad Hoc Committee on Classification of Headache 1962); however, tension-type headache sufferers do not reliably exhibit either abnormal resting levels of pericranial electromyographic (EMG) activity, or abnormal levels of EMG activity in response to stress (see reviews by Andrasik et al. 1982; Pikoff 1984). In recent years, research attention has shifted to possible abnormalities in pericranial pain sensitivity and possible abnormalities in central pain modulation. Although results obtained in studies of thermal and pressure pain thresholds have been conflicting (e.g., Schoenen et al. 1991a,c; Jensen et al. 1993a), when tenderness has been identified by manual palpation, higher levels of pericranial muscle tenderness have been reported in tension-type headache sufferers than in headache-free controls (Lous and Olesen 1982; Drummond 1987; Langemark and Olesen 1987; Schoenen et al. 1991a; Hatch et al. 1992) or in migraineurs (Lous and Olesen 1982; Schoenen et al.

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1991a). Unfortunately, many of these investigations were conducted prior to the implementation of the new International Headache Classification System (IHS) and suffer from methodological problems such as the utilization of control groups that were not age and sex matched.

More recently, the second temporalis / masseter exteroceptive suppression period (ES2), or 'silent period' has been found to be significantly shortened or abolished in chronic tension headache, but not in migraine or in most other symptomatic headaches (Schoenen et al. 1987; Nakashima and Takahashi 1991; Wallasch et al. 1991; Gobel and Weigle 1991; Mathew 1992; Schoenen 1993a,b). Temporalis/masseter ES2 is a transient suppression of voluntary activity of temporalis and masseter muscles produced by stimulation of the trigeminal nerve, and is mediated in the brain stem by multisynaptic neuronal nets (Cruccu and Bowsher 1986). The inhibitory brain stem interneurons that mediate ES2 in jaw-closing muscles receive strong input from limbic pathways (Kupfers 1958; Nazaki et al. 1983; Yasui et al. 1985; Holstege and Van Krimpen 1986), some of which are serotonergic and are implicated in pain modulation (Holstege 1990). This suggests abnormal ES2 responses observed in chronic tension-type headache may reflect an excessive inhibition of these interneurons secondary to a disturbed limbic control of brain stem relays such as the periaqueductal gray and the raphe magnus nucleus (Schoenen et al. 1987). The shortened second exteroceptive silent period observed in chronic tension-type headache may thus index a dysfunction of the endogenous central pain control system (Schoenen et al. 1987; Wallasch et al. 1991), and represent an interface between the psychogenic and myogenic factors putatively involved in the pathogenesis of chronic tension-type headache (Schoenen et al. 1987).

Although the existing literature suggests that chronic tension headache sufferers exhibit a shortened or absent ES2 response and elevated levels of pericranial muscle tenderness, these studies have been conducted primarily in patient samples; observed abnormalities may thus be influenced by long-term recurrent headaches or medication use and/or concomitant physical or psychological disorders that are frequently observed in clinic samples (Gobel et al. 1992b). Subjects in many of the previous studies reported headache chronicity ranging from 10 to 18 years (e.g., Lous and Olesen 1982). Thus, these studies do not allow us to determine if a shortened or absent ES2 response or the pericranial muscle tenderness might play an etiological role in chronic tension-type headaches, or are consequences of recurrent headaches and their treatment. Examination of ES2 and pericranial muscle tenderness in young recurrent headache sufferers might help us determine if these abnormalities are present early in the development of the disorder. Thus, the present study utilized a sample of young adults carefully diagnosed according to IHS criteria and examined the ability of ES2 and pericranial muscle tenderness to distinguish chronic tension-type headache sufferers from other diagnostic groups of recurrent headache disorders and headache-free controls.

Materials and Methods

Subjects

112 female recurrent headache sufferers were identified in a large survey of college students. Subjects for this study met International Headache Society (IHS) criteria in the following diagnostic classifications: 31 chronic tension-type headache, 31 episodic tension-type headache, 33 migraine without aura and 17 migraine with aura. None of these subjects met IHS criteria for more than one type of headache: Tension-type headache sufferers did not have coexisting migraine, migraineurs did not have coexisting tension-type headache, migraine with aura subjects did not have coexisting migraine without aura and migraine without aura subjects did not have coexisting migraine with aura. An additional 31 females who did not experience problem headaches were recruited for the control group. Overall the mean age of the participants was 19 years (range 17–26), and no significant differences in age were observed

across groups. Because duration of ES2 decreases during menses compared to mid-cycle (Schoenen et al. 1991b), the stage of each subject's menstrual cycle was calculated on the day of assessment, and no significant differences were found across groups.

Over 3000 college students were administered a screening questionnaire that inquired about the subject's headache symptoms and headache history. Females who appeared to meet International Headache Society (IHS) criteria on the basis of their questionnaire responses were scheduled for a structured diagnostic interview in order to obtain additional information about the individual's headaches. The diagnostic interviews were conducted by advanced graduate students in psychology trained in the IHS diagnostic criteria for recurrent headache disorders. Only women who clearly met IHS diagnostic criteria in the structured diagnostic interview were included in the headache groups. Individuals who were currently taking antidepressant medication, reported more than one type of headache or presented with mixed headache symptomatology, or who reported a pain disorder other than headache were excluded. Controls reported fewer than 5 minor tension-type headaches per year, none of which was reported to be a problem headache. Prior to the assessment, subjects were given oral and written descriptions of the procedures and informed consent was obtained. Subjects were unpaid but received experimental credit in their introductory psychology courses for their participation in this investigation.

It is noteworthy that of the 3000 students screened, less than 2% of the females met IHS criteria for chronic tension-type headache (without coexisting migraine) and 1% of females met IHS criteria for migraine with aura (without coexisting tension-type headache or migraine without aura). The proportion of chronic tension-type headache sufferers and the proportion of migraine with aura sufferers are somewhat smaller than those reported previously (Rasmussen et al. 1991; Rasmussen 1995), and may be due to the young age of our sample and our strict screening for diagnostically distinct samples of the four major headache classifications (chronic tension-type, episodic tension-type, migraine without aura, migraine with aura).

Apparatus

Physiological recording—Masseter electromyographic (EMG) activity (in μV) was recorded using a pair of 10 mm Beckman bipolar electrodes (Sensor Medics, Yorba Linda, CA, USA), a Sensor-Medics silver earclip reference electrode and a World Precision Instruments (Sarasota, FL, USA) DAM-50 differential amplifier. Masseter EMG activity was collected and processed using RC Electronics (Goleta, CA, USA) Computer-scope data acquisition and analysis software, and a DELL (Austin, TX, USA) 486 personal computer. Stimulation for the assessment of ES2 was applied using a Digitimer DS7 constant-current electrical stimulator, a Digitimer DG2 trigger generator and an Electrode Store DDY-15 reusable pediatric bar electrode with a 15 mm spacing of 6 mm contacts.

Procedure—The assessments were conducted in a sound-attenuated room. For the ES2 assessment, subjects were seated in a heavily padded chair in a room adjacent to the recording and stimulation equipment. For the pericranial muscle tenderness assessment, subjects were seated in a straight-back chair with their feet flat on the floor and their hands resting uncrossed on their thighs.

ES2 assessment The right masseter muscle was palpated during voluntary contraction of the jaw. Electrode sites were cleansed with 70% isopropyl alcohol. The first masseter electrode was placed 2 cm from the most lateral point on the mandibular angle. The second masseter electrode was placed 2 cm (measured center to center) superior and slightly medial to the first electrode. TECA Electrolyte gel (TECA, NY, USA) was used as a conductive medium. Each

EMG placement was tested to ensure a maximum resistance of 10 k Ω . The earclip reference electrode was clipped to the right earlobe.

The stimulating electrode was vertically centered 5 mm lateral to the right labial commissure with anode superior to cathode, and the electrode site was prepared in the same manner as the EMG placement. Because a relatively consistent level of jaw clenching force must be maintained across stimulations, two additional Beckman electrodes were attached to the left masseter muscle, and an EMGJ33 electromyogram biofeedback unit (Cyborg, Boston, MA, USA) was used to provide visual and auditory feedback to the subject. A threshold level was preset, and the subject was told to clench her jaw hard enough to elicit a tone. Subjects placed a disposable Lancer Orthodontics (Carlsbad, CA, USA) Therabite rubber dental wafer between their upper and lower teeth during jaw occlusion. The intensity of the electrical stimulation began at 2 mA and was increased to 30 mA in increments of 2 mA. 10 square-wave stimulations were administered with an inter-stimulation interval of 10 s. Each stimulation was 30 mA and lasted 0.1 ms. A 250 ms sweep of EMG activity was collected for each stimulation. EMG recording began 75 ms prior to electrical stimulation onset and was terminated 175 ms after stimulation. Raw EMG activity was sampled at a rate of 2 kHz using a signal amplification gain setting of 1000 AC and a 10–3000 Hz bandwidth. 10 raw EMG sweeps were rectified and then averaged on-line to create a single wave form for scoring ES responses.

ES scoring Fig. 1 illustrates a sample waveform after ten individual 250 ms sweeps of EMG activity have been rectified, averaged and plotted in relation to pre-stimulus baseline levels. Exteroceptive suppression was defined as any reduction of EMG activity below a pre-stimulus baseline (Nakashima et al. 1990). The pre-stimulus baseline was defined as the mean level of EMG activity between –75.0 and –25.0 ms before electrical stimulation. Duration of ES2 was computed by measuring the length of time that EMG activity remained below the pre-stimulus baseline. Offset of ES2 was determined to occur when EMG activity returned to the pre-stimulus baseline level for more than 1 ms.

Pericranial muscle tenderness All subjects were headache-free during the examination for pericranial muscle tenderness. Three trained experimenters conducted the assessments with manual palpation, and evaluated the same number of subjects in each of the five groups. The experimenter palpated the pericranial muscles with the subject in a seated position. Manual pressure was applied to the suboccipital, posterior cervical, upper trapezius, masseter, sternocleidomastoid and temporalis muscles bilaterally. Palpation was performed systematically over the surface of the muscle by applying deep finger pressure while making small circular movements. The subject's response was recorded on a 4-point scale as follows: 1 = denial of tenderness; 2 = verbal report of mild pain and no withdrawal response; 3 = verbal report of moderate pain and slight withdrawal response; and 4 = verbal report of severe pain and vigorous withdrawal response, or verbal response that palpation reproduced typical headache pain (Langemark and Olesen 1987; Hatch et al. 1992). Muscles were evaluated for general tenderness as well as for trigger-point activity. A Total Tenderness Score (TTS) was calculated for each subject by adding the scores from each palpated muscle site (Langemark and Olesen 1987).

Statistics

Analyses of variance (ANOVAs) were used to compare the five groups on pericranial muscle tenderness, exteroceptive suppression and demographic variables (age, stage of menstrual cycle). For a comparison of muscle tenderness between experimenters, an ANOVA was computed. Post-test comparisons were made with Tukey's Honestly Significant Difference test which controls for Type I error due to multiple comparisons. For comparisons of frequency, intensity, and duration of headache between migraine groups, Student's *t*-test was used.

Comparisons between groups of number of subjects with tenderness in various muscle groups were made using χ^2 tests. Correlation coefficients were computed to examine any relation between pericranial muscle tenderness and headache variables (chronicity, frequency, headache intensity).

Results

Headache characteristics

The percentage of subjects in the four headache groups meeting each of eight IHS diagnostic criteria is presented in Table I. It can be seen that the migraine and tension-type headache groups were clearly distinguished by these diagnostic criteria. In addition, a family history of headache was reported by over three-quarters (77%) of the migraine with aura subjects, 58% migraine without aura subjects, 52% of chronic tension-type headache subjects and 45% of episodic tension-type headache subjects. Medical treatment for headache had been sought by 83% of migraine with aura subjects, 58% of chronic tension-type headache subjects, 42% of migraine without aura subjects and 19% of episodic tension-type headache subjects.

Table II lists the means and ranges for the four headache groups on headache frequency, chronicity and intensity, and duration of typical headache. It can be seen that the chronic tension-type headache subjects clearly share clinical features of patient samples of chronic tension-type sufferers ¹.

ES2 variables

Table III lists the means and standard deviations for the five groups on the latency and duration of the masseter ES2 ². Two one-way ANOVAs were conducted to compare mean ES2 scores across groups, and no statistically significant differences were found between the five groups, $P > 0.05$.

Pericranial muscle tenderness

A one-way ANOVA found no significant differences in pericranial muscle tenderness (TTS) among experimenters, $P > 0.05$. It can be seen in Table IV that chronic tension-type headache sufferers exhibited the highest TTS, and the control group exhibited the lowest scores. Subjects in the remaining three headache groups exhibited intermediate levels of muscle tenderness. A one-way ANOVA confirmed that the TTS of the five groups differed significantly ($F(4,137) = 5.91$, $P < 0.001$). Post-tests revealed that the chronic tension-type headache group exhibited significantly higher levels of pericranial muscle tenderness than migraine with aura and control groups (Tukey HSD, $P < 0.05$). The migraine without aura group also exhibited significantly higher pericranial muscle tenderness than the control group ($P < 0.05$).

TTS were uncorrelated with the frequency of headache ($r = 0.15$, $P > 0.05$), chronicity of headache ($r = 0.05$, $P > 0.05$), or typical pain intensity ($r = 0.12$, $P > 0.05$).

Muscle tenderness in a single muscle was common across groups and was reported by 100% of the chronic tension-type headache subjects, 80% of episodic tension-type headache subjects, 76% of both migraine groups and 52% of controls. We attempted to determine if significant muscle tenderness, defined as tenderness in more than one muscle group, distinguished chronic tension-type headache sufferers from subjects in the remaining groups. It can be seen in Fig. 2 that 90% of the chronic tension-type headache sufferers, but only 32% of the controls,

¹Migraineurs with aura and migraineurs without aura did not differ significantly on frequency, intensity, or duration of headache.

²Equipment difficulties were experienced that resulted in inadequate stimulation of 47 participants. The 47 cases were removed from the data base, leaving $n = 96$ for the masseter ES2 analyses. All groups were affected.

exhibited tenderness in at least two of the six pericranial muscle groups ($\chi^2(4, n = 142) = 23.77$, $P < 0.001$). Moreover, pairwise comparisons indicated that by this criterion, chronic tension-type headache sufferers exhibited pericranial muscle tenderness significantly more frequently than subjects in each of the other headache disorder groups as well (all tests, $P < 0.01$). Muscle tenderness was, however, common in the other headache groups, occurring in 40–60% of subjects with other recurrent headache disorders. Pericranial muscle tenderness was thus quite successful in distinguishing chronic tension-type headache sufferers from controls, but only moderately successful in distinguishing chronic tension-type headache sufferers from individuals with other recurrent headache disorders.

Chronic tension-type headaches were most frequently associated with tenderness in the upper trapezius (84% of subjects), cervical (74% of subjects), suboccipital (71% of subjects) and sternocleidomastoid (65% of subjects) muscles. Of the six muscle pairs examined, the masseter (39% of subjects) and temporalis (39% of subjects) were the least likely to be tender in chronic tension-type headache sufferers. The upper trapezius was tender in over 80% of chronic tension-type headache sufferers, but only 56% of the controls ($\chi^2(4, n = 142) = 11.06$, $P < 0.05$). However, pairwise comparisons indicated that chronic tension-type headache sufferers were no more likely than other headache sufferers to experience tenderness in this muscle ($\chi^2(4, n = 111) = 2.42$, $P = 0.05$). The upper cervical muscles were tender in 74% of chronic tension-type headache sufferers, but were only tender in 26% of controls ($\chi^2(4, n = 142) = 19.50$, $P < 0.001$). Moreover, pairwise comparisons indicated that tenderness in the upper cervical muscles successfully distinguished chronic tension-type headache sufferers from controls and individuals with other recurrent headache disorders (all tests, $P < 0.05$). The cervical muscles were tender in only 46% of the migraine without aura group, 37% of the episodic tension-type headache group, 24% of the migraine with aura group and 26% of controls. Tenderness in the remaining muscle pairs failed to distinguish chronic tension-type headache sufferers from individuals with other recurrent headache disorders.

Discussion

Abnormalities in pericranial muscle tenderness, but not in the second exteroceptive silent periods (ES2), successfully distinguished young chronic tension-type headache sufferers from controls who did not suffer from headache problems. Elevations in pericranial muscle tenderness observed in this young sample of chronic tension-type headache sufferers are consistent with previous findings from the handful of studies that have assessed this variable in either tension-type headache patients or in representative population samples (Lous and Olesen 1982; Langemark and Olesen 1987; Drummond 1987; Schoenen et al. 1991a; Hatch et al. 1992; Jensen et al. 1992). However, the shortened exteroceptive silent period that has been observed in patient samples of chronic tension-type headache sufferers (Schoenen et al. 1987; Nakashima and Takahashi 1991; Schoenen 1993a,b) was not evident in our sample of young chronic tension-type headache sufferers. These findings raise the possibility that abnormalities in central pain modulation indexed by ES2 emerge late in the course of chronic tension-type headache problems, while abnormal levels of muscle tenderness appear early in the evolution of this disorder.

Pericranial muscle tenderness

Chronic tension-type headache sufferers in our sample exhibited significantly higher total tenderness scores than controls who did not suffer recurrent headache problems. Using a liberal criterion of tenderness in at least one pericranial muscle (Hatch et al. 1992), all of our chronic tension-type subjects fit the IHS sub-classification of chronic tension-type headache with disorder of pericranial muscles, and 52% of controls exhibited muscle tenderness. This high incidence of muscle tenderness may be due in part to the composition of our sample. A recent

population study of muscle tenderness found that females exhibit more tenderness than males, and younger people report more tenderness than older people (Jensen et al. 1992). Nonetheless, the high incidence of muscle tenderness observed here and in other studies (Lous and Olesen 1982; Langemark and Olesen 1987; Schoenen et al. 1991c) suggests that a viable subclassification of tension-type headache with disorder of pericranial muscles will require the development of more precise criteria for abnormal tenderness.

We arbitrarily defined significant muscle tenderness as tenderness in at least two of the pericranial muscle groups evaluated because tenderness in one muscle was common in both recurrent headache sufferers and controls, and no other accepted criterion is available. Using this criterion, chronic tension-type headache sufferers exhibited significant muscle tenderness almost three times as frequently as controls. Chronic tension-type headache sufferers also exhibited significant muscle tenderness more frequently than individuals with either episodic tension-type headache or migraine; however, 40–60% of subjects in these latter two groups also exhibited significant tenderness. Pericranial muscle tenderness was thus most strongly associated with chronic tension-type headaches, but also more weakly associated with other recurrent headache disorders.

Pericranial muscle tenderness was not specific to one or two sets of muscles; on the other hand, not all muscle groups were equally able to distinguish diagnostic groups. Trapezius muscles were most likely to be tender in chronic tension-type headache sufferers, but consistent with observations made by other investigators (Sola et al. 1955; Jensen et al. 1992), were frequently tender in individuals with other recurrent headache disorders and in control subjects. On the other hand, cervical muscles were frequently tender in chronic tension-type headache sufferers, but relatively infrequently tender in individuals suffering from other recurrent headache disorders or in controls. This suggests that particular patterns of muscle tenderness might be identified that are characteristically associated with chronic tension-type headaches.

Although headache disorders, particularly chronic tension-type headaches, were very frequently associated with muscle tenderness absolute levels of tenderness were only moderately elevated. This may be because our subjects were assessed when headache-free, as similar levels of tenderness have been reported in other subjects when subjects are assessed headache free (e.g., Drummond 1987; Hatch et al. 1992). Higher tenderness levels have been reported when assessments are conducted during a headache episode rather than during headache-free periods (e.g., Drummond 1987; Jensen et al., 1993a,b). Tenderness is also correlated with the presence of headache during examination and with the temporal proximity of last attack at least in some studies, although most of the increased tenderness observed in tension-type headache sufferers does not appear to be related to these factors (Jensen et al. 1993b). In our investigation, tenderness was not significantly related to frequency of tension-type headache or migraine. Thus, muscle tenderness could reflect a psychophysiological abnormality that contributes to chronic tension-type headaches, and possibly migraine without aura.

Surprisingly little is known about the mechanism of muscle tenderness in recurrent headache disorders (Langemark and Olesen 1987; Olesen 1991). Histological analyses of the tender areas in pericranial muscles have not been conducted in recurrent headache sufferers. However, tender spots primarily in the trapezius muscle have been intensively studied in patients with fibromyalgia, a chronic disorder characterized by muscular pain and areas of muscle tenderness (Wolfe et al. 1990), and no specific muscle pathology has been found (Simons 1976; Bengtson et al. 1986; Hendrikson and Lindman 1993). In the present study elevated levels of tenderness were observed in the absence of the abnormalities in ES2 responses hypothesized to index deficits in central pain modulation associated with chronic tension-type headache. A sensitization of peripheral nociceptors or of second-order trigeminal tract neurons thus may

play a role in the early pathogenesis of chronic tension-type headache. Unfortunately the stimuli that might initiate peripheral sensitization are unknown, although muscular strain, muscular fatigue, inflammation, neuropeptides, ischemia, hypoxia and microlesions have been offered as candidates (see Langemark and Jensen 1988; Boissevain and McCain 1991, for reviews; Jensen et al. 1994). Research that clarified the origins of this muscle tenderness would likely add significantly to our understanding of chronic tension-type headaches.

Exteroceptive suppression

Abnormalities in ES2 responses reported in patient samples (Schoenen et al. 1987; Nakashima and Takahashi 1991; Schoenen 1993a,b) were not observed in our relatively young sample of chronic tension-type headache sufferers. The mean age of participants involved in previous ES2 studies varies between the late twenties and the late thirties; it is common for patients to have reported a 10–20 year history of chronic tension-type headaches. In contrast, the mean age of the present sample was 19 years and the average history of chronic tension-type headaches was less than 4 years, although subjects typically experienced episodic tension-type headaches for a longer period of time. Previous samples may thus have included subjects with long histories of recurrent headache problems and possibly analgesic use, who may have had concomitant physical or psychological problems that can complicate the interpretation of psychophysiological data. The only other study that used a general sample of young recurrent headache sufferers also failed to detect ES2 abnormalities (Gobel et al. 1992a), but also used a different assessment methodology than has been used in more recent studies (Schoenen 1993a,b). Additional unpublished data ($n = 178$) collected in our laboratory utilizing the ES2 assessment methodology recently recommended by the European Headache Foundation (Schoenen 1993a,b) are consistent with the findings of the present study.

It may be that the shortening of ES2 suppression periods emerge later in the evolution of the disorder, possibly as a long-term adaptation to recurrent pain. Consistent with this possibility is a recent report indicating ES2 suppression periods are shortened in chronic low back pain sufferers (Wallasch et al. 1992). Alternatively, the current methodology for assessing exteroceptive suppression periods may be insufficiently sensitive, or insufficiently reliable to detect deficits in central pain modulation early in the development of chronic tension-type headache (Wang and Schoenen 1994). Consistent with this latter possibility is the considerable overlap in the ES2 values of controls and chronic tension-type headache patients (Schoenen 1993b; Wang and Schoenen 1994), and the failure to detect differences in the ES2 durations of controls and chronic tension-type headache patients when assessment methodology is varied (Zwart and Sand, 1995). Future studies might examine relationships between ES2 durations and age, chronicity of headache problems and analgesic use, and the presence of other psychological and physical disorders.

Limitations and conclusion

Some limitations should be recognized in interpreting the results of the present study. Although we found manual palpation to be a sensitive test for chronic tension-type headache, this technique has been criticized because it is subject to bias (Langemark and Olesen 1987). Experimenter bias cannot be ruled out in the present study because the experimenters were not unaware of the subjects' headache diagnosis. Nonetheless, tenderness scores did not differ across experimenters, a standard examination protocol was used, and the present results are similar to previous findings obtained using blinding techniques (e.g., Langemark and Olesen 1987; Jensen et al. 1988). Moreover, a more recent study conducted in our laboratory in which the assessor was blind to subjects' headache diagnosis replicated these results.

In conclusion, this study is the first to examine the relationships between pericranial muscle tenderness and exteroceptive suppression and headache diagnosis defined by IHS criteria in a

general sample of young adults with recurrent headache disorders. Our findings raise the possibility that pericranial muscle tenderness is present early in the development of tension headache, and possibly migraine without aura. This early onset of pericranial muscle tenderness in headache sufferers suggests that this muscle tenderness might provide clues about the etiology of recurrent headache disorders. On the other hand, it appears that ES2 abnormalities might not emerge until later in the evolution of the disorder.

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Fig. 1.
Scoring of exteroceptive suppression from 10 rectified and averaged masseter EMG responses to electrical stimulation.

Fig. 2.
Percent of subjects with tenderness in more than one muscle group.

TABLE I
PERCENTAGE OF SUBJECTS MEETING IHS DIAGNOSTIC CRITERIA

	Chronic tension (<i>n</i> = 31)	Episodic tension (<i>n</i> = 31)	Migraine no aura (<i>n</i> = 33)	Migraine with aura (<i>n</i> = 17)
Pain characteristics				
Unilateral	19	10	61	76
Bilateral	81	90	39	23
Pulsating	23	13	73	82
Non-pulsating	77	87	27	18
Associated symptoms				
Photophobia	26	22	82	82
Phonophobia	10	6	55	41
Nausea	26	6	61	71
Headache aggravated by routine activity	32	22	61	82

TABLE II
HEADACHE VARIABLES

	Chronic tension (n = 31)	Episodic tension (n = 31)	Migraine no aura (n = 33)	Migraine with aura (n = 17)
Frequency (headache days per year)				
Mean	261.55	85.35	84.85	111.59
Range	180–360	12–156	12–310	12–280
Chronicity (in years)				
Mean	3.90	5.27	6.18	6.47
Range	1–10	1–14	1–14	1–16
Intensity (pain rating on a scale from 1–10)				
Mean	5.98	5.45	6.97	7.47
Range	4–8	3–8	5–10	4.5–9.5
Duration of typical headache (hours per day)				
Mean	7.68	4.18	10.36	9.70
Range	1–24	1–24	4–72	4–48

TABLE III

MEANS AND STANDARD DEVIATIONS FOR ES2 VARIABLES

	Chronic tension (<i>n</i> = 27)	Episodic tension (<i>n</i> = 15)	Migraine no aura (<i>n</i> = 19)	Migraine with aura (<i>n</i> = 14)	Control (<i>n</i> = 21)	<i>F</i> ratio (<i>df</i> = 4,91)	<i>P</i> value
Latency (msec)							
Mean	43.48	45.43	44.63	46.64	44.10	0.53	NS
SD	8.28	5.98	7.06	8.37	5.15		
Duration (msec)							
Mean	30.83	30.77	30.94	33.86	34.17	0.23	NS
SD	12.01	9.70	16.32	12.91	11.50		

NS, not significant.

TABLE IV

MUSCLE TENDERNESS (TTS)

	Chronic tension (<i>n</i> = 31)	Episodic tension (<i>n</i> = 31)	Migraine no aura (<i>n</i> = 33)	Migraine with aura (<i>n</i> = 17)	Control (<i>n</i> = 31)	<i>F</i> ratio (<i>df</i> = 4,137)	<i>P</i> value
Mean	20.06, ^{<i>ab</i>}	17.17	18.64 ^{<i>b</i>}	15.41	14.26	45.16	<0.001
SD	6.15	5.48	6.21	3.34	3.22		

^{*a*} Significantly different from migraine with aura group, *P* < 0.05.

^{*b*} Significantly different from control group (*P* < 0.05).