Chapter 71

Neurophysiology of Tension-Type Headaches

Jean Schoenen and Lars Bendtsen

Various neurophysiologic methods have been used in patients with tension-type headache. By far, the most frequent reports have dealt with electromyographic (EMG) recordings of pericranial muscle activity. This situation is explained easily by the fact that in the former Headache Classification of the Ad Hoc Committee (1), *tension headache* was used as a synonym for *muscle-contraction headache,* and was considered to be "associated with sustained contraction of the skeletal muscles in the absence of permanent structural change, usually as part of the individual's reaction to life's stress." Contrary to the first edition, the second edition of the Headache Classification prepared by a subcommittee of the International Headache Society (ICDH-II) (30) has abandoned this concept, since the three-digit code subtypes are now identified according to presence (code 1) or absence (code 2) of pericranial tenderness without reference to pericranial muscle contraction.

Recently, attention has focused on the inhibitory reflexes of the jaw-closing muscles (66). Other neurophysiologic methods, in particular those exploring the activity of the brain, such as electroencephalography (EEG), have not disclosed any clear-cut abnormality in tension-type headache (63), as is also the case for contingent negative variation, an event-related potential, which is abnormal in migraine between attacks but normal in tension-type headache (63). By contrast, abnormalities have been found in tension-type headache with nociceptive-specific laser-evoked potentials (16).

ELECTROMYOGRAPHY

Contraction of head and neck muscles has been thought to play a pathogenetic role in some patients with tension-type headache. Contradictory results have been reported by recording pericranial muscular activity in tension-type headache or, as previously termed, muscle-contraction headache. Relevant data obtained with surface EMG recordings are summarized in Table 71-1, in which positive data are those that favor a role of increased muscular activity in the pathogenesis of tension-type headache, and negative data represent those that do not. Most of the EMG studies published up to 1983 were critically reviewed by Pikoff (53): About half of the studies appeared to be normal, and in the other half, it was concluded that pericranial EMG levels were increased. Most subsequent studies, however, tend to indicate concordantly that EMG levels are abnormal compared with controls, despite wide variations in experimental conditions. For instance, EMG levels were found to be increased in tension headache sufferers during postural changes (71), during mental stress (17), and during the headache phase compared with the headache-free interval (54) by analyzing the increment of EMG between rest and maximal contraction (74) or by comparing different electrode placements (29). In a recent study (26) of episodic tension-type headache sufferers recorded in the headache-free phase, higher levels of average temporal EMG levels were found in the patient group compared with controls. Because only 11% of headache subjects had elevated (two or more standard deviations higher than the mean of controls) EMG activity in at least one muscle, it was believed that EMG data were of little use in assigning individual subjects to diagnostic groups.

We recorded EMG activity in 32 female patients suffering from chronic tension-type headache (code 2.2) over the frontalis, temporalis, and trapezius muscles in supine and standing positions as well as during a mental task (66). On average, EMG activity was significantly higher in patients compared with healthy female volunteers of comparable ages, and this was the case in all three muscles studied and under any condition (Fig. 71-1). Of the patients, 62.5% had at least one EMG level exceeding the mean control values by two standard deviations. Among these patients, five

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TABLE 71-1 Pericranial Surface EMG Activity

"Positive" Data	*"Negative" Data*
Increased in 50% of studies (53)	Normal in 50% of studies (53)
Increased on average in CTTH (61)	Significantly increased in less than 34% of CTTH patients (61,66)
Increased on average in ETTH (61)	Significantly increased in only 11% of ETTH patients (26)
	Not correlated with headache intensity (12,31,58,61)
Superior stress-induced increase (26,55,63)	Stress-induced increase similar in controls (61)
Increase in frontalis during headache in population-based study (34)	No increase during headache in patients (45,75)
Experimental tooth-clenching produces headache (35)	Headache appears after delay of several hours when EMG is decreased (35); also in migraine (33)
Increased in subgroup 2.2.1 (32)	No difference between 2.2.1 and 2.2.2 (66)

CTTH, chronic tension-type headache; EMG, electromyographic; ETTH, episodic tension-type headache

had one abnormal value (of a maximum of six), and only two patients had six abnormal values. If one muscle and one condition were considered, for example, the frontalis in the supine position, 11 patients (34%) would have been considered abnormal. No correlation was found between EMG levels and headache severity or the score on an anxiety scale or pressure pain thresholds. After 10 sessions of muscular biofeedback therapy, EMG levels tended to decrease in all muscles, but this modification was significant only in some muscles and under certain recording conditions. After biofeedback therapy, a slight but significant positive correlation was found between improvement in the headache severity index and maximal reduction of EMG levels; that is, the EMG level that decreased the most considering the three recording sites and the three conditions. Another study found no significant clinical difference between patients with abnormal EMG values and those in whom all EMG recordings were normal (67).

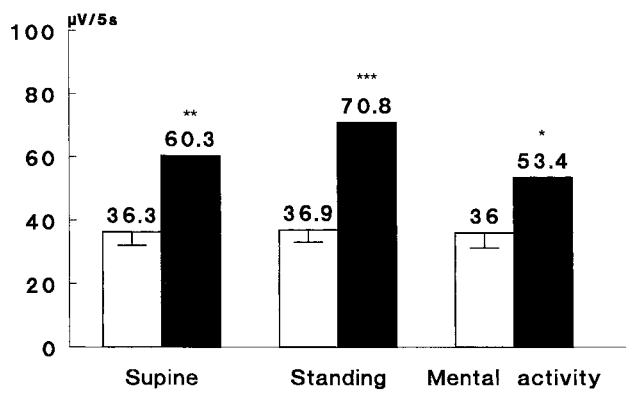

FIGURE 71-1. Electromyographic (EMG) levels (mean in μV/5 s – SD) of frontalis, temporalis, and trapezius muscles recorded with the patient in a supine position, in a standing position, and during an arithmetic task. Thirty-two female patients with chronic tension-type headache (open bars) and 20 healthy female controls (tippled bars) were included. $^{*}P < 0.05$; $^{**}P < 0.002$; $^{***}P < 0.001$.

Studies of the possible relation between pericranial EMG activity and stressful situations have produced conflicting results. Increased EMG activity during laboratory stressors have been reported in tension headache patients (13,18,75). Conversely, a large group of investigations did not find a reliable difference between the EMG activity of tension-type headache patients and headache-free controls in their response to laboratory stressors (3,4,7,17,19,44,66,72,76). In a recent study using ambulatory EMG recordings, higher levels of EMG activity were found on "high-stress days" both in headache-free students and in students with episodic tension-type headache; however, no statistically significant difference was found between the two groups (26,58).

Evidence has been found that pericranial and neck muscle activity is neither quantitatively nor chronologically related to head pain. The EMG levels are not higher in more severely affected patients (31,58,61,67); during prolonged ambulatory EMG recordings, muscle activity varies with daily activities and stress but not with pain levels (12,58). During an actual headache, EMG levels increased over the frontalis muscle in only one population-based study (34), but not in patients (12,35). No difference in EMG levels was found between chronic tension-type headache associated (code 2.2.1) or unassociated (code 2.2.2) with a muscular disorder in one study (67); on the other hand, levels were higher than those in healthy controls in only the 2.2.1 subgroup in another study in which normal EMG levels were found in both subgroups of episodic tension-type headache (32). Two subgroups of episodic tension-type headache sufferers were distinguished on the basis of their pericranial EMG response to experimental pressure-induced pain over the temples, one group with an exaggerated EMG activity and the other with an abnormally low EMG response (23).

Ischemic pain induced in the arm by a tourniquet was not accompanied by higher EMG levels in tension-type headache sufferers compared with controls, although the headache patients rated the tourniquet as more painful (39). Tooth clenching may induce headache in tension-type headache sufferers and has been used as a model to study pathogenetic mechanisms (35). The headache, however, increases gradually over several hours and is associated with pericranial EMG levels that are somewhat lower than the initial ones (35); these levels also can be produced in 54% of migraine patients (33).

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It has been argued that myofascial pain syndromes could be related to changes in the viscoelastic properties of muscles, that is, so-called thixotropy (70). The recent findings (using a new device, the muscle hardness meter) of increased muscle stiffness in tension-type headache sufferers, irrespective of the presence of headache (6,59) favors such a mechanism. Within the same concept, the pain in tension-type headache might be caused by muscle contracture; in this case, increased electric activity would be localized at the level of trigger points (70). With monopolar needle EMG recordings, increased activity was found at trigger points in patients with chronic tension-type headache (28), more often during experimental stress (45). It is not clear whether the EMG activity was recorded from muscle spindles or from the motor end plate zone, and some investigators did not find any abnormality when placing needle electrodes in trigger points. In one recent study, botulinum toxin injections decreased after 12 weeks temporalis EMG levels in patients with chronic tension-type headache, but not the headache, which another indication that muscle activity per se is not the culprit in chronic tension-type headache (56).

To summarize, EMG recordings have no diagnostic usefulness in tension-type headache, although they may help in understanding its pathogenesis. Contradictory results have been reported for EMG levels of pericranial and neck muscles. Some of these contradictions may be attributable to the different recording conditions. Taken together, however, the published results indicate that there is no causal relationship between the headache and surface EMG activity, although some patients may have EMG levels over certain muscles that exceed those of control subjects. In this respect, the findings may be comparable to those reported in chronic low back pain, where muscle activity is considered a normal protective adaptation and not the cause of pain (5,43). It remains to be determined whether muscular contracture and muscle hardness or localized muscle fiber contraction under trigger or tender points play a role in the initiation or the boosting of pain in tension-type headache. In future studies it is also worthwhile to explore more accurately frequency-related subtypes of tension-type headache by using the new ICHD-II subdivision into infrequent (<1 day per month), frequent (>1 but <15 days per month), and chronic (>15 days per month) forms, and the more stringent diagnostic criteria for TTH proposed in the ICDH-II appendix (A2) (30).

BRAINSTEM REFLEXES

The measurement of brainstem reflexes constitutes a noninvasive method for investigating central processing of sensory information from the cephalic region. Brainstem reflexes have therefore been studied in several neurologic disorders. In tension-type headache, exteroceptive suppression of jaw closing muscle activity has been investigated extensively, and the blink reflex and the trigeminocervical reflex have been examined in a few studies.

Exteroceptive Suppression

Electrical stimulation of the trigeminal nerve fibers normally elicits suppression of voluntary contraction in the masseter and temporal muscles (24). This suppression is mediated by an inhibitory brainstem reflex, which has been called *exteroceptive suppression, cutaneous silent period,* or *inhibitory period*. *Exteroceptive suppression* has traditionally been used in headache studies (67) to avoid confusion with the proprioceptive silent period of peripheral limb muscles. Exteroceptive suppression is divided into two periods, an early period (ES1) mediated by an oligosynaptic pathway, and a late period (ES2) mediated by a polysynaptic pathway (24). Because ES1 has not generally been reported abnormal in tension-type headache, only ES2 is considered here. The interneurons responsible for ES2 probably belong to the bulbar reticular formation (27). This area receives afferents from the periphery, but also from limbic structures, the orbitofrontal cortex, the nucleus raphe magnus, and the periaqueductal gray matter (63) (Fig. 71-2). Studies on ES2 may therefore provide information on the excitability control of these brainstem interneurons.

Exteroceptive suppression of jaw closing muscle activity has been used extensively in investigations of pain mechanisms (20,56), motor control (24), lesions within the brainstem (27), Parkinson disease (49), generalized anxiety disorder (57), whiplash (37), and tension-type headache (2,10,13,15,21,22,40–42,48,51,52,62,65,67,73,77,79,80). In this section, we briefly discuss the methodology and the results obtained from tension-type headache studies as well as some data on physiologic or pharmacologic modulation of exteroceptive suppression.

Methodology

Exteroceptive suppression of jaw closing muscle activity can easily be measured using a conventional EMG apparatus. The results obtained are, however, heavily influenced by the methodology used (8,15,63). Standardized, reliable, and blinded methods for recording and analyzing ES2 should therefore be employed. We briefly outline a methodology, which has proved reliable (8,25). While the subject is voluntary clenching his teeth, a slightly painful electrical stimulus (e.g., 20 mA) is applied to the labial commissure. The level of voluntary contraction should exceed 50% of

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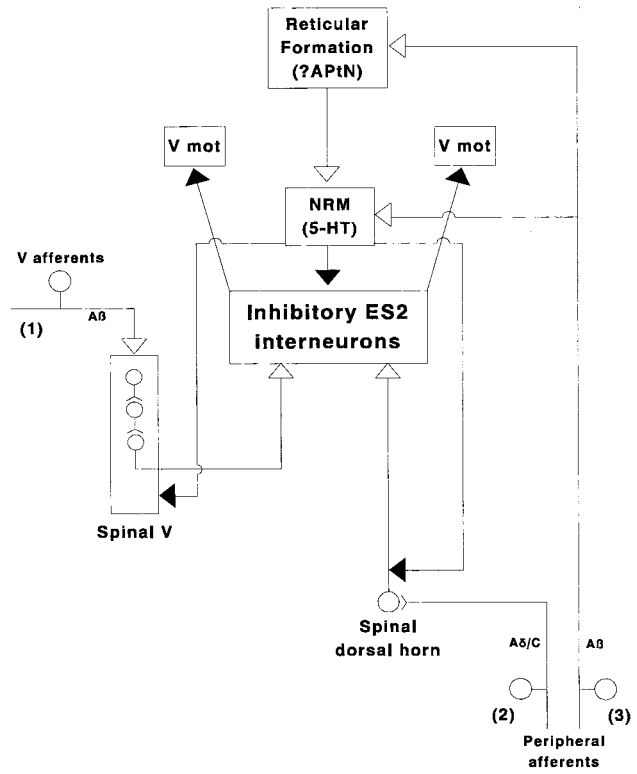

FIGURE 71-2. Diagram illustrating some of the neural pathways and their transmitters involved in the modulation of jaw-closing muscles and late exteroceptive suppression.

maximum (25,63), and the electrical stimuli should be applied with intervals of at least 10 seconds to avoid habituation (63). Recording of jaw closing muscle activity can be performed with surface electrodes, because this method gives results, which are comparable to those obtained with needle electrodes (25). In each recording series, at least 10 electrical stimuli should be delivered and the resulting EMG activity should be rectified and averaged. This ensures a fairly smoothed EMG signal (Fig. 71-3). Because suppression periods start and finish gradually, a cut-off point for taking measurements has to be defined. From the averaged EMG signal, the duration of ES2 can, for example, be defined as the duration in which there is at least 50% suppression compared to prestimulus baseline EMG activity (see Fig. 71-3). The analysis should be performed by a blinded observer.

Findings in Headache Patients

In 1987, Schoenen et al. (67) reported that the duration of temporalis ES2 was reduced in chronic tension-type headache. The 25 patients who were included in the study had approximately a 50% reduction of ES2 compared to the 22 healthy controls (Table 71-2). This original finding has later been confirmed by several unblinded studies (48,62,73,77) and one small blinded study (15) (see Table 71-2). However, two unblinded (2,42) and four blinded studies (9,41,42,80) including two large studies including examining 55 (9) and 245 (41) patients found normal ES2 values. The discrepancy between the studies cannot be easily explained. As discussed (9), it is possible that differences in the methods used for analyzing ES2 play a role, because the analysis is the most critical part of the methodology (8). Thus, at the present it is unclear whether ES2 per se is abnormal in chronic tension-type headache. In contrast, it has consistently been reported that the duration of ES2 is not significantly correlated to either clinical characteristics (e.g., headache frequency) or to various pain parameters (e.g., pain thresholds in patients with tension-type headache) (42,67). In patients with episodic tension-type headache and in patients with migraine, ES2 has been reported largely normal (21,48,51, 67,77).

Physiologic and Pharmacologic Modulations of Exteroceptive Suppression

In addition to baseline measurements of ES2, it is of interest to examine whether ES2 can be modulated by physiologic or pharmacologic interventions, and if so, whether the effect of such interventions differs between headache patients and healthy controls. In healthy controls, ES2 is reduced during experimental pain (8,69) and reduced on the first compared with the second day of examination (8). The latter tends to be more pronounced in patients with chronic tension-type headache than in healthy controls (9). Because ES2 can be reduced by attentional factors (11), this suggests that headache patients are more aroused in the experimental situation than healthy controls (9). Furthermore, it has been demonstrated that ES2 duration is reduced by a preceding electrical stimulus applied at the periphery (69), and that this reduction is more pronounced in patients with tension-type headache than in healthy controls (69) (Fig. 71-4). The latter finding may be caused by hyperexcitability of brainstem relays interposed between the afferent peripheral input and the medullary interneurons in patients with tension-type headache (79).

In a preliminary study, Schoenen et al. (68) investigated the modulation of ES2 in healthy volunteers following administration of various serotonergic drugs. It was found that methysergide, a serotonin antagonist, prolonged ES2, that fluoxetine, a serotonin reuptake inhibitor, reduced ES2, and that sumatriptan, a serotonin agonist, had no significant effect on ES2. These data indicate that drugs that increase serotonin levels tend to decrease ES2, and drugs that block serotonin receptors tend to increase ES2. This was supported by Bendtsen et al. (10), who demonstrated that amitriptyline, a combined serotonin and

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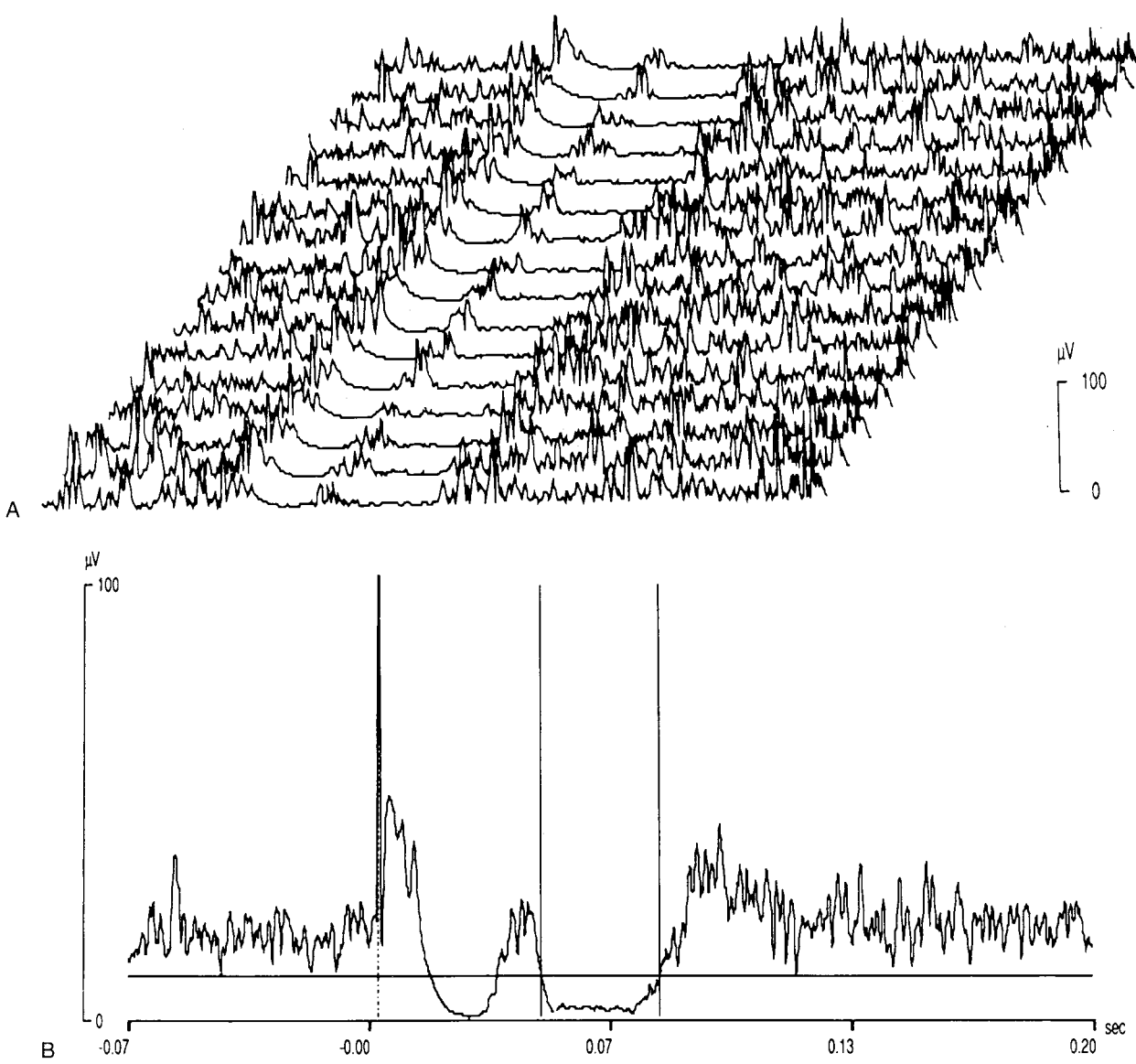

FIGURE 71-3. Exteroceptive suppression of temporalis muscle activity in a healthy volunteer. **(A)** Sixteen single signals were recorded and rectified. **(B)** Average of the 16 rectified signals. The electrical stimulus was delivered at $t = 0$ s. A horizontal cursor, placed automatically, indicated 50% of the prestimulus mean electromyographic (EMG) amplitude. Vertical cursors, placed manually, indicated the beginning and the end of the ES2 period. Amplitude values have to be multiplied by 10. From Bendtsen et al. (8) with permission.

noradrenaline inhibitor, reduced ES2 in patients with chronic tension-type headache. In contrast to this, Göbel et al. (22) could not detect any difference in ES2 before and after treatment with amitriptyline in patients with chronic tension-type headache. In an earlier study, Göbel et al. (21) found that acetylsalicylic acid prolonged ES2 in both tension-type headache patients and healthy controls. Further pharmacologic studies may help to identify the neurotransmitters involved in the regulation of ES2 and to clarify possible central actions of drugs of interest for headache treatment.

The Blink Reflex and the Trigeminocervical Reflex

Sensory stimulation of the ophthalmic territory evokes reflex responses of the orbicularis oculi muscle, the so-called blink reflex. The blink reflex is a protective brainstem reflex that consists of two separate responses, an early ipsilateral (R1) and a late bilateral (R2) component (14). The early component is transmitted through at least one interneuron in the pons, and the late component relays in the spinal trigeminal nucleus, and then ascends along a

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TABLE 71-2 Studies of the Late Exteroceptive Suppression Period, ES_2, in Patients with Chronic Tension-Type Headache and Healthy Controls

Study	Method	N	Blinded Design	ES_2 Duration		Significant Difference
				Patients	Controls	
Schoenen et al. 1987 (67)	Averaging 10 responses	25	No	25 msec	47 msec	Yes
Nakashima and Takahashi 1991 (49)	Averaging 32 responses	17	No	21 msec	43 msec	Yes
Wallasch et al. 1991 (77)	Mean of 3 responses	29	No	21 msec	41 msec	Yes
Zwart and Sand 1995 (80)	Mean of 10 responses	11	Yes	36 msec	34 msec	No
Bendtsen et al. 1996 (9)	Averaging 16 responses	55	Yes	33 msec	35 msec	No
Lipchik et al. 1996 (40)	Averaging 10 responses	27	No	31 msec	34 msec	No
Lipchik et al. 1997 (42)	Averaging 10 responses	22	Yes	11 msec	19 msec	No
Schepelmann et al. 1998 (62)	Averaging 20 responses	18	No	23 msec	33 msec	Yes
Lipchik et al. 2000 (41)	Averaging 10 responses	245	Yes	29 msec	28 msec	No
Aktekin et al. (2)	Averaging 10 responses	20	No	30 msec	31 msec	No
Tataroglu et al. (73)	Mean of 5 responses	25	No	37 msec	46 msec	Yes
de Tommaso et al. (15)	Averaging 10 responses	15	Yes	23 msec	40 msec	Yes

N is number of patients evaluated.

bilateral polysynaptic pathway, possibly through the lateral bulbar reticular formation (14). In analogy to exteroceptive suppression of jaw closing muscles, the blink reflex may provide information on central mechanisms in tension-type headache. Sand and Zwart (60) compared the blink reflex in 11 patients with chronic tension-type headache and 9 healthy controls. No significant differences regarding the latencies of the R1 or the R2 component was found between the two groups. Aktekin et al. confirmed this result but found that the recovery cycle of the blink reflex was diminished in chronic tension-type headache (2). The latter might reflect reduced excitability of brainstem interneurons (2).

Supraorbital nerve stimulation may elicit a reflex response from the sternocleidomastoid muscle entitled trigeminocervical reflex (46). The pathway for this brainstem reflex is unknown, but it is probably transmitted through a polysynaptic route (47). The trigeminocervical reflex may therefore possibly be used for the study of brainstem interneuron activity (46,50). Milanov and Bogdanova (46) and Nardone and Tezzon (50) found reduced latency of the trigeminocervical reflex in patients with chronic tension-type headache, suggesting decreased activity of brainstem interneurons. Recently a method was developed allowing to better identify the nociception-specific component of the blink reflex, but up to now it has chiefly been used in migraine patients (36).

To summarize, the blink reflex and the trigeminocervical reflex may be abnormal in chronic tension-type headache and thereby provide important information on

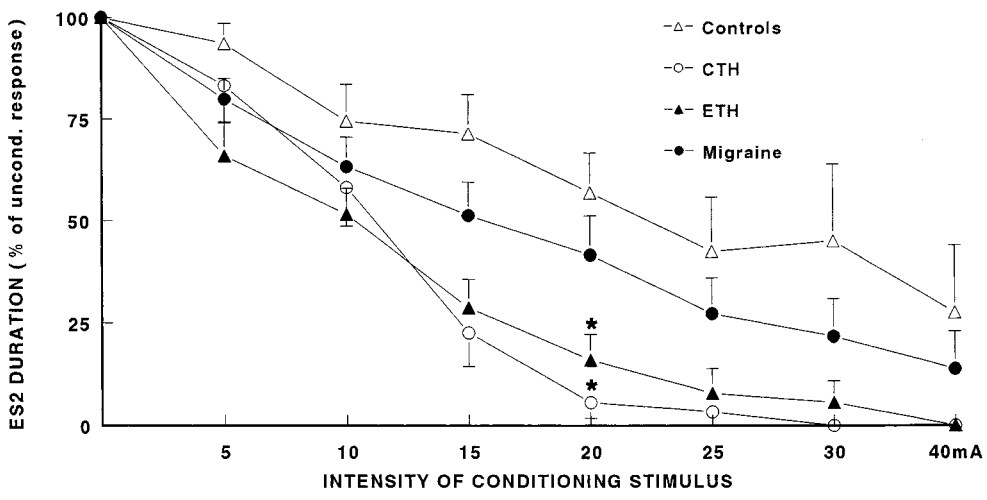

FIGURE 71-4. Decrease of temporalis ES2 duration (expressed as percentage of unconditioned response) with increasing intensities of a conditioning stimulus applied at the index finger 60 ms before the labial commissure stimulation in healthy controls, chronic tension-type headache, episodic tension-type headache, and migraine without aura.

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the pathophysiology of this disorder. However, more studies, which have to be blinded, are needed before final conclusions can be made.

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