The history of tension-type headache: a bibliographical journey from Arnold P. Friedman to today

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ABSTRACT

Introduction. Tension-type headache is a disease whose conception has changed over time.

Material and methods. We performed a literature search of the PubMed database using the keywords "tension-type headache" and "tension headache."

Results. We found publications from the 1950s highlighting differences between this type of headache and migraine, such as its better response to placebo. However, it was not until 1990s that research activity rapidly grew, and a more comprehensive perspective gradually took shape, despite the conception of the disease remaining somewhat incomplete. In the 2000s, the number of publications on tension-type headache trebled, further clarifying the concept, with clear distinctions based on episode frequency, and the recognition of other more complex elements such as headache of genetic origin.

Conclusions. The history of the concept of tension-type headache shows that the initial focus was on the possible psychic origin of this entity. Unlike migraine, a surprisingly small amount of research has been performed on this type of headache. However, it is worth mentioning the contributions of the different classifications of headache to the clarification of this condition. Today, a paradigm shift is to be expected that may give rise to improvements in the quality of life of these patients.

KEYWORDS

Headache, history, medical terminology, muscle contraction headache, tension headache, tension-type headache

Introduction

The term "tension-type headache" has evolved over the decades from its first description to today. The current edition of the International Classification of Headache Disorders (ICHD-3) defines it as "infrequent episodes of headache, typically bilateral, pressing or tightening in quality and of mild to moderate intensity, lasting minutes to days. The pain does not worsen with routine physical activity and is not associated with nausea, although photophobia or phonophobia may be present."[1]

The expressions "tension-type" or "tension" refer to the prior belief among the scientific community that the cause was sustained contraction of the muscles of the head and neck.

This hypothesis has progressively been disproven over the years by electromyographic studies, which did not demonstrate activity of these muscles in association with headache attacks, as well as other studies that led to the acceptance that, conceptually, this entity in fact constituted part of a migraine–tension-type continuum.[2]

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Figure 1. Arnold P. Friedman (photograph taken in 1955). Source: U.S. National Library of Medicine.

The conception of this condition as a functional process gradually changed and started to consider its neurobiological basis, at least in more severe cases.[3]

This study aims to provide a clear view of the history of the concept by reviewing the literature published in each decade.

Material and methods

We used the PubMed search engine to find publications including the keywords "tension-type headache" and "tension headache"; we used the year of publication filter to classify results per decade, from the 1950s to today.

We selected the most relevant articles from each decade (organised with the search engine's "best match" function) to study the conception of the term during each of these historical periods.

Results

1950s

In this decade, we found numerous publications from Arnold P. Friedman (1909-1990) (Figure 1), an international authority on headache for several decades and founder of the first clinic exclusively dedicated to the treatment of headache.

1. Concept

In these articles, Friedman[4,5] refers to tension headache as headache related to a mood disorder and reports a clear causal association between emotional conflict and headache, establishing a parallelism with the causal association between trauma and post-traumatic headache. He reports that this type of headache generally manifests in patients who are partially aware of constant or periodic mood conflicts.

The clinical description is similar to that used today, although it accepts nausea as a possible frequent accompanying symptom. This may have led to a misdiagnosis of some cases of migraine, which may be wrongly classified as tension headache. Furthermore, Friedman describes variability in pain characteristics, from pressing to pulsating pain, which further supports this hypothesis.

2. Pathophysiology

Friedman proposes two different mechanisms leading to the development of this type of headache. According to the first hypothesis, an emotional conflict would stimulate the sympathetic nervous system, which would lead to a change in the vascular calibre and to a mainly cervical muscle contraction. The second mechanism proposed by this author would consist of a headache of changing nature.

The mechanism by which excessive contraction of the cervical muscles may trigger pain is explained by three different hypotheses: firstly, ischaemia caused by the muscle spasm (this theory would subsequently be accepted by the international scientific community for a long period); secondly, the release of potassium (partly as a consequence of ischaemia, partly due to the excessive muscle contraction), activating different chemoreceptors; and lastly, by constituting a noxious stimulus for the propagation of an excitatory signal in the central nervous system.

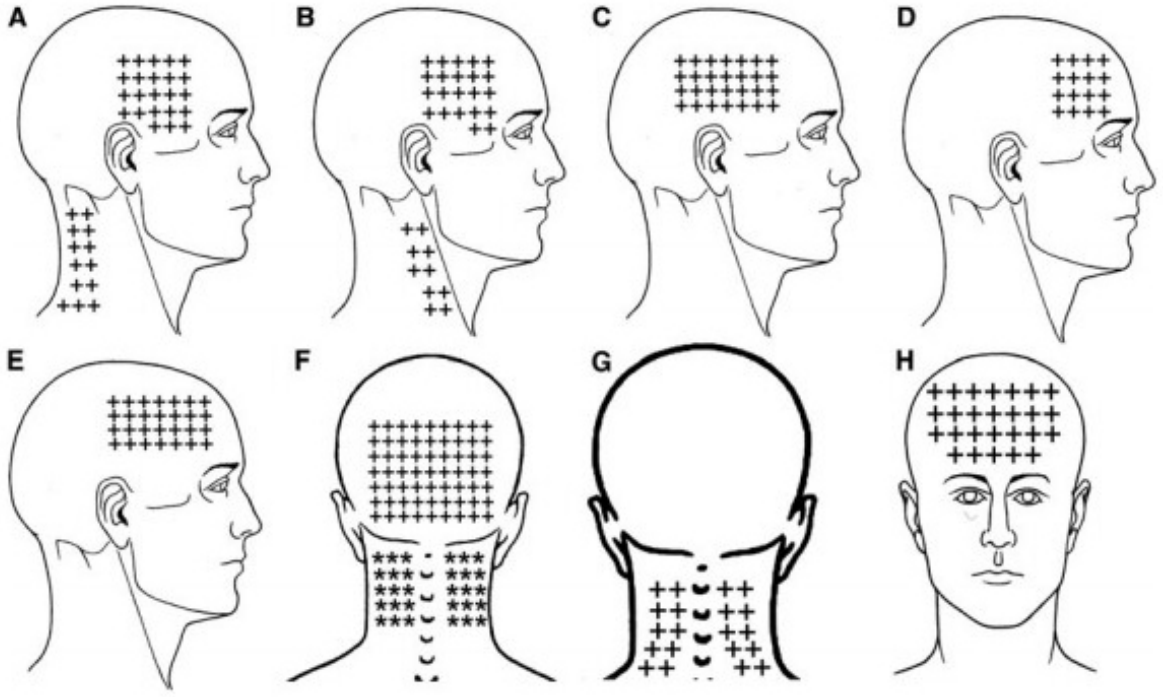

Figure 2. Referred pain from active trigger points in the upper trapezius (a), sternocleidomastoid (b), temporalis (c), superficial masseter (d), suboccipital (e), combined suboccipital-upper trapezius (f), levator scapulae (g), and superior oblique muscles (h). Source: Fernández-de-las-Peñas et al.[7] (2011).

However, Friedman also reports the similarity with post-traumatic headache in terms of clinical characteristics and the good response to similar drugs, and therefore the possible proximity between these two entities, a hypothesis that is still accepted today.

We should mention that all of Friedman's conclusions assume a classification of tension headache in accordance with diagnostic criteria proposed by himself or his team.

Therefore, despite working with sample sizes of up to 2000 patients, it is no surprise that the conclusions drawn today are different from Friedman's.

3. Treatment

Friedman proposed treatment with psychotherapy, together with sedatives (barbiturates, chlorpromazine) and analgesics; no long-term treatment was recommended.

1960s

1. Concept

In the 1960s, Friedman[6] continued publishing on this topic, substituting the term "tension headache" with "muscle contraction headache" on several occasions.

Many of his considerations from the previous decade persisted; however, he also stresses that patients usually described pain as pressing or "as a band" around the entire head. Furthermore, not only does he not mention muscle contraction, but he also describes "tension points" that are possibly comparable to those currently accepted as myofascial trigger points (Figure 2).[7]

In 1962, the first official classification of headaches (by the Ad Hoc Committee on the Classification of Headache of the National Institute of Neurological Diseases and

Blindness) was published; this document was frequently referred to by authors in the 1960s and 1970s, although it presents clear differences with the current classification. In this classification, among the first types of headaches, ordered according to their supposed frequency, we find "vascular headache," "muscle contraction headache," and "combined vascular and muscle contraction headache," which are comparable to the current terms "migraine," "tension-type headache," and the almost forgotten "mixed headache."

The emergence of the concept of a headache halfway between the two other major types paved the way for the scientific community to reconsider whether many cases diagnosed as tension headache in previous decades should really be included in the category of migraine, as occurs today.

The definition of muscle contraction headache in that classification was an "ache or sensation of tightness, pressure, or constriction, widely varied in intensity, frequency, and duration, sometimes long-lasting, and commonly suboccipital. It is associated with sustained contraction of skeletal muscles in the absence of permanent structural change, usually as part of the individual's reaction during life stress."

2. Pathophysiology

Friedman reports that electromyography shows the presence of muscle contraction, citing a study[8] from the previous decade that assessed 26 subjects during 85 headache episodes, as well as during asymptomatic periods. Despite the small size of the sample, the authors concluded that:

— Subjects with headache presented increased vasoconstriction, even during asymptomatic periods. This conclusion came from the reduced mean pulse amplitude in the temporal artery.

— Subjects with headache presented contraction in the temporal muscles. They observed a tenfold increase in motor unit action potential amplitudes measured with needle electromyography during headache episodes.

— When muscle contraction and cerebral vasoconstriction were sustained, headache occasionally appeared, but attacks were much less frequent when these two phenomena manifested separately.

Friedman also provided data on the measurements performed by his group using a technique based on the use of radioactive sodium. They did not confirm the hypothesis suggested in the previous decade that pain was caused by ischaemia; however, they did observe increased flow in the vascular bed. Despite this finding, many publications were based on this hypothesis, even in subsequent decades.

3. Treatment

No additional contributions were made to the field of treatment, with symptom resolution reported in 80% of cases after combined sedative and analgesic treatment, probably due to a synergistic effect; this contrasted with a 55% response rate when these drugs were administered alone.

1970s

1. Concept

Again, as in previous decades, authors insisted on the particular characteristic that, despite the assumed causal relationship between the supposedly stressing event and the onset of this type of headache, many patients did not recognise such an association. Once again, it was taken for granted that this disparity was explained by a perception defect, rather than questioning the existence of such event. However, many authors, including Ziegler,[9] began to doubt this association, especially after assessing regulated studies[10] measuring dimensions of personality with such inventories as the California Personality Index, which found no differences with respect to the rest of the population.

Regarding this matter, we should also mention Harrison,[11] who conducted an exhaustive review of the studies performing different psychological tests on patients with headache; he was highly critical and declared that "only a controlled trial of psychotherapy can demonstrate that personality factors are causally linked to headache."

Mandibular contraction or signs related to bruxism are described as symptoms suggesting this type of headache, which may be confused with the current concept of "headache attributed to temporomandibular disorder."

Furthermore, authors continued to insist on the finding of "exquisitely sensitive nodules," comparable to today's concept of myofascial trigger points, previously referred to by Friedman as "tension points," as mentioned above.

It was postulated for the first time in the 1970s that these patients may present a low pain threshold; this

is consistent with numerous contemporary studies,[12] though these focus more on allodynia in patients with migraine.

The question that emerged in the previous decade as to whether migraine and tension headache were a system of unconnected compartments rather than a continuum remained latent. Nonetheless, Ziegler[9] wondered: "In any single headache episode, how sharply and accurately can the line be drawn between the defining characteristics of tension headache and migraine?," later asking whether: "If, for example, a patient has a unilateral headache attack with nausea, is such an attack migraine, even though the patient also has the characteristic diurnal pattern of tension headache and also suffered from painful cervical muscles?" Thus, researchers also observed that almost all patients diagnosed with migraine also presented episodes with similar characteristics to those of tension headache.

2. Pathophysiology

Furthermore, a critical spirit began to emerge, questioning the hypothesis of excessive contraction of head and neck muscles in these patients,[13,14] with researchers reviewing the different studies conducted to date and considering that data were often inconclusive. Other studies describe muscle activity at rest in any type of headache, without distinguishing between types.[15]

3. Treatment

By the end of the 1970s, results began to be published[16,17] of several studies with amitriptyline and clomipramine as a treatment for tension headache and migraine, with positive results in both cases, although sample sizes were no larger than 100 patients. However, during this time period, no changes were made to the recommendations on the pharmacological treatment of these patients.

It is also noteworthy that at the same time, researchers began to mention (although in a discreet manner and probably with highly uncertain assumptions regarding the possible action mechanisms) the apparent effectiveness of treatment with infiltration of local anaesthetic at myofascial trigger points.

The term "holistic treatment" began to be used,[18] referring to the use of non-pharmacological therapies such as biofeedback (although acknowledging a significant placebo effect[19] in this type of therapy), massage, or physical exercise, although the disease was still conceived as the consequence of anxiety and internal conflict. Similarly, the terms "tension headache" and "headache secondary to muscle contraction" were often used interchangeably.

Biofeedback therapies guided by electromyography were used to relax the cranial and cervical muscles. A study on biofeedback using a three-phase guided programme of increasing complexity achieved a 38% reduction in electrical activity vs a 66% reduction in headache activity.[20] Considering that the latter reduction is practically twice as large as the former, these data, as previously mentioned, point to the possibility of a considerable placebo effect.

Also noteworthy are some other devices designed to control vasoconstriction by biofeedback techniques. For example, one device was used at the patient's home to measure the temperature in a finger.[21] This device, more frequently used in patients with migraine and with good results in almost half of the patients analysed, was also used in some patients with tension headache, but with poorer results.

1980s

The 1980s began with the foundation in 1981 of the journal *Cephalalgia*, which represents a milestone in the history of the study of headache, given the clinical relevance of the articles published in that journal.

1. Concept and pathophysiology

One of the first articles published in *Cephalalgia*[22] precisely addressed tension headache, suggesting that this type of headache may not be reliably associated with anxiety as, in patients with both clinical entities, each followed a different clinical course, behaving quite independently and improving or worsening very differently; they even manifest at different times of the patient's life. Furthermore, authors in the 1980s demonstrated that tension headache was not associated with contraction of frontal muscles, using electromyography studies. The most accepted aetiological hypothesis for this type of headache at that time was that it was actually a conversion process.

However, despite the controversy on whether muscle contraction constitutes a causal factor in this type of headache, the terms "muscle contraction headache" and "tension headache" were frequently used interchangeably,[23] probably due to the fact that the

first was the term of choice in the official or applicable headache classifications.

Researchers in the 1980s continued to question the dilemma that began in previous decades on whether the difference between migraine and tension headache was as clear-cut as previously thought.

Some studies published during this period reported that patients with tension headache presented a lower pain threshold than the general population.[24] Based on the conclusions drawn from these data, the hypothesis that the pathophysiology of tension headache may largely be due to the sensitisation of nociceptors became increasingly accepted.

A new classification of headaches published in 1988, "Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain,"[25] renamed this type of headache "tension-type headache." This new classification also distinguishes between chronic and episodic variants, and discards the pathophysiological hypothesis that it is caused by excessive contraction of pericranial muscles.

The proposed criteria for defining the condition are closer to the currently accepted concept, requiring two of the following criteria to be met: pressing pain, mild or moderate intensity, bilateral location, and pain that is not aggravated by such physical activities as climbing stairs. Also, patients may present neither nausea nor vomiting, and only one of photophobia or phonophobia.

2. Treatment

Regarding pharmacological treatment, the evidence in favour of amitriptyline continued to grow, with sedative treatment, which had been generally accepted up to that time, becoming less widely used. There was stronger support for the infiltration of local anaesthetics, as well as for the performance of rhizotomies. Recommendations on non-pharmacological treatments remained applicable, especially regarding relaxation psychotherapy and electromyographic biofeedback.

1990s

1. Concept and pathophysiology

In the 1990s, while some ideas in line with those of previous decades still persist, new studies were published[26,27] that categorically declared previous aetiological hypotheses on excessive muscle contraction secondary to emotional conflicts as the cause of tissue ischaemia to be incorrect. It was reported on many occasions that muscle contraction is far more pronounced in cases of migraine than in patients with tension-type headache, with electromyographic biofeedback being equally effective for both entities. Furthermore, other studies argued that there was no correlation between the degree of muscle contraction and the presence of headache. No further evidence on the presence of ischaemia in the temporal or pericranial muscles was obtained.[28]

However, the theory that both tension-type headache and migraine are triggered by alterations to central pain control became increasingly prominent, leading to the emergence of the central sensitisation theory. Authors continued highlighting the role of myofascial trigger points, even hypothesising a parallelism with fibromyalgia.

Particularly relevant in this decade were electrophysiological studies of the late exteroceptive silent period of temporal muscle activity, which was observed to be decreased in the early studies, suggesting that limbic pathways in the brainstem were affected.[29,30] However, these studies could not subsequently be replicated,[31] and this theory was not widely accepted.

2. Treatment

In terms of non-pharmacological treatment, researchers proposed such options as local application of heat or cold, electrical or ultrasound stimulation, posture education, or injections at trigger points and anaesthetic blocks of the occipital nerve. Changes in lifestyle habits, with the implementation of regular physical exercise programmes, stretches, relaxation techniques, balanced diet, and good sleep hygiene were recommended.

Regarding pharmacological treatment, researchers recommended early treatment with painkillers (ibuprofen, ketoprofen, or naproxen, and paracetamol in case of intolerance) to avoid provoking central hypersensitivity, but also taking care not to trigger medication overuse headache. Furthermore, combinations of different analgesics, especially those containing narcotic compounds or caffeine, may cause habituation when prescribed for long periods; therefore, they were not recommended.

Preventive treatment, in turn, was recommended for patients with headache frequency higher than twice per

week or episodes lasting longer than 3 or 4 hours, risk of medication overuse headache, or headache causing significant disability.

The treatment proposed was similar to the preventive treatment indicated for migraine, starting with antidepressants, which were probably recommended due to the tendency in previous decades to understand this process as being closely linked to a psycho-emotional disorder. In fact, by the end of the decade, researchers believed that there was a clear association between high levels of stress, emotional conflict, or mood disorders and this type of migraine, even in its episodic variant.[32]

This represented the strongest evidence regarding the use of amitriptyline to treat tension-type headache, and the use of benzodiazepines or muscle relaxants was no longer recommended; while they still used in routine clinical practice, no randomised clinical trial supported their use.

2000s

1. Pathophysiology

In this time period, the central sensitisation theory,[33] based on the sensitisation and decreased antinociceptive activity of supraspinal structures, remained relevant. This theory was corroborated by using painful stimulation with progressive pressure at different parts of the body to demonstrate that the pain threshold was decreased in these patients.[34]

The previous theory was supported by the effectiveness of amitriptyline as a preventive treatment for this type of headache, and it was hypothesised that the cause was not serotonin reuptake (as previous researchers had believed), but rather N-methyl-D-aspartate (NMDA) receptor blockade, as the activation of these receptors is essential in the development of sensitisation of the dorsal horn.

The possible role of nitric oxide was also mentioned, as infiltration of this compound was observed to reproduce symptoms of headache in patients with tension-type headache.[35] In this decade, the nitric oxide inhibitor N^G-methyl-L-arginine was also researched as a possible treatment for this type of headache, with very promising results.[36]

Through all these developments, the neurobiological pathophysiological basis was clearly established, in opposition to the previously proposed hypothesis of a psychosomatic basis. It was even suggested that tension-type headache and migraine may share a common biological basis,[37] with particular emphasis on the observation that tension-type headache is much more frequent in patients diagnosed with migraine than in the general population, and vice versa.

However, the most surprising and perhaps revolutionary development in this decade is the use for the first time, thanks to technological advancements, of brain magnetic resonance and voxel-based morphometry studies to observe microstructural alterations in patients with tension-type headache.[38] These studies described significantly decreased cortical thickness in regions related to pain processing, such as the dorsorostral and ventral pontine region, the anterior cingulate cortex, the bilateral anterior and posterior insular cortex, the right posterior temporal lobe, orbitofrontal cortex, bilateral parahippocampus, and the right cerebellum.

2. Treatment

It was widely accepted that amitriptyline was the drug with the most evidence, although the previously mentioned non-pharmacological therapies used in earlier decades (biofeedback, relaxation exercises, etc) and transcutaneous electrical nerve stimulation were also used. Acupuncture also began to be used in this context,[39,40] but with inconclusive results.

Furthermore, early studies were performed that used botulinum toxin to treat this type of headache,[41,42] with less promising results than expected. Different selective serotonin reuptake inhibitors were also widely studied,[43] with negative results, supporting the hypothesis that the effect of amitriptyline in tension-type headache was due to blockade of NMDA receptors and not serotonin reuptake, as mentioned above.

The use of muscle relaxants to treat this headache was categorically rejected and research began into the use of triptans, reporting modest but significant results for subcutaneous administration but no effect with oral administration. The effectiveness of subcutaneous administration may be explained by a possible association with the mechanism observed in animal studies, in which the drug inhibits the excitability of the neurons of the central nervous system.[44] In general, these drugs are not recommended for the acute management of pain in tension headache.

2010s

1. Concept

In this decade, the third and most recent version of the International Classification of Headache Disorders was published.[1] No significant changes were made with regard to the previous classification of tension-type headache, with the exception of its subclassification, with the following new subtypes: infrequent episodic tension-type headache (frequency of less than one episode per month), frequent episodic tension-type headache (1-14 monthly episodes), chronic tension-type headache (more than 14 monthly episodes), and probable tension-type headache (for patients not meeting all the criteria for these subtypes of tension-type headache nor the criteria for any of the other headaches in the classification).

2. Pathophysiology

It is surprising that, in the most recent decade, publications were more limited than in the earliest period addressed in this historical review, with authors being aware of the huge gaps still to be filled in our knowledge, asserting that the precise pathophysiology of tension-type headache remained to be clarified.[45] In fact, there was a revival of the hypothesis of the possible involvement of a component related to the peripheral nervous system in the pathophysiology of the disorder, in addition to the previously mentioned central nervous system component.

The following peripheral nervous system mechanisms were postulated: inflammatory reactions, decreased blood flow, increased muscle activity, and muscle atrophy. Regarding the latter point, new evidence was published on the presence of abnormal metabolism in the muscles of these patients.[46] It is unclear which of these mechanisms would trigger the other, or whether both act simultaneously, leading to pain; it was also suggested, although more cautiously than in the past, that the origin may be at the central level.

Furthermore, some researchers proposed a genetic association between this type of headache and certain polymorphisms, as well as a protective role, with the most relevant being related to catechol-O-methyltransferase,[47] which continues to be researched. Psychological factors continued to be studied. A novel aspect was the proposal of new trigger factors, such as vitamin D deficiency[48] or increased interleukin 1 levels,[49] associated with a proinflammatory vascular state.

3. Treatment

In the 2010s, mirtazapine was established as an alternative to amitriptyline, and new trials were developed with such other drugs as topiramate,[50] with favourable results despite the limited number of studies, and memantine, an NMDA antagonist, with limited effectiveness.[51] Subcutaneous administration of such local anaesthetics as lidocaine is effective,[52] which is consistent with results reported in previous decades; one study analysed subcutaneous administration of gonyautoxin,[53] which also seems to be effective, although no more evidence was published on this treatment. Authors continued to support the use of non-pharmacological therapies with similar techniques to those used previously, but accumulated further evidence on the use of acupuncture,[54] for instance, although further studies are still needed.

Conclusions

In conclusion, we can highlight some key ideas from our review of the modern history of tension-type headache.

The first refers to the fact that when the basis of a nosological entity is unknown, it is frequently attributed to a psychic process. This is probably a means of excusing our own lack of understanding and perhaps also to appease consciences uneasy with our inability to help patients.

Also surprising is the limited progress in biomedical research in possible preventive or abortive treatments specific to this type of headache. Some authors have suggested that this lack of research may easily be attributed to the greater emphasis on migraine. Another possible explanation is the classification of this condition almost as a psychosomatic disorder; for years, it was treated practically as a muscle contraction.

At least since the year 2000, we can no longer excuse this lack of interest in tension-type headache at this level. Shifts in mentality probably take time to occur and we probably have not left behind certain attitudes from the past, underestimating this entity and considering it a less disabling and more frequent headache that in many cases does not constitute a medical problem. However, we may expect to see growing scientific interest in this entity, which may soon lead to the development of new therapeutic options.

Table 1. Comparison of the different international classifications of headaches.

Document	**First International Classification of Headache Disorders (Ad Hoc Committee on the Classification of Headache of the National Institute of Neurological Diseases and Blindness)**	**International Classification of Headache Disorders-2: *Classification and diagnostic criteria for headache disorders, cranial neuralgia, and facial pain***	**International Classification of Headache Disorders-3: *The International Classification of Headache Disorders, 3rd edition***
Year of publication	1962	1988	2018
Term	Muscle contraction headache	Tension-type headache	Tension-type headache
Duration	Variable, sometimes long-lasting	From 30 minutes to 7 days	From 30 minutes to 7 days
Criteria	Ache or sensation of tightness, pressure, or constriction, widely varied in intensity, frequency, and duration, sometimes long-lasting, and commonly suboccipital. It is associated with sustained contraction (...) usually as part of the individual's reaction during life stress.	At least two of the following: *1)* bilateral location; *2)* pressing/tightening (non-pulsating) quality; *3)* mild or moderate intensity; *4)* not aggravated by routine physical activity such as walking or climbing stairs. Both of the following: 1) no nausea or vomiting (anorexia may occur); 2) no more than one of photophobia or phonophobia.	At least two of the following: *1)* bilateral location; *2)* pressing/tightening (non-pulsating) quality; *3)* mild or moderate intensity; *4)* not aggravated by routine physical activity such as walking or climbing stairs. Both of the following: 1) no nausea or vomiting; 2) no more than one of photophobia or phonophobia.

It is also important to highlight the contributions of the different classifications of headache (Table 1), which clarify concepts, help prevent selection bias in studies, and lend authority to the most reasonable line of thought on headache at each historical moment. The first classification may seem not to have achieved these objectives, even leading to confusion regarding the nature of the entity by changing the nomenclature to an even more unclear terminology; however, it is also true that the starting point was a speculative disarray and vague epistemological basis, precisely because these were early days in the history of the entity.

Conflicts of interest

The authors have no conflicts of interest to declare.

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